



*The University Library  
Leeds*



*Medical and Dental  
Library*



MEDICAL LIBRARY  
STACK. V5 B3 Q4 H

— E. B. S. W. F. R. 1111  
31 B. E. R. H. M. B. I. E. A. 1111

# DICTIONARY OF MEDICINE

VOL. I.

Digitized by the Internet Archive  
in 2015

A

LIBRARY OF THE  
MEDICAL SOCIETY

# DICTIONARY OF MEDICINE

INCLUDING GENERAL PATHOLOGY, GENERAL THERAPEUTICS  
HYGIENE AND THE DISEASES OF WOMEN AND CHILDREN

BY VARIOUS WRITERS

EDITED BY

RICHARD QUAIN, BART., M.D. LOND., LL.D. ED., F.R.S.

President of the General Council of Medical Education: Member of the Senate of the University of London  
Hon. M.D. Trin. Coll. Dublin: Hon. M.D. Royal University of Ireland  
Fellow, late Vice-President and Senior Censor of the Royal College of Physicians  
Hon. Fellow of the Royal College of Physicians of Ireland  
Physician Extraordinary to Her Majesty the Queen  
Consulting Physician to the Hospital for Diseases of the Chest, Brompton  
and to the Seamen's Hospital, Greenwich

ASSISTED BY

FREDERICK THOMAS ROBERTS, M.D. LOND., B.Sc.

Fellow of the Royal College of Physicians: Fellow of University College  
Professor of Materia Medica and Therapeutics, University College  
Physician and Professor of Clinical Medicine, University College Hospital  
Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton

AND

J. MITCHELL BRUCE, M.A. ABDN., M.D. LOND.

Fellow of the Royal College of Physicians  
Physician and Lecturer on the Principles and Practice of Medicine, Charing Cross Hospital  
Physician to the Hospital for Consumption and Diseases of the Chest, Brompton

NEW EDITION, REVISED THROUGHOUT AND ENLARGED

IN TWO VOLUMES

VOL. I. : ABDOMEN—LYSIS

LONDON

LONGMANS, GREEN, AND CO.

1894

UNIVERSITY OF LEEDS  
MEDICAL LIBRARY

UNIVERSITY OF LEEDS  
MEDICAL LIBRARY.

603187

# P R E F A C E

1894

THE Preface to the original issue of this Dictionary, which will be found at page vii of the present volume, contains a statement of the motive which originally induced the Editor to enter on the preparation of the work. Briefly, that motive was supplied by a desire to place in the hands of the practitioner, the teacher, and the student, a means of ready reference to the accumulated knowledge which we possessed of scientific and practical Medicine, rapid as was its progress, and difficult of access as were its scattered records.

That the object in view was successfully attained, has been rendered abundantly evident by the reception accorded to the Dictionary. Since its appearance in 1882 more than 33,000 copies have been issued in this country and America, whilst the Editor personally has received most gratifying assurances of the value and utility of the work.

Having made this reference to the past, he has now to say that the motive which then influenced him is felt with greatly increased force at the present time. Never in the history of Medicine has progress been so rapid as during the last twelve years. Not only has our knowledge of old and familiar facts been improved, but an entirely new development of our science has occurred, more especially with reference to the causation of disease and, it may be added, the preservation of health. It had therefore become necessary to make a complete revision of the work. In the performance of this duty, it has been the aim of the Editor to combine what was good in the original, amended as experience suggested, with such new matter as proved to be acceptable after full consideration.

In order to accomplish these objects, the several articles have been revised by the original writers, so far as this has been practicable. When death unhappily prevented such revision, the task was committed to other not less competent writers. The services of equally competent contributors

were secured for the preparation of new articles. To these gentlemen, friends and colleagues, the Editor offers his grateful thanks for the ability and zeal with which they have discharged the duties undertaken by them. He has the gratification of feeling that the contributors to the Dictionary may regard with great satisfaction the result of their labours.

The Editor has also to speak with sincere thanks of the valuable services which have been rendered him by his fellow-labourers, Dr. FREDERICK T. ROBERTS and Dr. J. MITCHELL BRUCE. In the Preface to the former Edition he stated that without the help afforded by them he would have found it impossible to accomplish the undertaking. He appreciated then the marked ability by which their assistance had been characterised, and he desires now to repeat the expression of his appreciation, recognising that their services have been rendered of much greater value by their increased experience. He has on these grounds felt it a pleasing duty to place the names of these gentlemen on the title-page as his Assistant-Editors. At the same time he feels it right to say that he is personally responsible for the work, of which no portion has been issued without being carefully revised by him. He has also heartily to thank his friend Mr. JOHN HAROLD, Medical Registrar to Charing Cross Hospital, not only for his contributions to the Dictionary, and for the assistance which he has rendered in revising the proof-sheets and passing the work through the press, but also for the pains which he has bestowed upon the conduct of the necessary correspondence with various writers, and in collecting the materials.

It only remains to state that the work, consisting now of two volumes, has been entirely reprinted in larger type, that the number of pages has been increased from 1,834 to 2,518, and that 43 new illustrations have been added to those in the former Edition, so that the illustrations now amount to 181.

# P R E F A C E

1882

THE VAST NUMBER of facts and observations, by which the recent progress of scientific and practical medicine has been marked, is diffusely recorded in the Transactions of learned societies, in journals, in monographs, and in systematic treatises. With progress so rapid, and information so diffused, it is extremely difficult alike for the practitioner, the teacher, and the student to keep pace. It was the perception of this difficulty which induced the Editor, when invited to undertake the production of a new Medical Dictionary, to engage in a task which, he was fully conscious, must be one of great labour and of great responsibility. He felt, however, that he would be rendering useful service to his Profession if he could bring together the latest and most complete information in a form which would allow of ready and easy reference. Accordingly, he invited the co-operation of certain of his colleagues and professional friends, both in this country and abroad; and evidence of the readiness with which this invitation has been accepted, is afforded by the list of contributors. Each contributor volunteered or was invited to write on a subject with which he was specially familiar. The present work, which is the result of these combined efforts, may therefore be regarded not only as a dictionary, but also as a treatise on systematic medicine, in which the articles on the more important subjects constitute monographs in themselves, whilst definitions and descriptions of matters having less claim to extended notice are given as fully as is required. Thus an endeavour has been made to supply, in a clear, condensed, and readily accessible form, all the information that is at present available for the use of the practitioner of medicine.

As indicated on the title-page, the work is primarily a dictionary of Medicine, in which the several diseases are fully discussed in alphabetical order. The description of each includes an account of its ætiology and anatomical characters; its symptoms, course, duration, and terminations; its diagnosis, prognosis, and, lastly, its treatment. General Pathology comprehends articles on the origin, characters, and nature of disease, and

the many considerations which these topics suggest. General Therapeutics will be found to include articles on the several classes of remedies—medicinal or otherwise—which are available for the purpose of treatment; on the modes of action of such remedies; and on the methods of their use. The articles devoted to the subject of Hygiene will be found to treat of the causes of disease, of its prevention, of the agencies and laws affecting public health, of the means of preserving the health of the individual, of the construction and management of hospitals, and of the nursing the sick. Lastly, the diseases peculiar to Women and Children are discussed under their respective headings, both in aggregate and in detail.

It may be well to explain that, although it has been found necessary to include some notice of diseases which fall more generally under the care of the surgeon, the work does not pretend to be a dictionary of Surgery; and also that, although certain drugs are enumerated in discussing subjects of general therapeutics, and of poisons and their actions, there has been no intention to invade the domain of *Materia Medica*.

It is right to observe that all the articles have been edited and revised with great care, so as to ensure a completeness and unity in the work, which it is not always possible to obtain in books composed by a number of writers.

The Editor desires also to state that, although the work has occupied several years in preparation, arrangements were made with the printers which have enabled him to revise every article which required revision, up to the time of going to press. Further, by the addition of an Appendix, it has been possible to incorporate the latest contributions to medical knowledge.

Having thus set forth the aims and objects of his undertaking, and how far they have been carried out, the Editor has the great satisfaction of offering his thanks to his friends and colleagues, for the valuable assistance which he has received from them. He is fully conscious of the trouble which must often have been necessary in order to condense extended knowledge of a familiar subject within the limited space which the nature of this work could afford.

The Editor has further the pleasing duty of offering his special thanks to Dr. FREDERICK T. ROBERTS and to Dr. J. MITCHELL BRUCE, who from the

first have been his Assistant-Editors and fellow-labourers. Without the help which they have afforded him, it would have been impossible for him to have fulfilled the duties which he undertook. He is well aware of the time and labour which their assistance has involved; and he appreciates most fully the marked ability by which it has been characterised.

The Editor cannot conclude without a reference to some of those who were his friends and colleagues when this work was commenced, but who have since been taken away by death. He would specially mention the names of EDMUND PARKES, CHARLES MURCHISON, GEORGE CALLENDER, THOMAS BEVILL PEACOCK, JOHN ROSE CORMACK, LOCKHART CLARKE, TILBURY FOX, THOMAS HAYDEN, HARRY LEACH, ALEXANDER SILVER. The loss of these eminent men, many of them dear and valued friends, and all of them taken too soon from their unfinished labours, is to him a source of personal sorrow. The articles written by them for these pages were in most instances their last contributions to medical literature, and will be valued accordingly.

LONDON : *September* 1882.



PLoS ONE  
 10.1371/journal.pone.0081111

# LIST OF ILLUSTRATIONS

## IN VOLUME I

FIGURE	PAGE	FIGURE	PAGE
1. Actinomycosis . . . . .	17	19. Adenoid of breast (common type) <i>to face</i>	270
2. Retinal Hæmorrhage in Pernicious Anæmia . . . . .	60	20. Ulcerated Adenoid of parotid (malignant) . . . . .	270
3. Microscopic appearances of the red blood - corpuscles in Pernicious Anæmia . . . . .	61	21. Adenoid of breast (epithelial element in excess) . . . . .	270
4. Section of the liver in Pernicious Anæmia after treatment with ferrocyanide of potassium and hydrochloric acid . . . . .	62	22. Adenoid of breast (Adeno-sarcoma) ,,	270
5. Aphasia. Diagram showing the commissural connexions between the different Word-centres . . . . .	100	23. Cardiogram . . . . .	277
6. Diagram showing the possible location of an Aphasia-producing lesion . . . . .	101	24. Renal casts—blood- . . . . .	280
7. Papilloma of soft palate . . . . .	<i>to face</i> 270	25. „ hyaline . . . . .	280
8. Epithelioma of lip . . . . .	270	26. „ epithelial . . . . .	280
9. Edge of Rodent Ulcer . . . . .	270	27. „ fatty . . . . .	281
10. Simple Polypus of rectum . . . . .	270	28. „ granular . . . . .	281
11. Columnar Epithelioma of intestine ,,	270	29. „ enclosing crystals, and a smaller cast; also of seminal tubule with spermatozoa . . . . .	281
12. Colloid of breast . . . . .	270	30. Side view of the Left Hemisphere of the Monkey . . . . .	394
13. Cancer of liver (Scirrhus-encephaloid) . . . . .	270	31. Side view of the Left Hemisphere in Man . . . . .	394
14. Scirrhus infiltrating fat . . . . .	270	32. <i>Cysticercus (telæ) cellulosæ</i> , removed from the human eye . . . . .	580
15. Encephaloid Cancer . . . . .	270	33. Unarmed head of <i>Tænia mediocanellata</i> . . . . .	580
16. Cicatrising Cancer . . . . .	270	34. Armed head of <i>Tænia solium</i> . . . . .	580
17. Scirrhus of mamma . . . . .	270	35. Proglottis of <i>Tænia mediocanellata</i> . . . . .	581
18. Adenoid of upper jaw (benign) . . . . .	270	36. Proglottis of <i>Tænia solium</i> . . . . .	581
		37. <i>Tænia echinococcus</i> . . . . .	581

FIGURE	PAGE	FIGURE	PAGE
38. Hydatid of four weeks' growth, showing ectocyst and endocyst . . . . .	582	53. <i>Filaria sanguinis hominis perstans</i> . . . . .	697
39. The so-called Echinococcus head, showing hooks, suckers, cilia, and corpuscles . . . . .	582	54. <i>Filaria sanguinis hominis nocturna</i> . . . . .	698
40. Group of Echinococci, with their hook-crowns inverted . . . . .	582	55. Anterior end of mature <i>Filaria sanguinis hominis nocturna</i> . . . . .	698
41. <i>Bilharzia hæmatobia</i> , male and female sexually combined . . . . .	584	56. Portion of mature <i>Filaria sanguinis hominis nocturna</i> , showing uterine tubules filled with ova in various stages of development; also the intestinal tube . . . . .	698
42. Ovum of <i>Bilharzia hæmatobia</i> , with contained embryo and free sarcodengranules . . . . .	584	57. Ova and embryos of <i>Filaria sanguinis hominis nocturna</i> . . . . .	698
43. <i>Ascaris lumbricoides</i> , male with exerted spicules . . . . .	585	58. Fungoid filaments and capsules obtained after prolonged maceration in caustic potash of the dark masses of Fungus-disease of India . . . . .	711
44. <i>Oxyuris vermicularis</i> , female . . . . .	585	59. Connective-tissue Hypertrophy of the Heart . . . . .	794
45. Eggs of <i>Oxyuris vermicularis</i> , enclosing tadpole-shaped embryos . . . . .	585	60. Fatty Degeneration of the Heart . . . . .	804
46. <i>Trichocephalus</i> , male (a) and female (b) . . . . .	586	61. Fatty Growth in the substance of the Heart . . . . .	808
47. <i>Ankylostoma duodenale</i> , male (a) and female (b) . . . . .	586	62. Pulse-tracing in aortic stenosis . . . . .	848
48. <i>Filaria medinensis</i> . . . . .	586	63. Pulse-tracing in aortic insufficiency . . . . .	849
49. <i>Trichina spiralis</i> , male (a) and female (b) . . . . .	587	64. Pulse-tracing in mitral stenosis . . . . .	850
50. Portion of human muscle, enclosing a single capsuled <i>Trichina</i> . . . . .	587	65. Pulse-tracing in mitral insufficiency . . . . .	850
51. <i>Filaria sanguinis hominis diurna</i> . . . . .	697	66. Crystals of Leucin . . . . .	1101
52. <i>Filaria sanguinis hominis nocturna</i> . . . . .	697	67. Blood in Leucocythæmia . . . . .	1103

# LIST OF CONTRIBUTORS

1882

- ADAMS, WILLIAM, Consulting Surgeon to the Great Northern Central Hospital.
- AITKEN, The late SIR WILLIAM, M.D., LL.D., F.R.S., Professor of Pathology in the Army Medical School, Netley.
- ALLBUTT, T. CLIFFORD, M.A., M.D., LL.D., F.R.S., Regius Professor of Physic in the University of Cambridge; Consulting Physician to the Leeds General Infirmary.
- ALLCHIN, WILLIAM H., M.D., F.R.S.E., Physician to, and late Joint Lecturer on the Principles and Practice of Medicine at, the Westminster Hospital; late Physician to the Victoria Hospital for Children.
- ANDREW, JAMES, M.D., late Physician to, and Joint Lecturer on Physic at, St. Bartholomew's Hospital; Consulting Physician to the City of London Hospital for Diseases of the Chest.
- BALFOUR, GEORGE W., M.D., F.R.S.E., Consulting Physician to the Royal Infirmary, and to the Royal Hospital for Children, Edinburgh.
- BANHAM, G. A., late Veterinary Assistant at the Brown Institution.
- BARNES, ROBERT, M.D., Consulting Obstetric Physician to, and late Lecturer on Midwifery and Diseases of Women at, St. George's Hospital; Consulting Physician to the Seamen's Hospital, and to the Royal Maternity Charity.
- BASTIAN, H. CHARLTON, M.A., M.D., F.R.S., Physician to University College Hospital, Professor of the Principles and Practice of Medicine and of Clinical Medicine, and late Professor of Pathological Anatomy, at University College; Physician to the National Hospital for the Paralysed and Epileptic.
- BÄUMLER, C. G. H., M.D., Professor of Clinical Medicine, and Director of the Medical Clinic, University of Freiburg in Baden.
- BECK, The late MARCUS, M.B., M.S., Surgeon to University College Hospital, and Professor of Surgery at University College.
- BEDDOE, JOHN, B.A., M.D., F.R.S., late Physician to the Bristol Royal Infirmary.
- BELLAMY, The late EDWARD, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital.
- BENNET, The late J. HENRY, M.D., formerly Physician-Accoucheur, Royal Free Hospital.

- BENNETT, The late SIR J. RISDON, M.D., LL.D., F.R.S., President of the Royal College of Physicians; Consulting Physician to St. Thomas's Hospital, and to the City of London Hospital for Diseases of the Chest.
- BEVERIDGE, The late ROBERT, M.B., Physician to, and Lecturer on Clinical Medicine at, the Aberdeen Royal Infirmary.
- BINZ, CARL, M.D., Professor of Pharmacology in the University of Bonn.
- BIRKETT, JOHN, Consulting Surgeon to Guy's Hospital.
- BISHOP, The late JOHN, M.D., C.M., Assistant Surgeon to the Royal Infirmary, Edinburgh.
- BLANDFORD, GEORGE F., M.A., M.D., Lecturer on Psychological Medicine at St. George's Hospital.
- BOWLES, R. LEAMON, M.D., Consulting Physician to the Folkestone Hospital.
- BRISTOWE, J. SYER, M.D., LL.D., F.R.S., Consulting Physician to, and late Joint Lecturer on Medicine at, St. Thomas's Hospital.
- BROADBENT, SIR WILLIAM H., Bart., M.D., Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital.
- BROWN-SÉQUARD, The late C. E., M.D., LL.D., F.R.S., Professor of Medicine, Collège de France.
- BRUCE, J. MITCHELL, M.A., M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Charing Cross Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.
- BRUCE, WILLIAM, M.A., LL.D., M.D., Senior Surgeon to the Ross Memorial Hospital, Dingwall.
- BRUNTON, T. LAUDER, M.D., LL.D., D.Sc., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital.
- BUCHANAN, SIR GEORGE, B.A., LL.D., M.D., F.R.S., late Medical Officer of H.M. Local Government Board; Consulting Physician to the London Fever Hospital.
- BUTLIN, H. TRENTHAM, D.C.L., Surgeon to St. Bartholomew's Hospital.
- BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic.
- CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital.
- CALLENDER, The late G. W., F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital.
- CANTLIE, JAMES, M.A., M.B., C.M., late Senior Assistant Surgeon to, and Demonstrator of Anatomy at, the Charing Cross Hospital.
- CARPENTER, The late W. B., C.B., M.D., LL.D., F.R.S.
- CARTER, R. BRUDENELL, Consulting Ophthalmic Surgeon to, and late Lecturer on Ophthalmic Surgery at, St. George's Hospital.

- CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Consulting Physician to the London Fever Hospital.
- CLARKE, The late J. LOCKHART, M.D., F.R.S., Physician to the Hospital for Epilepsy and Paralysis.
- CLARKE, The late W. FAIRLIE, M.A., M.D., Assistant Surgeon to the Charing Cross Hospital.
- CLOVER, The late J. T., Lecturer on Anæsthetics at University College Hospital.
- COBBOLD, CHARLES S. W., M.D., late Medical Superintendent, Asylum for Idiots, Earlswood; late Senior Assistant Medical Officer, Middlesex County Asylum, Colney Hatch.
- COBBOLD, The late T. SPENCER, M.D., F.R.S.
- COLLIE, ALEXANDER, M.D., late Medical Officer, Fever Hospital, Homerton.
- COOPER, ARTHUR, Surgeon to the Westminster General Dispensary; late House Surgeon to the Male Lock Hospital.
- CORMACK, The late SIR JOHN ROSE, K.C.B., M.D., F.R.S.E., Physician to the Hertford British Hospital, Paris.
- CUNNINGHAM, D. DOUGLAS, C.I.E., M.B., C.M., Brigade Surgeon-Lieutenant-Colonel H.M. Bengal Army.
- CURLING, The late T. B., F.R.S., Consulting Surgeon to the London Hospital.
- CURNOW, JOHN, M.D., Physician to King's College Hospital; Professor of Anatomy in King's College; Senior Visiting Physician to the Seamen's Hospital, Greenwich.
- DALBY, SIR WILLIAM B., B.A., M.B., Consulting Aural Surgeon to, and late Lecturer on Aural Surgery at, St. George's Hospital.
- DAVIDSON, ALEXANDER, M.A., M.D., Physician to the Royal Infirmary, Liverpool, and Emeritus Professor of Pathology in University College, Liverpool.
- DE ZOUICHE, ISAIAH, M.D., Honorary Physician to the Dunedin Hospital, New Zealand.
- DUNCAN, The late J. MATTHEWS, M.A., M.D., LL.D., F.R.S.E., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital.
- DURHAM, ARTHUR E., late Senior Surgeon to, and Lecturer on Surgery at, Guy's Hospital.
- ECHEVERRIA, M. G., M.D., late Physician-in-Chief to the Hospital for Epileptics and Paralytics, and to the City Asylum for the Insane, New York.
- EWART, JOSEPH, M.D., Retired Deputy Surgeon-General H.M. Bengal Army; late Professor of Medicine, Principal, and Senior Physician, Calcutta Medical College.
- EWART, WILLIAM, B.A., M.D., Physician to St. George's Hospital; late Assistant Physician and Pathologist to the Hospital for Consumption and Diseases of the Chest, Brompton.
- FARQUHARSON, ROBERT, M.D., M.P., late Physician to the Belgrave Hospital for Children; late Assistant Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital.

- FAYRER, SIR JOSEPH, K.C.S.I., M.D., LL.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales; President of the Medical Board, India Office; Consulting Physician to the Charing Cross Hospital.
- FENWICK, SAMUEL, M.D., Physician to, and late Lecturer on Medicine at, the London Hospital; late Assistant Physician to the City of London Hospital for Diseases of the Chest.
- FERRIER, DAVID, M.A., M.D., LL.D., F.R.S., Physician to King's College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; Professor of Neuropathology in King's College.
- FINNEY, J. MAGEE, B.A., M.D., Physician to the City of Dublin Hospital; King's Professor of the Practice of Medicine at the School of Physic in Ireland, and Professor of Clinical Medicine in Sir Patrick Dun's Hospital.
- FOSTER, SIR WALTER, M.D., D.C.L., M.P., Consulting Physician to the General Hospital, and Emeritus Professor of the Principles and Practice of Physic in Queen's College, Birmingham.
- FOX, E. LONG, M.D., Consulting Physician to the Bristol Royal Infirmary, and late Lecturer on the Principles and Practice of Medicine at the Bristol School of Medicine.
- FOX, T. COLCOTT, B.A., M.B., Physician for Diseases of the Skin, Westminster Hospital; late Physician to the Victoria Hospital for Children.
- FOX, The late TILBURY, M.D., Physician to the Skin Department, University College Hospital.
- GALTON, SIR DOUGLAS, Late Royal Engineers, K.C.B., D.C.L., F.R.S.
- GASCOYEN, The late GEORGE G., Surgeon to the Lock Hospital; Assistant Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital.
- GEE, SAMUEL JONES, M.D., Physician to St. Bartholomew's Hospital; Consulting Physician to the Hospital for Sick Children, Great Ormond Street.
- GODLEE, RICKMAN J., B.A., M.B., M.S., Surgeon to, and Professor of Clinical Surgery at, University College Hospital; Surgeon to the Hospital for Consumption and Diseases of the Chest, Brompton; Consulting Surgeon to the North-East Hospital for Children.
- GODSON, CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital; late Assistant Physician-Accoucheur to St. Bartholomew's Hospital.
- GOWERS, WILLIAM R., M.D., F.R.S., Consulting Physician to, and late Professor of Clinical Medicine at, University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic.
- GREEN, T. HENRY, M.D., Physician to, Special Lecturer on Clinical Medicine and late Lecturer on Pathology at, the Charing Cross Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.
- GREENFIELD, WILLIAM S., M.D., Professor of General Pathology and Clinical Medicine in the University of Edinburgh.
- GRIMSHAW, THOMAS W., M.A., M.D., Registrar-General for Ireland; Consulting Physician to the Fever Hospital, Cork Street, and to Steeven's Hospital, Dublin.

- HAWARD, J. WARRINGTON, Surgeon to St. George's Hospital; late Assistant Surgeon to the Hospital for Sick Children, Great Ormond Street.
- HAYDEN, The late THOMAS, M.D., Physician to the Mater Misericordiæ Hospital, Dublin; Professor of Anatomy and Physiology, Catholic University, Dublin.
- HERMAN, G. ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; Physician to the Royal Maternity Charity.
- HICKS, J. BRAXTON, M.D., F.R.S., late Physician-Accoucheur to, and Lecturer on Midwifery and Diseases of Women and Children at, Guy's Hospital.
- HILL, The late BERKELEY, M.B., Surgeon to, and Professor of Clinical Surgery at, University College Hospital; Surgeon to the Lock Hospital.
- HOLMES, TIMOTHY, M.A., Consulting Surgeon to, and late Lecturer on Surgery at, St. George's Hospital.
- HORSLEY, VICTOR A. H., M.B., B.S., F.R.S., Professor of Pathology in University College; Surgeon to University College Hospital, and to the National Hospital for the Paralysed and Epileptic.
- HOWARD, BENJAMIN, M.D., late Professor of Medicine, and Lecturer on Medicine, in the University of New York.
- HUTCHINSON, JONATHAN, LL.D., F.R.S., late President of the Royal College of Surgeons; Consulting Surgeon to the London Hospital, to the Hospital for Diseases of the Skin, Blackfriars, and to the Royal London Ophthalmic Hospital.
- IRVINE, The late J. PEARSON, B.A., B.Sc., M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Charing Cross Hospital; Physician to the Victoria Hospital for Children.
- JENNER, SIR WILLIAM, Bart., G.C.B., M.D., D.C.L., LL.D., F.R.S., Physician-in-Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; late President of the Royal College of Physicians; Consulting Physician to University College Hospital.
- JONES, JOSEPH, M.D., Professor of Chemistry and Clinical Medicine, New Orleans, and President of the Board of Health, State of Louisiana, New Orleans.
- LANGDON-DOWN, JOHN, M.D., Consulting Physician to, and late Lecturer on Clinical Medicine at, the London Hospital.
- LATHAM, PETER W., A.M., M.D., Senior Physician to Addenbrooke's Hospital; late Downing Professor of Medicine in the University of Cambridge.
- LEACH, The late HARRY, Medical Officer of Health for the Port of London, and Physician to the Seamen's Hospital, Greenwich.
- LEGG, J. WICKHAM, M.D., late Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Bartholomew's Hospital.
- LEWIS, The late TIMOTHY, M.D., F.R.S. (Elect), Surgeon-Major H.M. Army.
- LITTLE, JAMES, M.D., Physician to the Adelaide Hospital, Dublin; late Professor of Practice of Medicine in the Royal College of Surgeons in Ireland; Consulting Physician to the Rotunda Lying-in Hospital.

- LIVEING, ROBERT, M.A., M.D., late Physician for Diseases of the Skin, and Lecturer on Diseases of the Skin, at the Middlesex Hospital.
- McCARTHY, JEREMIAH, M.A., M.B., Surgeon to, and late Lecturer on Surgery at, the London Hospital.
- MACCORMAC, SIR WILLIAM, M.A., M.Ch., Consulting Surgeon to, and late Lecturer on Surgery at, St. Thomas's Hospital.
- McKENDRICK, J. GRAY, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Glasgow.
- MACKENZIE, STEPHEN, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the London Hospital.
- MACLEAN, Surgeon-General W. CAMPBELL, C.B., M.D., Inspector-General of Hospitals; late Professor of Military Medicine in the Army Medical School, Netley.
- MACNAMARA, CHARLES NOTTIDGE, Surgeon to the Westminster Hospital; Consulting Surgeon to the Royal Westminster Ophthalmic Hospital.
- MACPHERSON, The late JOHN, M.A., M.D., Inspector-General of Hospitals, H.M. Bengal Army.
- MADDEN, T. MORE, M.D., Obstetric Physician to the Mater Misericordiæ Hospital, Dublin.
- MANSON, PATRICK, M.D., LL.D., Physician to the Seamen's Hospital, Greenwich; late Lecturer on Medicine in the College of Medicine for the Chinese, Hong Kong.
- MEREDITH, W. APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children.
- MERYON, The late EDWARD, M.D., Physician to the Hospital for Epilepsy and Paralysis.
- MUIRHEAD, CLAUD, M.D., Consulting Physician to, and formerly Lecturer on Clinical Medicine at, the Royal Infirmary, Edinburgh.
- MURCHISON, The late CHARLES, M.D., LL.D., F.R.S., Physician to, and Special Professor of Clinical Medicine at, St. Thomas's Hospital; Consulting Physician to the London Fever Hospital.
- MYERS, ARTHUR B. R., late Brigade-Surgeon, Brigade of Guards.
- NETTLESHIP, EDWARD, Ophthalmic Surgeon to St. Thomas's Hospital, and to the Royal London Ophthalmic Hospital; late Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street.
- NIGHTINGALE, FLORENCE.
- OLIVER, GEORGE, M.D., Harrogate.
- ORD, W. MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital.
- PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital.
- PARKES, The late EDMUND A., M.D., F.R.S., Professor of Hygiene in the Army Medical School, Netley.

- PAVY, FREDERICK W., M.D., LL.D., F.R.S., Consulting Physician to, and late Lecturer on Medicine at, Guy's Hospital.
- PAYNE, J. FRANK, B.A., B.Sc., M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital.
- PEACOCK, The late T. BEVILL, M.D., Honorary Consulting Physician to St. Thomas's Hospital; and Consulting Physician to the City of London Hospital for Diseases of the Chest.
- PLAYFAIR, WILLIAM S., M.D., LL.D., Physician-Accoucheur to H.I. and R.H. the Duchess of Edinburgh; Physician for Diseases of Women and Children to King's College Hospital, and Consulting Physician to the General Lying-in Hospital; Professor of Obstetric Medicine in King's College.
- POORE, G. VIVIAN, M.D., Physician to, and Professor of Clinical Medicine at, University College Hospital; Professor of Medical Jurisprudence at University College.
- POWELL, R. DOUGLAS, M.D., Physician Extraordinary to H.M. the Queen; Physician to the Middlesex Hospital, and Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.
- QUAIN, SIR RICHARD, Bart., M.D., LL.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton, and to the Seamen's Hospital, Greenwich.
- RADCLIFFE, The late J. NETTEN, Assistant Medical Officer, Local Government Board.
- RALFE, CHARLES H., M.A., M.D., Physician to the London Hospital; late Physician to the Seamen's Hospital, Greenwich.
- REDWOOD, The late RALFE THEOPHILUS, Ph.D., Professor of Chemistry and Pharmacy, Pharmaceutical Society of Great Britain.
- ROBERTS, FREDERICK T., M.D., B.Sc., Physician to, and Professor of Clinical Medicine at, University College Hospital; Professor of Materia Medica at University College; Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.
- ROBERTS, SIR WILLIAM, B.A., M.D., F.R.S., late Physician to the Manchester Royal Infirmary; late Professor of Medicine in the Victoria University.
- ROSE, WILLIAM, M.B., B.S., Surgeon to King's College Hospital; Professor of Clinical Surgery, King's College; Consulting Surgeon to the Royal Free Hospital.
- ROY, CHARLES S., M.A., M.D., F.R.S., Professor of Pathology, University of Cambridge; late Professor Superintendent of the Brown Institution, London.
- RUSSELL, The Right Hon. JAMES A., M.A., M.B., C.M., Inspector of Anatomy for Scotland; late Lecturer on Sanitation, Watt's Institution, Edinburgh.
- SALTER, S. J., M.B., F.R.S., F.L.S., late Dental Surgeon to Guy's Hospital.
- SANGSTER, ALFRED, B.A., M.B., Physician for Diseases of the Skin to, and Lecturer on Skin Diseases at, the Charing Cross Hospital.
- SAUNDBY, ROBERT, M.D., Physician to the General Hospital, and Professor of Medicine in Mason College, Birmingham.

- SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology in University College.
- SEATON, The late EDWARD C., M.D., Medical Officer to the Local Government Board.
- SHAPTER, THOMAS, M.D., LL.D., Consulting Physician to the Devon and Exeter Hospital.
- SIBBALD, JOHN, M.D., F.R.S.E., Commissioner in Lunacy for Scotland.
- SILVER, The late ALEXANDER, M.A., M.D., Physician to, and Lecturer on Physiology at, the Charing Cross Hospital.
- SIMON, SIR JOHN, C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; late Medical Officer to Her Majesty's Privy Council, and to the Local Government Board.
- SIMPSON, ALEXANDER RUSSELL, M.D., Physician to the University Clinical Ward for Diseases of Women, Royal Infirmary, Edinburgh; Professor of Midwifery and Diseases of Women and Children in the University of Edinburgh.
- SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the City of London Hospital for Diseases of the Chest, and to the East London Hospital for Children.
- SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.
- SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; late Physician to, and Lecturer on Forensic Medicine and Hygiene at, St. Bartholomew's Hospital.
- SPARKS, The late EDWARD I., M.A., M.B., Physician for Diseases of the Skin to the Charing Cross Hospital, and Physician to the Royal Infirmary for Women and Children.
- SQUIRE, WILLIAM, M.D., Physician to the St. George's Dispensary, and late Physician to the North London Hospital for Diseases of the Chest.
- STEVENSON, THOMAS, M.D., Lecturer on Chemistry and Medical Jurisprudence at Guy's Hospital; Scientific Analyst to the Home Office.
- STEWART, T. GRAINGER, M.D., F.R.S.E., Physician in Ordinary to H.M. the Queen for Scotland; Professor of Practice of Physic and of Clinical Medicine in the University of Edinburgh.
- STREATFEILD, The late J. F., Surgeon to the Royal London Ophthalmic Hospital; Professor of Clinical Ophthalmic Surgery at, and Ophthalmic Surgeon to, University College Hospital.
- THIN, GEORGE, M.D., London.
- THOMPSON, E. SYMES, M.D., Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.
- THOMPSON, SIR HENRY, Surgeon Extraordinary to H.M. the King of the Belgians; Consulting Surgeon to University College Hospital; Emeritus Professor of Clinical Surgery at University College.
- THORNTON, W. PUGIN, late Surgeon to the St. Marylebone General Dispensary.
- THOROWGOOD, JOHN C., M.D., Physician to the City of London Hospital for Diseases of the Chest, and to the West London Hospital; late Lecturer on Materia Medica at the Middlesex Hospital.

- TUKE, J. BATTY, M.D., F.R.S.E., formerly Lecturer on Mental Diseases at the Royal College of Surgeons, Edinburgh.
- WALKER, THOMAS J., M.D., Surgeon to the Peterborough Infirmary and Dispensary.
- WARD, The late STEPHEN H., M.D., Consulting Physician to the Seamen's Hospital, Greenwich; and Physician to the City of London Hospital for Diseases of the Chest.
- WARDELL, The late J. R., M.D., Consulting Physician to the Tunbridge Wells Infirmary.
- WATERS, A. T. H., M.D., Consulting Physician to the Royal Infirmary, Liverpool; late Professor of the Principles and Practice of Medicine in University College, Liverpool.
- WEBER, HERMANN, M.D., Consulting Physician to the German Hospital.
- WELLS, SIR T. SPENCER, Bart., late President of the Royal College of Surgeons; Surgeon to the Queen's Household; Consulting Surgeon to the Samaritan Hospital for Women and Children.
- WILLIAMS, C. THEODORE, M.A., M.D., Senior Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.
- WILLIAMS, JOHN, M.D., Consulting Obstetric Physician, University College Hospital.
- WILSON, The late SIR ERASMUS, LL.D., F.R.S., President of the Royal College of Surgeons; Professor of Dermatology, Royal College of Surgeons.
- WILTSHIRE, The late ALFRED, M.D., Physician-Accoucheur to, and Joint Lecturer on Obstetric Medicine at, St. Mary's Hospital; Physician for Diseases of Women to the West London Hospital.
- WOOD, The late JOHN, F.R.S., Surgeon to King's College Hospital, and Professor of Clinical Surgery in King's College.

---

## ADDITIONAL CONTRIBUTORS

1894

- ACKLAND, ROBERT CRAIG, Assistant Dental Surgeon to St. Bartholomew's Hospital.
- BAILEY, G. HEWLETT, Chloroformist to the Dental Hospital of London.
- BAKER, W. MORRANT, late Surgeon to St. Bartholomew's Hospital.
- BALL, JAMES BARRY, M.D., Assistant Physician, and Physician to the Department for Diseases of the Throat and Nose, to the West London Hospital.
- BANKS, SIR JOHN, K.C.B., M.D., D.Sc., LL.D., Physician in Ordinary to H.M. the Queen in Ireland; Regius Professor of Physic in the University of Dublin; Consulting Physician to Sir Patrick Dun's Hospital.

- BOWLBY, ANTHONY A., Assistant Surgeon, and Surgeon in Charge of the Throat Department, to St. Bartholomew's Hospital.
- BOYD, STANLEY, M.B., B.S., Surgeon to, and Lecturer on Anatomy at, the Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital.
- BRUCE, DAVID, M.B., C.M., Surgeon-Captain, H.M. Army.
- CASSON, WILLIAM A., Barrister-at-Law, Local Government Board.
- CHARTERIS, MATTHEW, M.D., Professor of Materia Medica and Therapeutics in the University of Glasgow.
- CRAIGIE, J. HAMILTON, Surgeon Dentist to the Chelsea Hospital for Women.
- CROCKER, H. RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital.
- CURRIE, ANDREW STARK, M.D., late Assistant to the Professor of Physiology in the University of Glasgow.
- FENWICK, W. SOLTAU, M.D., B.S., Physician for Out-Patients to the Evelina Hospital for Sick Children.
- GERVIS, HENRY, M.D., Consulting Obstetric Physician to St. Thomas's Hospital.
- GOODHART, JAMES F., M.D., Physician to, and late Lecturer on Pathology at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children.
- HAROLD, JOHN, Medical Registrar to the Charing Cross Hospital.
- HEBB, RICHARD GRAINGER, M.A., M.D., Assistant Physician and Pathologist to, and Lecturer on Pathology and Morbid Anatomy at, the Westminster Hospital.
- JACOBSON, WALTER H. A., M.A., M.B., M.Ch., Assistant Surgeon to, and Lecturer on Anatomy at, Guy's Hospital.
- KIDD, PERCY, M.A., M.D., Assistant Physician and Pathologist to the London Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.
- LITTLE, JOHN FLETCHER, M.B., Physician to the North London Hospital for Consumption.
- LUFF, ARTHUR PEARSON, M.D., B.Sc., Physician for Out-Patients to, and Lecturer on Medical Jurisprudence at, St. Mary's Hospital; Official Analyst to the Home Office.
- McKEE, ALEXANDER BAILLIE, B.A., M.B., B.Ch., late Curator of the Museum, Royal College of Surgeons, Ireland.
- MAC MUNN, CHARLES A., M.A., M.D., Pathologist to the Wolverhampton and Staffordshire General Hospital.
- MAGUIRE, ROBERT, M.D., Physician for Out-Patients to, and Joint Lecturer on Pathology at, St. Mary's Hospital; Assistant Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.

- MAPOTHER, E. DILLON, M.D., late President of the Royal College of Surgeons of Ireland.
- MARTIN, SIDNEY H. C., M.D., B.Sc., Assistant Physician to University College Hospital, and to the Hospital for Consumption and Diseases of the Chest, Brompton.
- MORGAN, The late JOHN EDWARD, M.A., M.D., Consulting Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Victoria University.
- MOTT, FREDERICK W., M.D., B.S., Assistant Physician to, and Lecturer on Physiology at, the Charing Cross Hospital.
- MUIR, ROBERT, M.D., C.M., Senior Demonstrator of Pathology in the University of Edinburgh.
- MURPHY, J. SHIRLEY FORSTER, Medical Officer of Health of the Administrative County of London.
- MURRAY, H. MONTAGUE, M.D., Physician for Out-Patients to the Charing Cross Hospital, and to the Victoria Hospital for Children. Lecturer on Pathology and Morbid Anatomy at the Charing Cross Hospital.
- NIXON, CHRISTOPHER J., B.A., M.D., LL.D., Physician to the Mater Misericordiæ Hospital, Dublin.
- NORTON, ARTHUR T., Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital.
- POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.
- PRINGLE, JOHN J., M.B., C.M., Assistant Physician, Lecturer on Practical Medicine, and Physician to the Skin Department, at the Middlesex Hospital.
- RUSSELL, JAMES S. RISIEN, M.B., C.M., London.
- SANSOM, ARTHUR ERNEST, M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; Consulting Physician to the North-East Hospital for Children.
- SEATON, EDWARD, M.D., Lecturer on Public Health at St. Thomas's Hospital; Medical Officer of Health to the Administrative County of Surrey.
- STONHAM, CHARLES, Assistant Surgeon to, and Lecturer on Operative Surgery at, the Westminster Hospital.
- SUTTON, JOHN BLAND, Assistant-Surgeon to the Middlesex Hospital.
- TAYLOR, FREDERICK, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children.
- TAYLOR, JAMES, M.A., M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic.
- TREVES, FREDERICK, Surgeon to, and Lecturer on Surgery at, the London Hospital.
- TURNER, SIR WILLIAM, M.B., D.Sc., LL.D., D.C.L., F.R.S., Professor of Anatomy in the University of Edinburgh.

TWEEDY, JOHN, Professor of Ophthalmic Medicine and Surgery in University College; Ophthalmic Surgeon to University College Hospital; Surgeon to the Royal London Ophthalmic Hospital.

WHISTLER, W. MACNEILL, M.D., Physician to the London Throat Hospital.

WHITELEGGE, BENJAMIN A., M.D., B.Sc., D.P.H., County Medical Officer of Health, West Riding of Yorkshire; Lecturer on Public Health at the Charing Cross Hospital.

WILLIAMS, DAWSON, M.D., B.S., Assistant Physician to the East London Hospital for Children.

WRIGHT, ALMROTH EDWARD, B.A., M.D., B.Ch., Professor of Pathology in the Army Medical School, Netley.

105 3 WEST BOUNDING  
J. C. - SURGICAL BOOKS

# DICTIONARY OF M E D I C I N E

## A

**ABDOMEN, Diseases of the.**—Before entering upon the study of the particular diseases which are liable to be met with in connexion with each of the principal regions of the body, it is expedient to regard them from a general point of view, as such a course helps materially in clearing the way for their clinical investigation. This general survey is particularly advantageous in the case of abdominal diseases, which are necessarily very numerous and varied, both as regards the structures affected and the nature of the morbid changes they present; they are consequently difficult to recognise with certainty in many instances, and are occasionally involved in much obscurity.

Excluding a few peculiar affections, the diseases of the abdomen may be arranged under the following groups:—

I. Diseases of the anterior abdominal walls.

II. Diseases of the peritoneum and its folds.

III. Diseases of the organs contained within the abdominal cavity, namely:—

1. Stomach and Intestines; 2. Hepatic organs, including the liver, gall-bladder, and gall-ducts; 3. Spleen; 4. Pancreas; 5. Supra-renal capsules; 6. Urinary apparatus, viz., the kidneys and their ducts, and the bladder; 7. Female generative organs, including the uterus and its broad ligaments, the Fallopian tubes, and the ovaries; 8. Absorbent glands.

IV. Diseases of the abdominal vessels, especially the aorta and the iliac arteries.

V. Diseases of the sympathetic or other nerves contained within the abdomen.

VI. Diseases originating in connexion with the cellular tissue, such as inflammation or abscess.

VII. Diseases springing from the posterior boundary of the abdomen; from the pelvis or the structures lining it; or from the diaphragm, and invading the abdominal cavity.

VIII. Diseases encroaching upon the abdomen from other parts, especially from the thorax.

It must be borne in mind that the groups of diseases above mentioned may be presented in various combinations, two or more structures being not uncommonly implicated at the same time.

The special nature and mode of origin of the diseases thus summarised will be discussed under their appropriate headings, but a few general observations on this subject may prove serviceable. Several of the abdominal organs are very liable to so-called *functional disorders*, being much exposed to the repeated action of various disturbing influences, and these disorders often give rise to prominent and troublesome symptoms, which are urgently complained of by the patient. Definite *organic diseases* are also of common occurrence, many of them being of a very serious character. Some of the organs contained within the abdomen are liable to *malposition* or *displacement*, as well as to *malformations*, these being either congenital or acquired; while the hollow viscera may be the seat of *obstruction* or *accumulations* of different kinds. Each of these conditions may become clinically important.

Abdominal lesions are frequently purely *local* in their origin, but several of them are but local manifestations of some general condition, being associated either with certain acute febrile diseases, e.g. typhoid fever; or with some constitutional cachexia, such as cancer. Again, symptoms connected with the abdomen may depend upon disease in some remote part of the body; or some of its organs may become the seat of morbid changes as a consequence of disease in other structures. For instance, vomiting is frequently associated with cerebral disorders; while affections of the heart are liable to lead to troublesome symptoms, as well as to serious lesions, in connexion with many of the abdominal viscera. Lastly, a morbid condition of one organ within the abdomen may be the direct means of originating *secondary* mischief in other structures.

**CLINICAL INVESTIGATION.**—The clinical examination of cases in which the symptoms

point to the abdomen as the seat of mischief should always be conducted with particular care and thoroughness, as well as in a systematic manner, otherwise serious mistakes are liable to be made. It is also very desirable to avoid forming any definite conclusion as to the nature of the complaint hastily or on insufficient data, but rather to wait and observe the course of events in any doubtful case, repeating the investigation from time to time, when any obscurity which may exist will often be cleared away. The past and family history of the patient, with the course and progress of the symptoms, are often of material assistance in diagnosis, and demand due attention in every instance. The chief clinical phenomena which may be associated with abdominal affections, and with reference to which it is requisite to inquire, may be thus indicated. First, there are usually symptoms directly connected with the structure implicated, such as pain and other morbid sensations, disorders of secretory or other functions, or excited action. Secondly, several of the organs mutually affect each other, either from being anatomically or physiologically related, or from a morbid condition of one part causing pressure upon or irritation of some neighbouring structure. In this way numerous symptoms are liable to arise, sometimes in remote parts, and often of material significance. Thirdly, sympathetic or reflex phenomena in connexion with organs in other regions of the body are frequently excited by many abdominal disorders, such as palpitation of the heart, convulsions, and other nervous disturbances. Fourthly, the general system often suffers seriously, and in various ways. For instance, pyrexia may be excited; the blood may become impoverished or impregnated with noxious materials; or more or less general wasting and debility may be induced. Where an abdominal disease is but a local manifestation of some constitutional condition, it commonly aggravates materially the general symptoms; while in connexion with lesions of certain of the abdominal viscera these general symptoms constitute in many cases the most prominent clinical features. Fifthly, morbid conditions within the abdomen not unfrequently interfere directly with the diaphragm and the thoracic organs; occasionally also they encroach upon the chest, or actually make their way into this cavity through the diaphragm. In rare instances morbid products, such as pus, may find their way to distant parts of the body. In these different ways a variety of symptoms may be caused, sometimes of a curious nature and difficult to explain. Lastly, abdominal diseases are frequently attended with abnormal physical or objective signs, which are revealed on physical examination, and these are of such importance that they demand separate consideration.

**PHYSICAL EXAMINATION.**—The neglect of submitting patients to a satisfactory physical examination is a frequent source of error in diagnosis in cases of abdominal disease, and there ought to be no hesitation or delay in resorting to this method of clinical investigation whenever it seems called for. The precise course to be pursued must vary according to circumstances, but the following outline will serve to indicate the plan of procedure ordinarily required.

First, there are certain modes of examination which are applied to the abdomen externally, including *Inspection*; *Palpation* or *Manipulation*; *Mensuration* or *Measurement*; *Percussion*; and *Auscultation* (see **PHYSICAL EXAMINATION**). Of these, inspection, palpation, and percussion are by far the most important, and have, in the large majority of cases, to be relied upon for the information required. In exceptional instances *Succussion*, or shaking the patient, proves serviceable, by bringing out certain sensations or sounds. In order to carry out these methods properly, it is necessary to expose the abdomen sufficiently, due regard being paid to decency in the examination of females; to place the patient in a suitable position; and to see that the muscles of the abdominal walls are duly relaxed. The best position usually is for the patient to lie on the back, in a half-reclining attitude, with the head and shoulders well raised, and the thighs and knees more or less flexed. This posture serves to relax the abdominal muscles, which may be further aided by taking off the patient's attention by conversation or in other ways, as well as by directing him to breathe deeply. The position, however, has often to be varied in the investigation of particular cases, and much information is frequently gained by noticing the effects of altering the posture.

The objective conditions which may be revealed by the modes of examination thus far considered are as follows:—1. The state of the superficial structures. 2. The size and shape of the abdomen, generally and locally, as indicating an alteration in the volume of the ordinary contents of the abdomen, or the presence of some new or fresh element, such as dropsical fluid or a tumour. 3. The characters of the abdominal respiratory movements; and the presence of any unusual sensations during the act of breathing, such as friction-fremitus. 4. The sensations experienced on palpation and percussion over the abdomen, either as a whole, or in any particular part of it, such as its mobility, degree of resistance, regularity, consistence, &c.; as well as the presence of certain peculiar sensations, *e.g.*, fluctuation, or hydatid-fremitus. 5. The presence and characters of any pulsation. 6. The occurrence of abnormal movements within the abdomen, as those of a fœtus. 7. The sounds elicited,

generally and locally, on percussion. 8. The presence of certain sounds within the abdomen, heard on auscultation, such as friction-sounds; murmurs connected with aneurysm or due to pressure on an artery; or murmurs and sounds associated with the pregnant uterus.

Secondly, it not uncommonly happens that special modes of examination have to be applied to particular organs within the abdomen, in order to arrive at a diagnosis with any certainty. And here it may be remarked that it is highly important in all cases to see that no accumulation of feces exists within the bowel, and that the bladder is properly emptied, otherwise very serious mistakes are liable to be made. Purgatives and enemata are needed in order to remove any fecal collection. The urine should also be properly tested in every instance; and much information may often be gained in the investigation of affections of the alimentary canal, from a personal inspection or more complete examination of feces or vomited matters. The abdominal organs to which special modes of examination are chiefly applicable are the female generative organs, which are investigated per vaginam (*see* WOMB, Diseases of); the bladder, by means of the catheter, the sound, and other surgical instruments; the stomach, by the use of the stomach-pump, probang, &c.; and the intestines, by examining with the finger, hand, or surgical instruments per rectum, or by injecting water or air through the anus into the bowel. The ordinary modes of examination already mentioned may afford assistance when employed along with some of the special methods just indicated.

Thirdly, occasionally it is requisite to have recourse to exceptional modes of investigation, such as the use of the exploring trocar or aspirator; or to the administration of chloroform. The latter may afford direct information in certain abdominal conditions, and it may also materially assist in carrying out other methods of exploration. Now and then more serious operative procedures have to be adopted for diagnostic purposes.

The abnormal conditions discoverable by physical examination may involve the entire abdomen, giving rise, for instance, to general enlargement or retraction; or they may be limited to some particular region, as exemplified by enlarged organs, tumours, or abscesses. This part of the body has been artificially divided by anatomists into regions, and the seat of any local morbid condition can thus be defined and described. The diseases peculiar to the several regions will be considered under their respective headings.

FREDERICK T. ROBERTS.

**ABDOMINAL ANEURYSM.**—Abdominal aneurysm includes aneurysm of the aorta, and of any of its branches within the abdomen.

**Aneurysm of the Abdominal Aorta** is essentially a disease of middle age. Of 59 cases collected by Dr. Crisp, 33 were under the age of forty. It is more common in the male than in the female sex, in the proportion of about 8 to 1; and is usually traceable to strain, or to a blow upon the abdomen or back. The aneurysm is most frequently located in that portion of the vessel included between the aortic opening in the diaphragm and the origin of the superior mesenteric artery, being seldom met with below this point. In this situation the tumour is deeply seated; liable to tension from the crura of the diaphragm; and likely to involve the great splanchnic nerves, the semilunar ganglia, and the solar plexus. Hence the occasional difficulty of diagnosis; and the frequency of boring pain in the back from erosion of the vertebræ, and of paroxysms of radiating pain in the abdominal viscera from stretching of the adjacent nerves. When situated lower down in the course of the aorta, the disease is less obscure, and the symptoms are less urgent. Aneurysm of the abdominal aorta is usually of the *false* variety; and, as contrasted with thoracic aneurysm, it is less often associated with extensive atheroma of the aorta, and with fatty or other structural disease of the heart. The symptoms referable to excentric pressure are also fewer, and, with the exception of pain, are less urgent than in the thoracic variety.

**SYMPTOMS AND SIGNS.**—Of the symptoms, *pain* is the most characteristic and the most urgent; it is of two kinds, which are not, however, necessarily associated. In its usual form the pain of abdominal aneurysm is essentially neuralgic; it is intermittent, lancinating, and paroxysmal,—encircling the body like a girdle, or radiating through the abdomen, back, pelvis, and base of the thorax, and not infrequently into either groin or testicle. The accession is sudden, and usually attributable to some definite cause of vascular excitement. The duration extends over a period varying from one to three hours, rarely longer; and the cessation is equally abrupt, leaving the patient in a state of exhaustion, but quite free from actual suffering. The second kind of pain referred to is continuous and boring; fixed at a particular point of the vertebral column; aggravated by pressure at this point, by active movement or stamping, and by gently turning the patient half round upon his axis in the standing posture; but relieved by assuming the prone position or by leaning forward. Pain so characterised is pathognomonic of erosion of the vertebræ. Pressure of an aneurysm may affect the functions of several organs within the abdomen. Thus *jaundice* may result from pressure upon the hepatic or common biliary duct: it is, however, more frequently due to an aneurysm of the hepatic or of the superior mesenteric artery. *Interference with the urinary*

*secretion*, and the consequences thereof, from pressure upon the renal vessels; *dysphagia*, from pressure upon the œsophagus; *vomiting*, from obstruction of the pylorus; *displacement* of the liver forwards, or of the heart upwards—though rare symptoms—may be likewise due to the same cause. The radial pulse is not often affected. Symptoms of *constitutional* irritation and impaired nutrition are rarely exhibited, and appear only at the termination of protracted and painful cases, associated with great suffering and want of sleep.

The *physical signs* are those discoverable by inspection, palpation, percussion, and auscultation. The tumour usually projects to the left of the mesial line, and tends to descend; it is smooth and elastic; communicating to the hand alternate movements of lifting and expansion with increasing tension, and of subsidence with relaxation. The pulsation is all but invariably single, and synchronous with the radial pulse; it is limited to the tumour, and occasionally accompanied by thrill. Pressure upon the aorta below the tumour will increase the force of impulse, diminish or abolish the thrill, and arrest the collapse. In a few recorded examples the tumour was hard and uneven on the surface, and non-expansile; and in a still smaller number no pulsation was perceptible, the aperture of communication with the artery having been blocked, or the vessel compressed on the proximal side by the growth of the aneurysm itself. Owing to the position of the hollow viscera in front, and the mass of lumbar muscles behind, the evidence from percussion is less conclusive in regard to abdominal than thoracic aneurysm. If, however, the abdominal muscles be relaxed, and the stomach and bowels free from flatus, absolute dullness to the extent of the tumour may be detected. A sound, single or double, as distinguished from murmur, is rarely heard in front in connexion with abdominal aneurysm; whereas the existence of sound without murmur, and usually double, at a point of the posterior wall of the abdomen corresponding to the tumour, is the rule, and, when detected, is of the utmost diagnostic value. Murmur in the recumbent posture is rarely absent in front; it is single, blowing, prolonged, post-systolic, and not transmitted into the vessel beyond. It may, however, be musical, or it may present both these characters, but at different points of the tumour; in one instance it was of a buzzing quality. Should the aneurysm have taken an exclusively backward course, which is the exception, a single murmur, not audible in front, may be heard in the back. In a few recorded cases a double murmur has been heard over the aneurysm in front. In the erect posture the murmur is usually suspended; but in a few published cases it was audible in both the erect and the recumbent posture, and in

one at least in the erect posture only. These peculiarities depend upon the various conditions of the sac, its orifice, and its contents. A small aneurysm engaging the posterior wall of the vessel only, and eroding the vertebræ, may be latent as to physical signs, though attended with severe fixed pain in the back. In some cases a slight retardation of the pulse in the femoral arteries has been noted.

**DIAGNOSIS.**—The diagnosis of abdominal aneurysm has reference mainly to its physical signs. Strong pulsation of the aorta, simulating that of aneurysm, may exist in connexion with hysteria, uterine or intestinal irritation, dyspepsia, or marked anæmia. But in all these cases, irrespective of the positive and specific evidence presented by each, throbbing exists throughout the aorta, and is propagated into the main arteries of the lower limbs, whereas it is localised and expansile in aneurysm; and a careful exploration of the aorta—if necessary, under the influence of chloroform—will show that its dimensions are at all points normal. In these cases, too, although a murmur may be produced by strong pressure with the stethoscope, it does not exist when pressure is withdrawn. A cancerous or other tumour pressing upon the aorta may likewise produce murmur, and may exhibit pulsation communicated from the aorta; but in most cases both these phenomena are promptly arrested by placing the body in the prone position; the tumour, in that position, gravitating from the vessel. The fixed local pain in the back, aggravated by pressure and motion, may be simulated by ordinary lumbago; and the paroxysmal visceral pain by biliary colic. The differential diagnosis must rest upon the specific evidence in each case, and upon the absence of the signs of aneurysm.

**Aneurysm of the Branches of the Abdominal Aorta.**—The branches most liable to aneurysm are the common iliacs and their divisions; the celiac axis and its branches; the renal, and the superior mesenteric. *Aneurysm of the Iliac Arteries* belongs to the domain of surgery, and will not be further referred to here. *Aneurysm of the Celiac Axis* and of its branches of division, and of the *Superior Mesenteric Artery*, are, in addition to the ordinary signs, equally characterised by mobility of the tumour; and the first two varieties by the occurrence of jaundice, hæmatemesis, and melæna, from pressure. Renal aneurysm may cause obstruction in the kidney, or renal colic, by pressure on the structures in the hilus.

**DURATION and TERMINATIONS.**—The duration of life in abdominal aneurysm in a number of cases observed, varied from fifteen days to eleven years. Death occurs usually: (1) by rupture of the sac into (a) the retro-peritoneal tissues; (b) the cavity of the peritoneum; (c) the left pleura or lung; (d) the intestinal

canal; (e) the inferior cava; (f) the psoas muscle; (g) the pelvis of the kidney; (h) the spinal canal; and (i) the ureter, biliary passages, or œsophagus; the order given represents the relative frequency of the fatal result; or (2) by exhaustion or syncope. The duration of life after the rupture of the aneurysm has ranged from a few minutes to several weeks. A consecutive false aneurysm, retro-peritoneal in site, is specially characterised by feeble pulsation of the tumour, and diminished or arrested circulation in the femoral artery of one or both sides.

**TREATMENT.**—The *curative* treatment of abdominal aneurysm may be considered under three heads—*operative*, *postural* and *dietetic*, and *medicinal*. *Operative* treatment consists in pressure applied to the aorta on the proximal side of the sac, or simultaneously on its proximal and distal sides, by means of tourniquets, so as completely to stop the circulation. The bowels should be first well moved and freed from flatus; and during the continuance of pressure the patient should be kept under the influence of chloroform or ether. As a rule, compression should not be employed above the level of the duodenum. Five cases, if not more, in which a cure was effected by these means have been reported. The object sought to be attained being that of effecting rapid coagulation in the sac, the period during which pressure needs to be continued in these cases varies from three quarters of an hour to ten hours and a half. Where space for the application of proximal pressure does not exist, distal pressure alone may be tried. Under all circumstances, pressure must be used with care, as inflammation of the peritoneum or of the bowels has been known to result from it. Galvanopuncture, and the introduction of iron wire into the aneurysmal sac (Moore's treatment), have also been employed. In a small proportion of cases the latter method has been attended with a favourable result.

Bellingham introduced the plan of treatment by *posture and restricted diet*. Under this plan perfect repose of mind and body is, as far as practicable, to be maintained; the bowels being kept moderately free, and the dietary restricted to 10 oz. of solids and 6 oz. of liquids daily. According to the method of the late Mr. Tufnell, which is based upon the same principle, but is more rigid, the patient is strictly confined to the horizontal posture for a period varying from eight to thirteen weeks, as determined by the effect upon the aneurysm, movement in bed being effected with caution; whilst, by a special arrangement, the bowels and the bladder may be evacuated without disturbance of the body. For breakfast, 2 oz. of white bread and butter, with 2 oz. of cocoa or milk, are allowed; for dinner, 3 oz. of meat, with 3 oz. of potatoes or bread, and 4 oz. of water or claret; and for supper, 2 oz. of bread and butter, and

2 oz. of milk or tea. The total amount in the twenty-four hours would be: solids 10 oz., liquids 8 oz. This system might be in some degree relaxed if the patient proved restive. Mild laxatives and opiates as required are the only medicines used. Ten cases of the successful treatment of aortic aneurysm by this method have been reported by Mr. Tufnell. Abdominal aneurysm was solidified in two instances, after treatment extending over thirty-seven and twenty-one days respectively.

Of the various *medicinal* agents used with a view to favouring or effecting a deposit of laminated fibrin in the sac, acetate of lead, iodide of potassium, aconite, and ergotin (administered hypodermically), alone claim attention. Iodide of potassium may be given with advantage in doses of 10 to 20 grs. thrice daily, with a view to reducing vascular tension, and thereby relieving pain and promoting deposition in the sac, whilst perfect rest in the recumbent posture and a restricted dietary are observed. The latter are, however, the more important factors in the treatment. Dr. G. W. Balfour has reported several cases successfully treated by means of iodide of potassium; and an example of a similar kind was subsequently published by Sir Dyce Duckworth. Dr. Grimshaw has recorded an example of cure mainly through the use of aconite. In all cases the allowance of liquids must be reduced to the lowest possible standard, whilst excretion is promoted. Alcoholic stimulants may be given in small quantity and at long intervals, if the pulse exhibit debility and the patient complain of a sensation of sinking; otherwise they should be prohibited.

The *palliative* treatment as applied to abdominal aneurysm will be found described in the article AORTA, Diseases of (*Aneurysm*). The application of a few leeches, followed by a warm poultice, is very efficacious in relieving pain. The hypodermic use of morphine is still more rapidly effective.

THOMAS HAYDEN.

C. J. NIXON.

**ABDOMINAL TYPHUS.** — A synonym for typhoid fever. See TYPHOID FEVER.

**ABDOMINAL WALLS, Diseases of.**—But little more will be needed in this article than to give a brief outline of the nature of the affections to which the abdominal walls are liable, as most of these are but local forms of diseases which are fully described in other parts of this work. The parietal peritoneum will be excluded from consideration, as its morbid conditions are treated of separately.

1. **SUPERFICIAL AFFECTIONS.**—*a.* The skin covering the abdomen may be the seat of various eruptions. The rash of typhoid fever is chiefly observed over this region.

b. When the abdomen is greatly enlarged, its cutaneous covering becomes stretched and thinned, often presenting a shining appearance; or it may even give way, so that it exhibits superficial cracks or fissures. If it has been distended for a considerable time or on several occasions, as after repeated pregnancies, the skin becomes impaired in its nutrition, and is often the seat of permanent white lines or furrows—*lineæ albicantes*. In this connexion allusion may be made to the umbilicus, which, in certain forms of distension of the abdomen, may become puffed out, everted, or actually obliterated. c. The veins of the skin frequently become enlarged and tortuous, when the return of the blood which is normally conveyed through them is in any way impeded. The particular vessels which are distended will necessarily depend upon the seat of the obstruction. d. The cutaneous sensibility over the abdomen is sometimes materially altered. In certain nervous diseases it may become more or less impaired or lost; but the most important deviation is a marked increase of sensibility—*hyperæsthesia*—which is occasionally observed in hysterical females, and which may simulate more serious affections, particularly peritonitis, especially if it is accompanied with symptoms of much depression. This condition is characterised by extreme *superficial* sensibility or tenderness of the abdomen, the slightest touch being resented; but if the patient's attention can be taken off, and deep pressure be then made, this is borne with little or no indication of distress. The aspect of the patient, the presence of other symptoms indicative of hysteria, and the absence of pyrexia usually serve to distinguish this affection from others of a graver nature. The surface of the abdomen may also be affected with neuralgia, which is sometimes very severe.

2. SUBCUTANEOUS ACCUMULATIONS.—a. The chief morbid condition coming under this head is *œdema* or *dropsy* of the subcutaneous tissue. This generally follows anasarca of the legs, and may be associated with ascites. The fluid tends to collect especially in the lower part of the abdominal walls and towards the flanks. The skin often presents a white pasty aspect; the abdomen may be more or less enlarged; the umbilicus appears depressed and sunken, if the œdema extends up to this level; the superficial structures pit on pressure, and yield the peculiar sensation of dropsical tissues; and the percussion note is frequently muffled. b. The abdominal subcutaneous tissue is, in many persons, the seat of an abundant collection of fat, which may be important from causing apparent enlargement, and simulating or obscuring other more serious morbid conditions which enlarge the abdomen. c. In rare instances gas accumulates under the skin in this region, constituting *subcutaneous emphysema*. The

abdomen then appears to be enlarged, but the condition is readily recognised by the peculiar crackling or crepitant sensation felt on digital pressure; the resonant or tympanic sound elicited by percussion; and the dry crepitant sound heard when pressure is made with the stethoscope.

3. AFFECTIONS OF THE MUSCLES AND APO-NEUROSSES.—a. The abdominal walls may be the seat of muscular rheumatism, which is particularly likely to follow undue straining, such as that caused by violent coughing or vomiting. It is characterised by pain, sometimes severe, evidently located in the muscular and tendinous structures, accompanied with much soreness and tenderness. The affected parts are kept as much at rest as possible, and any action which disturbs them materially aggravates the pain. b. As the result of violent strain, the muscular or aponeurotic tissues may be more or less torn or ruptured, or the normal openings enlarged. As a consequence a protrusion of some internal structure is likely to take place, forming a hernia. c. The abdominal muscles are liable to be the seat of spasmodic contraction, cramp, or rigidity. These disorders are not uncommonly excited in sympathy with grave disturbance of the alimentary canal, as in cholera. In certain painful internal affections, also, some of the abdominal muscles are occasionally kept in a state of more or less rigid tension, as if they were involuntarily contracted in order to protect the diseased parts underneath from injury. The spasmodic contractions in tetanus not infrequently cause great suffering over the abdomen. d. On the other hand, the abdominal muscles are occasionally paralysed, as the result of centric nervous disease. The movements of respiration are then altered in character; while the expulsive acts in which the abdominal muscles naturally take part are much interfered with.

4. RELAXED ABDOMINAL WALLS.—All the structures forming the walls of the abdomen are often in a relaxed and flabby state, yielding to any pressure from within, so that the abdomen becomes enlarged and prominent, especially if, as is frequently the case, this condition is associated with much flatulence. It materially weakens the act of defæcation, and promotes constipation.

5. INFLAMMATION AND ABSCESS.—Local inflammation may be set up in any of the abdominal structures, and this may terminate in suppuration and the formation of an abscess. Purulent accumulations from within, as in cases of pelvic abscess, as well as certain abscesses originating in diseases of bones or joints, may likewise extend among the tissues of the abdominal walls, causing thickening and induration, or may make their way outwards, directly or through a sinus. Subsequently permanent sinuses or fistulæ may be left.

6. The abdominal wall may be the seat of *extravasation of blood*; and various kinds of *tumour* or *new growth* may form in its structures.

FREDERICK T. ROBERTS.

**ABERRATION.**—A divergence or wandering from the usual course or condition; applied in medicine chiefly to certain disorders of the mental faculties. See INSANITY.

**ABORTION.**—The act of abortion signifies the expulsion of the contents of the pregnant uterus before the seventh month of gestation. An *abortion* is a designation given to a fœtus prematurely expelled. See MISCARRIAGE.

**ABSCESS** (*abscedo*, I depart). SYNON.: Fr. *abcès*; Ger. *Eiterbeule*; *Geschwür*.

**DEFINITION.**—A collection of purulent matter, one of the results of inflammation. See INFLAMMATION.

**PATHOLOGY.**—If the material which collects in a tissue as the consequence of inflammation softens and becomes liquid (suppuration), it does so either rapidly or slowly: if the former, the result is an *acute* abscess; if the latter, the abscess is termed *chronic* or *cold*. If the material thus softened and forming pus, often mingled with fragments of dead tissue, is limited by the condensation of the parts around, which are usually consolidated by the products of inflammation, the abscess is said to be *circumscribed*; but if the surrounding parts in their turn soften, so as practically to offer no barrier to the pus, then the abscess spreads and is said to be *diffused*. The term 'diffused suppuration' is, however, in this case, more usually employed. In an *acute circumscribed* abscess the lymph which collects around it as the result of inflammation becomes organised and forms a sac (pyogenic membrane); and this, with the compressed tissue about it, constitutes the wall; the entire abscess consisting therefore of contents (pus), of a limiting sac, and of condensed tissue around. The resistance offered to the extension of the suppuration is greatest when the parts adjacent are dense and tough, such as bones and fascia; yet, as the pus in an abscess increases in quantity, probably by breaking down of the pyogenic layer, sufficient pressure is exerted to cause the most dense structures ultimately to yield, and an abscess will thus make its way even through osseous tissue. As might be expected, an abscess always advances in the direction of least resistance, and this extension is spoken of as its *pointing*. This pointing may be towards the surface of the body, but an abscess may direct itself towards a serous cavity, such as the peritoneum; or along a track of cellular tissue, as when pus beneath the deep

cervical fascia points into the mediastinum. On the side at which the abscess is pointing, its wall, as the resistance lessens, projects; and by ulcerative absorption the parts covering it become quickly thinner, until they and the abscess-wall give way and the pus escapes. In by far the greater number of cases this absorption of tissue before the pointing abscess is towards the surface, and it is by ulceration of the skin that the opening for the discharge of the matter is effected. The wall of the abscess then contracts, pus continuing for a time to be discharged; and in the end, aided by the resilience of the tissues around, the sac of the abscess is obliterated, and the orifice through which its contents were discharged heals by granulation process. To ensure this result the walls must be left at rest, or the granulations which cover them may fail to unite, and the obliteration of the sac will not then take place. This happens, for example, in the case of an abscess situated between the movable rectum on the one side and the ischium on the other, where the opposite abscess-walls are prevented from joining by muscular movements on the side of the bowel, and will only unite after such movements have been stopped by cutting across the muscular fibres which occasion them. The track which results from such failure of the healing of an abscess is called a *sinus* or *fistula*.

In a *diffused* abscess the inflammation of the parts around does not limit the suppuration by organisation of the effused lymph, but such lymph, itself degenerating, forms more pus, and so the abscess extends rapidly and widely, unless checked by some barrier of dense tissue. In this way matter often spreads along tracts of cellular tissue, as along the course of veins, and in the subcutaneous structures. An abscess when formed between bone and periosteum, or otherwise hindered from reaching the surface by pointing, also tends to diffuse itself by following the course of least resistance. In most of these cases, by direct pressure upon the resisting tissue, or by cutting off the blood-supply (as of the skin when its subcutaneous tissue is infiltrated with pus), sloughing of the parts covering in the abscess ensues, oftentimes to a considerable extent, and so the pus eventually makes its way to the surface.

A *chronic* abscess begins in some local inflammation without active symptoms, such as results in the deposit of aplastic lymph and subsequent ulcerative changes, as caries of bone, the irritation leading to suppuration. The formation of matter or 'pus' proceeds in a languid manner, so that it is only by slow degrees that it collects in any considerable quantity, although eventually these chronic abscesses may acquire great size. They slowly point, and in their tardy advance occasionally traverse even serous cavities, which

have been first obliterated in the line of transit by adhesive inflammation of their opposed surfaces: in this way an abscess formed in the liver (and this holds good also for those of a more acute character) may travel through the layers of the peritoneum, and may point through the anterior wall of the abdomen.

When an abscess discharges, its contents are seen to be either a thick yellow (*laudable*) pus, or pus stained with blood, or otherwise coloured, such as black or bluish-green; or the pus may be thin, almost watery, mingled with flakes of lymph; it may be inodorous or fetid, or irritating to the skin (*ichorous*). Abscesses may also contain sloughs of tissue, foreign bodies, masses of inspissated pus as hard occasionally as calculi, fragments of dead bone, or calculi of various kinds. Sometimes a chronic abscess ceases to enlarge; and if the irritation which occasioned it comes to an end, it may diminish by absorption of the fluid part of its contents, the solid part drying up into a shrunken putty-like mass. It may remain in this state without giving rise to further trouble, or it may become again the seat of suppuration by the formation of what under such circumstances has been termed by Sir James Paget a *residual* abscess; this term 'residual' being also applied to an abscess which has formed at the site of old inflammatory processes, although these may never have advanced previously to actual suppuration.

The *progress* of any abscess is largely influenced by the state of the general health. In persons otherwise robust an abscess commonly runs an acute course; in those weakened by acute illness, such as scarlet fever or typhus, they form quickly, but are slowly recovered from, and severely tax, by an exhausting discharge, the powers of the patient. Persons in feeble health, hereditary or acquired, usually suffer from the chronic and diffused forms; and chronic affections of internal organs, as of the liver or kidneys, are not infrequently associated with the development of such abscesses.

**ÆTIOLOGY.**—The cause of an *acute* abscess may be an injury, such as a blow or pressure, as often happens in persons weakened by continued fever; exposure; or the irritation of a foreign body, or that of a poison introduced from without. In the last case the abscess is often diffused. Abscess running an acute course may also be due to a foreign body or to an irritant from within, as when it follows necrosis of a portion of bone, or the escape of urine into the tissues of the perineum. It also arises in connexion with blood-poisoning, as in various fevers, and affections distinguished as septic. The cause of a *chronic* abscess is usually found in changes which go with deposits of a tuberculous character; or it is found in the changes which slowly occur around an irritating

body, such as a renal calculus; or chronic inflammation may culminate in one of these collections of matter. They may also form in parts which are long congested in connexion with obstructed venous circulation (*varix*); and they may follow, or conditions closely allied may follow, the occlusion of a main artery and the consequent cutting off of the supply of blood to a particular region.

**SYMPTOMS.**—The symptoms of an *acute* abscess are those of a local inflammation, with constitutional disturbance if the abscess is of any size; followed by a sense of cold or actual shivering, with increase of pain and swelling, tenderness, and throbbing. The tenderness can be recognised in the case of most abscesses; and, if pus is formed anywhere near the surface, the presence of the fluid is detected by its fluctuation. The severity of the pain is much influenced by the site of the abscess, as when the pus is held down and hindered from pointing by dense structures, such as fasciæ. Special symptoms may also arise in connexion with the situation of the suppuration, as when urgent dyspnoea is caused by the pressure on the larynx of an abscess deeply seated at the base of the tongue. A *diffused* abscess, if subcutaneous, is recognised by its rapid spreading, and may be suspected if other signs point to a part as the site of the abscess in which diffusion is the rule.

Of *chronic* abscess there is seldom any evidence in its early stage. The symptoms, if any, are those of failing health, and for the rest are marked by those of other changes from which the abscess is an outcome. Thus in disease of the hip-joint or of the spine, unless an attack of shivering chances to attract attention, an abscess is not, as a rule, suspected until it has broken through its first limits, and has attained considerable size. Our limits do not permit an examination of the symptoms which serve to distinguish between chronic abscess and other swellings, such as extravasated blood or soft tumours, especially malignant tumours; but this is the less requisite, for if a doubt in any case arises, it can be at once solved by the introduction of a grooved needle or of a fine trocar into the swelling. The true pulsation of an aneurysm sufficiently tells its nature, and is not easily mistaken for the impulse sometimes given to an abscess by an adjacent artery.

**VARIETIES.**—The chief local varieties of abscesses which are likely to be met with in medical practice may be thus arranged:—  
1. Subcutaneous or more deeply seated abscesses in the limbs, in connexion with low fevers, erysipelas, pyæmia, &c. 2. Abscesses of local origin in the walls of the abdomen or chest. 3. Abscesses originating in serous membranes. 4. Certain special abscesses associated with diseased bone, *e.g.* psoas and

lumbar abscess. 5. Abscesses formed in the cellular tissue around organs, *e.g.* peri-nephritic, peri-cæcal, &c. 6. Abscesses originating in inflammation of organs, the chief of which include hepatic, renal, pyelitic, pulmonary, mammary, cerebral, splenic, pancreatic. 7. Obscure abscesses formed in the deep cellular tissue, *e.g.* retro-pharyngeal, ischio-rectal, mediastinal. 8. Glandular abscesses, which are usually chronic, and of a scrofulous nature. 9. Suppurating hydatid cysts.

**TREATMENT.**—The treatment of an *acute* abscess consists in rest; soothing local applications, of which poppy or plain water fomentations, and warm poultices, as of boracic wool or linseed-meal, are usually the best; and the use of remedies to allay pain and constitutional disturbance, if required. As soon as the presence of pus is recognised the abscess must be opened, if possible where the matter is most dependent; and as soon as its contents have escaped, all troublesome symptoms will usually disappear. The opening is needed to relieve pain, in some cases to prevent diffusion of pus, and sometimes to relieve urgent distress, as when dyspnoea is caused by the pressure of an abscess upon the air-passages. It is also desirable to open an abscess to avoid the considerable scar which must result if the matter is left to escape by ulceration or sloughing of the superficial tissues. In most cases it is necessary to open an abscess by an incision. A narrow knife should be used; and if the matter is deeply seated, the superficial parts only need be cut, the deeper being torn through, as the late Mr. Hilton recommended, by a steel director and by dressing-forceps, the risk of dividing important structures, as in the neck, being thus avoided. After the abscess has been punctured, it should be left to contract by the resiliency of its own walls, all squeezing and handling being carefully refrained from, as harmful and unnecessary, and giving the patient needless pain. The wound should be kept open, if necessary, by means of a drainage-tube, or a slip of thin gutta-percha tissue or of oil-silk, and should be covered with absorbent carbolie gauze, iodoform wool, or some other of the numerous antiseptic dry dressings now employed for this purpose; or, where moist applications are needed, by wet boracic lint or wool, or by a linseed-meal poultice. In some cases it is necessary to protect the wound, whilst operating, by means of the carbolie spray, or by a piece of linen steeped in carbolie lotion (1 in 20), or by taking some other antiseptic precautions. The drainage-tube should be withdrawn after the first day if the abscess is superficial, but if the pus has been deeply seated it should be only gradually withdrawn, portions being cut off as the abscess contracts. If a foreign body has caused the formation of the abscess, it must be sought for and removed before the supuration can be expected to cease. Occa-

sionally the vascular wall of an abscess bleeds freely, or a vessel is opened in its progress. The hæmorrhage usually ceases on laying the abscess freely open; but if this does not suffice, it may be permanently controlled by pressure, and cases are rare in which further operative interference is called for. Inflammation of the sac used formerly not infrequently to follow the discharge of its contents, but under the treatment now employed such an occurrence is unknown. During the healing of any considerable abscess the general health should be attended to, and tonics and change of air may be useful to expedite recovery.

*Diffused* abscesses, whether subcutaneous or more deeply seated, require free incisions as soon as suppuration is even suspected, so as to avoid the damage which results from their spreading, and from the sloughing of tissue, as of the skin, which will otherwise occur; and this treatment is especially called for in the case of those due to poison introduced into the system, and those caused by infiltration of urine. These abscesses sometimes lead to fatal results.

A *chronic* abscess may have its contents drawn off by the aspirator; or it may, when it has come near the surface, be opened, drained, and dressed as above, but its ultimate closing will depend upon the removal of the cause; if, for example, it is due to disease of a joint, it cannot be cured until the disease in which it has originated has in some way ended. In some cases of suppurating lymphatic glands it is well to scrape out their interior, after making a clean incision into them, and to apply iodoform or some other antiseptic to the cavity which is left.

**Sinus.**—An abscess after being opened may contract until it forms a narrow track, *sinus* or *fistula*, leading to the site of primary irritation. Such a track has a dense fibrous wall from which purulent fluid escapes. It may also convey secretions, as from the liver (*hepatic fistula*) or stomach (*gastric fistula*), or excretion, as from the kidneys; or it may simply carry out the pus which forms around some irritant at the deep extremity, such as a foreign body, or a portion of carious or necrosed bone. Some such fistulæ are due to the movements of adjacent muscles preventing union of the abscess-walls. Unless the cause of the sinus can be removed, as by extracting necrosed bone, these fistulous tracks are difficult to manage, requiring special treatment according to their situation. Other *fistulæ* are those forming communications between mucous canals (*recto-vesical, vesico-vaginal fistulæ*); and these need special treatment, such as plastic operations, and operations diverting the course of excreta escaping through unnatural channels.

The tissue about healed abscesses, scar-tissue generally, and tissue spoiled by inflammation, are apt on slight provocation to in-

flame and suppurate, and to these collections of matter the term '*residual*' has been applied. The treatment of such abscesses in no way differs from that of others, and they usually heal in the ordinary manner.

G. W. CALLENDER. W. MORRANT BAKER.

**ABSINTHISM.** — DEFINITION. — The condition induced by an excessive consumption of absinthe.

From the mode in which absinthe is taken, we should expect that the symptoms induced by its excessive consumption would be generally obscured by, and intermixed with, those of alcohol (*see* ALCOHOLISM). That it has a special effect on the organism, and that this may be diagnosed from alcoholism, has been pointed out by Motet, Magnan, and other French physicians; and Magnan has clearly exemplified its action by numerous experiments on dogs. In persistent absinthedrinkers, vertigo and epileptiform convulsions are marked symptoms, and come on much earlier than when alcohol, in other forms, is habitually drunk. Hallucinations occur also without any other symptom of delirium tremens; and, when tremors coexist, these are limited more particularly to the muscles of the arms, hands, and shoulders. Absinthe acts chiefly on the cervical portion of the spinal cord, and this readily explains the special symptoms arising from its regular use in excessive quantities. JOHN CURNOW.

**ABSORBENT AGENTS.** — In *surgery*, absorbents are substances used to absorb fluids, as sponges, charpie, or tow; in *medicine*, drugs which neutralise excessive acidity in the stomach—a synonym for alkalis (*see* ALKALIS). The term is sometimes also made use of to designate remedies, such as the preparations of mercury and iodine, which are believed to possess the property of promoting the absorption of morbid products.

**ABSORBENT VESSELS and GLANDS, Diseases of.**—*See* LYMPHATIC SYSTEM, Diseases of; BRONCHIAL GLANDS, Diseases of; and MESENTERIC GLANDS, Diseases of.

**ACARUS.**—*Acari* or *mites* constitute an order of the class *Arachnida*, several species of which are parasitic. The *Acarus scabiei* or *Sarcoptes hominis*, and the *Acarus folliculorum*, or more properly the *Steatozoon folliculorum*, are the only human parasites belonging to this family.

DESCRIPTION.—1. The ACARUS SCABIEI is a small roundish animal, just visible to the naked eye. Examined under the microscope it is seen to be flattened, and to resemble a tortoise in shape; when fully developed it has eight legs, and on its under surface are

scattered filaments and short spines, which are for the most part directed backwards. The female is larger than the male, and is provided with terminal suckers on the four anterior legs, while filaments occupy a similar position on the posterior ones; in the male, however, the two extreme hind legs have suckers like those on its fore limbs. The young *acarus* has only six legs, the two hind-most ones, which are distinctive of the sex, being wanting; it acquires these after shedding its first skin. The male *acarus* lives near the surface of the skin, while the female burrows within the cuticle, and deposits from ten to fifteen eggs in the cuticulus or burrow; these eggs hatch in about a fortnight. The young *acari* escape from the burrow, but the parent does not leave it, and dies when she has finished laying eggs. The *acarus scabiei* is the cause of the skin-affection termed *Scabies* or *Itch*. *See* SCABIES.

2. The ACARUS FOLLICULORUM is a very minute parasite commonly found in the sebaceous and hair-follicles of the face, but its presence can hardly be regarded as indicating disease. In this animal the head is continuous with the thorax, and to the latter are attached eight very short legs, each armed with three strong claws. On each side of the head are short jointed palpi. The abdomen varies in length from twice to three or four times that of the thorax; it is pointed at its distal extremity. The presence of this parasite in the follicles of the skin is quite unimportant. ROBERT LIVEING.

**ACCOMMODATION, Disorders of.**  
*See* VISION, Disorders of.

**ACEPHALOCYST** (ἀ, priv.; κεφαλή, a head; and κύστις, a bag).—A headless cystolar hydatid; a sterile hydatid. *See* ENTOZOA—Hydatids.

**ACHOLIA** (ἀ, priv.; and χολή, bile).—Absence or deficiency of bile. *See* BILE, Disorders of.

**ACHOR.**—A small follicular pustule of the scalp. Willan's definition is as follows:— 'A small acuminated pustule containing a straw-coloured matter, which has the appearance and nearly the consistence of strained honey, and is succeeded by a thin brown or yellowish scab.' Both the pustule and the scab are constituents of the disease *porriigo*. The word has fallen into disuse, but is preserved by Schönlein in the name 'achorion' assigned by him to one of the varieties of parasitic cutaneous fungi. The Greek word ἀχώρα signifies scurf, or dandruff; ἀχρόν meaning chaff. ERASMUS WILSON.

**ACHORION** (ἀχώρα, scurf) is the name given to one of the three principal dermophytes or epiphytes of the skin. It is the

constituent of the crust of favus (achor), and belongs to the group of fungoid plants denominated *Oidium*. It consists of spores, sporidia or tubes filled with spores, and empty branched tubes or mycelium.

*Achorion* was the first discovered of the epiphytes of the skin, and in compliment to one of its early observers, Schönlein, has been named *Achorion Schönleini*. It is supposed to be the agent of contagion in favus; it has also been found in the loose cell-structure beneath the nail in onychogryphosis.

ERASMUS WILSON.

### ACHROMA (*ἀ*, priv.; and *χρῶμα*, colour).

Absence of colour; an achromatous or colourless state of an usually coloured tissue, due to absence of pigment. In reference to the skin, achroma is synonymous with leucoderma, albinism, and alpliosis. See PIGMENTARY SKIN-DISEASES.

**ACHROMATOPSIA**, (*ἀ* priv.; *χρῶμα*, colour; and *ὄψις*, sight).—More or less complete inability to distinguish colours from each other. See VISION, Disorders of.

**ACIDITY**.—Acids are constantly passing out of the body by the lungs, the skin, and the kidneys. These acids, if we except the small quantity introduced from without in the form of acid salts of certain articles of food, are formed within the body by the disintegration and oxidation of the tissues and food. If the oxidation of organic substances in the system were complete, the sole products of their combustion would be carbonic acid, water, and urea; but as this oxidation is never actually complete, other products, as lactic acid, oxalic acid, uric acid, &c., are formed; and the increased or diminished production of these intermediary products may be regarded as the measure of the completeness with which the oxidation processes are being performed in the body. The quantity of acid matter passing through the blood on its way to the lungs, the skin, and the kidneys is considerable; since it has been shown approximately, that a healthy man of eleven stone weight, under ordinary circumstances, passes by the first two channels an average of 890 grammes (about 28 ounces) of carbonic acid daily, and that the acid excreted by the kidneys in the same period is equivalent to two grammes (about 31 grains) of crystallised oxalic acid; whilst the volatile fatty acids passing off with the sweat have not yet been satisfactorily calculated. It is evident that if the regular elimination of acids, by any of these channels, be interfered with, they will tend to accumulate in the system. Acidity, or excess of acid in the body, therefore depends on two causes:—1. *excessive formation*, the result of incomplete oxidation of the elements of the tissues and the food; 2. *deficient elimi-*

*nation* of acid formed either in normal or abnormal quantities. Both these causes, however, are generally found acting in conjunction. Oxidation is imperfectly performed when an insufficient quantity of oxygen is introduced into the body, owing to insufficiency of the respiratory act, the result of disease or sedentary habits; or when the blood is poor in red corpuscles, the carriers of oxygen, as in leukæmia; or from functional derangement of some large gland, such as the liver, where oxidising processes are extensively carried on. Again, the materials submitted to the influence of the oxygen within the body may be so increased, as is the case in febrile conditions, or in general plethora induced by over-feeding and insufficient exercise, that the supply of oxygen may prove insufficient for their complete combustion. Defective elimination of the acids formed within the body is due either to diseased conditions which prevent the lungs, skin, and kidneys from exercising their respective functions properly, or to want of the physiological stimulus which excites them. It will be seen, therefore, that acidity may arise in consequence of the disturbing influence of disease; or may be acquired or inherited as the penalty of transgression of certain laws of health—as the result of unfavourable hygienic conditions. In the former case, acidity is only secondary, and is generally subordinate to the disease producing it, and has rarely to be considered apart from it; whilst in the latter instance acidity is usually at first the only trouble, leading, however, if disregarded to secondary mischief.

**EFFECTS**.—The mucous membranes and skin chiefly suffer in acidity. The former become subject to catarrh, produced, no doubt, by the irritating presence of the acid. Acidity may thus cause bronchitis, gastrointestinal catarrh, and catarrh of the genito-urinary tract. Sometimes the acid is poured out in such quantities from the mucous membrane of the stomach as to be ejected from the mouth. In these cases digestion is considerably interfered with by the too acid condition of the gastric juice. Sometimes, however, this acidity of the stomach is produced by an opposite condition—the deficiency of the digestive fluid, and consequent acid fermentation of the food. Abnormal acidity of the urine not only produces catarrh of the urinary passages, but by decomposing the salts of uric acid causes a deposit of insoluble uric acid in the passages, thus giving rise to attacks of gravel, or leading to the formation of a calculus. Acidity manifests itself in the skin by attacks of erythema, herpes, eczema, and urticaria. Rheumatism, too, may be considered as a disease resulting from the formation of acid, affecting chiefly fibrous and serous membranes; no one can witness the enormous quantities of acid sweat poured out, and the highly acid urine,

in the acute form of this disease, without acknowledging that an increased formation of acid is taking place somewhere in the body; though perhaps unwilling to commit oneself to accept any of the views hitherto advanced as to the nature of the acid.

**ESTIMATION OF ACID.**—For clinical purposes an estimation of the acidity of the urine furnishes the physician with an approximate clue as to the amount of acid formed in and passing out of the body. This is done by collecting the urine for twenty-four hours, placing 100 c.c. of this in a beaker, and then adding a solution of sodium hydrate (standardised so that 1 c.c.=·01 gramme of crystallised oxalic acid) from a Mohr's burette, till the fluid is neutralised. The number of c.c.'s of the standard solution required to effect this is to be multiplied by ·01, which gives the percentage acidity in terms of oxalic acid. To ascertain from this the total amount of acid in the twenty-four hours' urine is only a matter of calculation. Too much dependence must not, however, be placed on the urine as a means of estimating excess or deficiency of acid in the system; it sometimes happens that in highly acid conditions the urine is alkaline. This, as Dr. Bence Jones has shown, may occur when large quantities of acid fluid are poured out of the stomach; and Prout long ago observed, that in the eczema of gouty persons, the urine, so long as the disease persisted, was either of low acidity or alkaline, but that the subsidence of the eczema was frequently followed by an over-acid condition of the urine, accompanied with renal and vesical catarrh.

**TREATMENT.**—The general indications for the treatment of acidity consist in the promotion of oxidation, and the elimination of the acids formed. Active habits, which promote the pulmonary and cutaneous functions, should be encouraged. The diet should be just sufficient to meet the physiological requirements of the body; it should consist chiefly of fish, fowl, game, and eggs; saccharine and farinaceous articles being excluded. Sweet and cheap wines should be avoided; for those who cannot afford to purchase good wine, pure spirits and water is the best substitute. Carlsbad salts or Friedrichschall water may be given if there is much abdominal plethora; the habit of taking mercurials as a relief for this condition is to be deprecated. Alkaline medicines are frequently administered with a view of neutralising the effects of acid; their employment for this purpose seems, however, questionable. The late Dr. Parkes stated that the administration of bicarbonate of potassium (a favourite remedy in acid diseases), though rendering the urine alkaline, in reality increases very largely the excretion of the organic acids. This is not to be wondered at when we consider that the bicarbonate, although alkaline in reaction, is in constitution an acid salt.

Dilute nitric and hydrochloric acids, given in moderate doses about one hour before meals, certainly have a powerful oxidising effect, and diminish the quantity of uric acid excreted in the urine. In cases where the acidity is manifestly due to defective oxidation, consequent on poverty of blood from diminution of the red corpuscles, iron and food must be freely given. C. H. RALFE.

**ACIDS.**—**DEFINITION.**—Substances which combine with alkalis, and destroy their power of turning red litmus-paper blue. Most of the acids also redden blue litmus, and have a sour taste; but some—for example, carbonic acid—possess neither of these properties.

**ENUMERATION.**—Acids may be divided into *inorganic* or *mineral*, and *organic*. The mineral acids used in medicine are Boric, Carbonic, Chromic, Hydrochloric, Hydrobromic, Nitric, Nitro-hydrochloric, Phosphoric, Sulphuric and Sulphurous acids. The organic acids thus employed include Acetic, Benzoic, Carbolic, Carbonic, Chrysophanic, Citric, Gallic, Hydrocyanic, Lactic, Oleic, Salicylic, Tannic, Tartaric, and Valerianic.

**ACTION.**—The stronger acids—sulphuric, nitric, hydrochloric, chromic, glacial acetic and lactic acids—destroy animal tissues, and act as caustics when applied to the surface. When swallowed they produce the symptoms of irritant poisoning (*see* POISONS). An antidote for these poisons which is always at hand is carbonate of lime, in the form either of whiting or of plaster chipped from the nearest wall. Other antidotes are alkaline carbonates and bicarbonates, milk, oil and soap. Diluted acids, taken into the mouth, increase the secretion of saliva; and hydrochloric acid forms an important constituent of the gastric juice, without which digestion does not go on. When absorbed into the blood, dilute acids act on the heart generally, slowing its pulsations and reducing the temperature. They are excreted in the urine and milk.

**USES.**—Nitric acid is employed as a caustic application to piles, to poisoned wounds, and to spreading or unhealthy sores. Glacial acetic acid is used to destroy corns or warts. Diluted acetic acid or vinegar is applied as a lotion to relieve headache; to allay the itching of prurigo, lichen, and psoriasis; to check perspiration; and sometimes to hasten the appearance of exanthematous eruptions. Diluted acids, especially citric, tartaric, and hydrochloric, as well as acid tartrate of potassium, are administered in fevers as refrigerants, because they relieve the dryness of the mouth, and diminish the thirst by increasing the secretion of saliva, as well as lower the temperature and pulse-rate. Under the like circumstances, the organic acids, acetic, citric, and tartaric, when combined with alkaline carbonates in a state of effervescence or otherwise, form compounds which

act on the skin and kidneys. In febrile conditions, anæmia, and some forms of dyspepsia, the proportion of acid in the gastric juice is insufficient for the proper digestion of food, and the administration of dilute hydrochloric acid, immediately before or after meals, is useful both by aiding digestion and by preventing the formation of butyric and other acids, which give rise to sour eructations. Nitro-hydrochloric acid, before meals, is likewise beneficial in preventing acidity. It appears to have some action on the liver; and is used, both internally, and externally as a lotion or footbath, in jaundice and biliousness. It generally relieves the frontal headache common in young women, which is felt just above the eyebrows, and not accompanied by constipation. Dilute acids, especially aromatic sulphuric acid, are useful in checking diarrhœa, colliquative sweats, hæmorrhages, and mucous discharges. By lessening the alkalinity of the urine, they tend to prevent the formation of phosphatic calculi; phosphoric, nitric, and lactic acids being most frequently employed for this purpose. Care must be exercised in their administration to nursing mothers, as they are excreted in the milk, and sometimes cause griping and diarrhœa in infants at the breast. Several acids have a special action of their own, and are considered under their respective groups, such as hydrocyanic and hydrobromic acids, which are sedative; boric and carbolic, antiseptic; chrysophanic, parasiticide; salicylic, antipyretic; gallic and tannic, astringent.

T. LAUDER BRUNTON.

**ACINESIA.**—See **AKINESIA**.

**ACTIREALE**, in Sicily.—Cold sulphur waters, and climatic health-resort. See **MINERAL WATERS**.

**ACNE.**—SYNON.: *Acne vulgaris*; *Acne adolescentium*; *Acne disseminata*; Fr. *Acmé*; *acné*; Ger. *Acne*.

**DEFINITION.**—A chronic disease of the skin, confined to the face, back, shoulders, and chest. The eruption is met with in young adults, and consists of pimples, which are caused by an inflammation of the sebaceous glands and hair-follicles. The disease leaves small depressed scars.

**ÆTIOLOGY.**—The ætiology of acne is obscure. It is seldom met with before puberty, and is usually fully developed at about the age of eighteen or twenty, and then gradually disappears before the age of thirty. There are, however, many exceptions to this rule, especially in men, who are liable to a severe form of acne of the shoulders and back, which may continue until middle life. Acne appears to be equally common in males and females, and is especially met with in those who have what is called a slow circulation in the extremities—that is, who suffer

from cold hands and feet, and perhaps chilblains. In accordance with this, the disease often improves, or even disappears, during warm summer weather, to return again in the following winter.

**SYMPTOMS.**—Common acne is a pimply eruption, met with chiefly on the face and shoulders, less often on the back and chest. Many of the pimples are of a pale colour with a small dark centre, and consist simply of follicles, over-distended with sebum, the black centres marking the orifices of the follicles. These pale little papules, called *comedones*, are apt to become red and inflamed, thus forming the ordinary acne pimple, which ultimately suppurates, and discharges a minute quantity of pus; the spot then heals, leaving a small scar. In all severe cases of acne we find, in addition to the ordinary acne spots, larger pimples, resembling blind boils, which suppurate slowly, and often leave very ugly scars. Acne develops in successive crops, so that, as one set of pimples dies out, others appear, and thus the disease becomes chronic. Acne is almost always distributed symmetrically, so that both sides of the face and back are pretty equally affected. The exceptions to this rule are very rare.

**DIAGNOSIS.**—The differential diagnosis of acne is usually easy when the symmetrical distribution of the eruption, and its period of development, are taken into consideration. The two eruptions with which it is most likely to be confounded are: (1) certain forms of acne rosacea (*gutta rosea*), and (2) acne-like dermatosyphilis. Acne may best be distinguished from acne rosacea by the fact that this latter disease is chiefly met with in middle life, and is always attended with more or less general congestion of the face, and the subjective sensations of burning or tingling—characters which are absent in common acne. Acne rosacea is exclusively confined to the face, while common acne attacks also the shoulders and back.

Acne-like syphilitic eruptions are best distinguished by the absence of comedones; and by the tendency the pimples have to form into groups, which are not always symmetrical, and not strictly confined to the acne regions. Some other syphilitic symptoms would probably be also present.

**VARIETIES.**—There is an interesting eruption, commonly called *acne varioliformis*, which requires a very brief notice. In general appearance the pimples resemble those of acne, but are not especially associated with comedones. The eruption is usually confined to the scalp and upper part of the forehead. The scars formed by this disease are much deeper than those of common acne, and very closely resemble the pits left by small-pox. The region affected and the character of the scars are a sufficient guide to diagnosis

**TREATMENT.**—As acne arises, for the most part, from the formation of comedones and plugging of the follicles, the treatment should be directed to stimulating the sebaceous glands of the skin to action. For this purpose the application of sulphur is most useful. The sulphur may be applied in the form of an ointment or lotion. The objection to a lotion is, that during the night the powdered sulphur is apt to get into and inflame the eyes a little. There is not, however, the same objection to the use of a sulphur lotion during the day-time. A good plan is to apply a mild sulphur ointment or Wilson's hypochlorite of sulphur ointment every night, and wash it off in the morning with hot water, soap, and a soft flesh-brush. The daily use of a soft flesh-brush with hot water and soap is especially beneficial, as it prevents the formation of comedones.

When sulphur does not produce the desired effect, the linimentum saponis should be applied every night and allowed to dry on, and be washed off in the morning with warm water. In very severe cases of acne, there are always a certain number of boil-like spots, which suppurate very slowly, and sometimes last for months without coming to a head. These boils should be carefully touched with a small piece of wood dipped in pure carbolic acid, or a saturated solution of potassa fusa in water; or the acid nitrate of mercury solution may be used, great care being taken that only the top of the boil is touched with the caustic. Cod-liver oil is the most generally useful tonic in cases of acne, but other tonics are occasionally beneficial. ROBERT LIVEING.

**ACNE ROSACEA.**—SYNON.: *Gutta rosea*; Fr. *Couperose*; Ger. *Das kupfrige Gesicht*.

**DEFINITION.**—A chronic congestion of the face, attended with sensations of burning or stinging, and leading to a permanent enlargement of vessels and the formation of pimples.

**SYMPTOMS.**—There are two varieties of acne rosacea. The more severe form is met with chiefly in men, and is often the result of over-indulgence in alcohol. It especially attacks the nose, and leads to considerable enlargement of the vessels, and also, if of long standing, to hypertrophy of other parts of the skin, especially the sebaceous glands. The milder form of gutta rosea is very common in women after the age of thirty. In the first instance it may consist of a simple flushing of the face, coming on at certain periods of the day, as, for example, after meals or in the evening. This congestion has, however, a tendency to become more persistent; and then the vessels being permanently dilated, the redness does not entirely disappear, although it varies in degree from time to time. *Gutta rosea* in women is not confined to the neighbourhood

of the nose, but usually extends in a butterfly-shaped patch to both cheeks, and sometimes to the forehead and chin. It is always attended with subjective sensations, which are generally those of burning, and less frequently of stinging or itching. Acne-like pimples are only occasionally met with, and when present are due to follicular inflammation.

**ÆTIOLOGY.**—*Gutta rosea* in women is for the most part associated with cold hands and feet, and not uncommonly with dyspepsia and constipation, or irregularity in menstruation. In men, as before stated, the hypertrophic variety is sometimes, but by no means always, caused by over-indulgence in alcohol.

**TREATMENT.**—The severer form of gutta rosea, in men, is best dealt with by: (1) abstinence from alcohol; (2) the longitudinal division of the enlarged veins with a lancet; and (3) the subsequent daily use of sulphur ointment, which should be well rubbed on every night. Great hypertrophy of the nose (*lipoma nasi*) can only be removed by means of the knife. In treating gutta rosea, as it is commonly met with in women, the following points require special attention. (1) The diet should be carefully regulated, and medicines ordered to remove, as far as possible, any dyspepsia or any menstrual irregularity that may exist, and also to regulate the bowels, which are often constipated. (2) A very mild sulphur (or hypochlorite of sulphur) ointment should be applied in a small quantity every night, and washed off in the morning with warm, soft, oatmeal-water. In the daytime, a calamine and oxide of zinc lotion should be applied, and a little allowed to dry on. *Gutta rosea* is difficult to cure, but a steady perseverance in a rational plan of treatment will always produce a marked improvement in the disease, and sometimes quite remove it. ROBERT LIVEING.

**ACONITE, Poisoning by.**—SYNON.: Fr. *Empoisonnement par l'Aconit*; Ger. *Eisenhutvergiftung*.—The common garden-plant, *Aconitum napellus*, known also as 'wolfsbane' or 'blue-rocket,' as well as other species of *aconitum*, are poisonous, and owe their poisonous properties to the presence of an alkaloid, *aconitine* or *aconitia*, or perhaps to a mixture of alkaloids passing under this name. The same or similar alkaloids have been obtained from the Indian aconite, *A. ferox*, and from Japanese aconite roots. All parts of *A. napellus* are poisonous. Aconitine is perhaps the most poisonous alkaloid known. All parts of the aconite plant when chewed, and aconitine when placed upon the lips or tongue, produce, after a few minutes, a disagreeable acrid burning sensation, followed by numbness, loss of sensibility of the part, salivation, and an after-sensation of seariness. These sensations may last for several hours.

The fresh root of aconite has frequently been eaten in mistake for horse-radish, to which it bears a remote resemblance. The root of horse-radish is whitish on the exterior, is long and of fairly uniform diameter, has a pungent odour when scraped, and the scraped surface retains its white appearance; whereas aconite root is brown and conical, is destitute of pungent odour, and speedily acquires a pink colour when scraped and exposed to the air. Mistakes more frequently occur from liniments containing aconite being swallowed in error. In two cases the root has been administered with homicidal intent; and in one case a young man was killed by the administration of, as it is supposed, two grains of English aconitine. Accidents have also arisen from the administration of the potent English aconitine in mistake for the impure inert exotic or German alkaloid, or mixture of alkaloids passing under that name.

**ANATOMICAL CHARACTERS.**—After poisoning by aconite there may be gastric congestion or inflammation; but these may be absent.

**SYMPTOMS.**—When aconite, or any of its preparations, is taken by the mouth, the first sensation, transitory and mainly due to the action of the solvent, is followed in about three minutes by an intolerable burning and numbing pain, extending from the place of application to all the surrounding parts of the mucous membrane. There is salivation; and the burning sensation extends down the gullet to the stomach. Occasionally, when the poison has been rapidly swallowed, no marked symptoms may supervene for half an hour. The general symptoms are very varied, but may all be referred to weakening of the heart's action, disturbances of respiration, and paralysis of sensation on the surface of the body. This last may be described as 'numbness' or 'drawing of the skin,' or by some equivalent term. There is pain in the epigastrium, violent vomiting, occasionally purging; the pulse, at first rapid, quickly diminishes in frequency and force till it is imperceptible; the skin is cold, clammy, and livid; respiration is laboured. The pupils, at first contracted, afterwards dilate; and this dilatation sometimes occurs suddenly and transitorily, and is accompanied by blindness. Convulsions are not common; but vomiting is often due to spasmodic contraction of the diaphragm, causing frothing at the mouth. Consciousness is retained till near the end of life.

**DIAGNOSIS.**—The peculiar sensation in the mouth—burning, feeling of searedness, numbness, &c., the great cardiac depression, and the difficulty of respiration, will generally serve to determine the nature of the case.

**PROGNOSIS.**—Death usually occurs within four or five hours. If the patient survive twelve hours, recovery is usually rapid and complete.

*Fatal dose.*—Of the root. sixty grains—

probably much less might suffice. Of the pharmacopœial *tinctura aconiti* (1 in 8), two or three fluid drachms. *Fleming's tincture* is about six times as strong as the official tincture, and twenty-five minims have proved fatal. Four grains of the *alcoholic extract* have proved a fatal dose. English *aconitine* or *aconitina* (the alkaloid) is terribly potent: 1-2000th grain will produce a very decided sensation on the tongue, and it is probably as poisonous as the crystallised French aconitine-nitrate, one-sixteenth of a grain of which has killed an adult within five hours.

**TREATMENT.**—In proceeding to treat a case of poisoning by aconite we must, first, wash out the stomach by means of the stomach-pump, and promote vomiting by warm emetics, of which carbonate of ammonium is the best. Stimulants must be freely administered; also strong black coffee or tea. Brandy and ether may be injected subcutaneously. Digitalis is a counter-poison, and may be administered with effect subcutaneously, in doses of twenty minims of the tincture, repeated in an hour or so if necessary. Inhalations of nitrite of amyl may afford some relief. The patient must be kept strictly in the recumbent position, warmth being applied to the surface; and, as a last resort, artificial respiration must be used. *See POISONS.*

THOMAS STEVENSON.

**ACQUI**, in the Province of Alessandria, Italy.—Thermal sulphur waters. *See MINERAL WATERS.*

**ACQUIRED DISEASES.**—Diseases which originate independently of hereditary transmission. *See DISEASE, Causes of.*

**ACROCHORDON.**—An outgrowth of the integument in the form of a slender cylinder, which may be compared to the loose end of a piece of string or cord—*ἄκρον* signifying a point or end, and *χορδή* a string. Such outgrowths are usually met with in a feeble state of the skin, and particularly in elderly persons, their common seat being the neck or trunk. They are at first sessile, but become elongated; and are sometimes bulbous at the extremity, and more or less pedunculated. Microscopically, an acrochordon is composed of loose areolar tissue, firmer at the surface than within, and of a fine artery and vein, connected by a capillary loop or plexus, and sometimes a little ramified. It is popularly regarded as a wart, and in medical works is termed *verruca acrochordon*, but it differs from a wart very widely in structure.

**TREATMENT.**—This consists in snipping off the outgrowths with scissors, or touching them with a strong solution of potassa fusa (equal parts). When numerous and minute, they admit of being shrivelled up and removed by means of liquor plumbi subacetatis, or a lotion of perchloride of mercury, two grains to the

ounce. The latter, possibly by its stimulating property, also arrests their formation.

ERASMUS WILSON.

**ACRODYNIA** (*ἄκρος*, extreme; and *ὀδύνη*, pain).—This disease was epidemic in Paris in 1828, and was described by Alibert as a dermatitis affecting the hands and feet, particularly the palms and soles, accompanied with burning heat, stinging and smarting pains, and numbness. The pains sometimes extend to the whole system, and there is more or less disorder of the digestive and assimilative functions. The redness is at first bright, then deeper tinted and brown, with considerable pigmentation of the rete mucosum. Occasionally there are pimples, pustules, and blisters; the cuticle desquamates, and is sometimes cast in a single piece; the disease running on for several weeks.

**TREATMENT.**—This should be directed to the regulation of the digestive and assimilative organs; and to the relief of local inflammation by means of water-dressing, followed by the application of zinc ointment and a bandage.

ERASMUS WILSON.

**ACROMEGALY** (*ἄκρον*, an extremity; and *μέγας*, large).—**SYNON.**: Fr. *Acromégalie*.

**DEFINITION.**—A remarkable dyscrasia, the striking characteristic of which is an enlargement of the bones, and most obviously of the bones of the hands and feet. Hence the name applied to it by Marie, who first described the disease in 1880, though other cases had been noted previously under different titles.

**PATHOLOGY.**—Acromegaly occurs most frequently, but not exclusively, in females between the ages of thirty and forty, but has been observed as early as fifteen and as late as forty-eight. It is very chronic; and though probably often fatal, it sometimes appears to become arrested after advancing for some years. In females it is usually preceded by early stoppage of the menses. Its relation to rheumatism is very doubtful.

Acromegaly is generally associated with enlargement or atrophy of the thyroid body; and enlargement of the thymus gland has been observed. In three post-mortem examinations tumours of the pituitary body have been met with.

**DESCRIPTION.**—In this disease the bones of the hands and feet are very greatly enlarged, but the long bones do not escape, the natural prominences and elevations being exaggerated sometimes to the dimensions of exostoses. The scapulae, clavicles, and the sternal ends of the ribs become quite massive; the lower jaw also assumes vast proportions, the lower teeth spreading out and projecting far in front of those of the upper jaw. This gives a characteristic appearance to the face, which is that of an egg with the large end downwards, as opposed to the oval of myxœdema, and the inverted

egg shape of osteitis deformans, with both which diseases acromegaly may be confounded. The upper jaw and the cranial bones occasionally participate in the change. The bones, though enlarged, do not become curved; but the spine is often bent, so that the patient loses considerably in stature.

The cartilages of the nose, ears, eyelids, and larynx may be thickened; the last change probably accounting for an occasional great alteration in the voice.

The subcutaneous tissues are not obviously altered. The skin may be unchanged, but on the face it is apt to become coarse, and to show clearly the orifices of the sweat-glands. The hair does not become thin, as in myxœdema. Profuse perspiration and thirst are generally present.

The tongue is enlarged, and taste may be much impaired. Blindness is not uncommon, from atrophy of the optic nerve, possibly connected with a pituitary tumour. The sense of smell is often impaired; that of hearing does not appear to be modified, nor does that of touch.

There may be shortness of breathing, depending upon asthmatic attacks, and the heart may be hypertrophied. The urine is not usually abnormal. The temperature is not raised. The intellect remains perfect.

**DIAGNOSIS.**—It is not necessary to indicate the difference between this disease and osteitis deformans or myxœdema—practically the only two with which it can be confused, because, though there is certainly a superficial resemblance to each, the points of divergence are wide. The symptoms have been stated somewhat dogmatically for the sake of brevity; and, as only a few cases have as yet been accurately observed, it is probable that some of those here set down may be found to be incorrect or not characteristic.

**TREATMENT.**—None of the numerous methods of treatment hitherto applied in acromegaly have proved of the slightest use.

RICKMAN J. GODLEE.

**ACTINOMYCOSIS** (*ἄκτις*, a ray; and *μύκης*, a fungus).—**DEFINITION.**—A chronic disease, attended by the formation of abscesses, chronic interstitial inflammations, or tumour-like growths of granulation-tissue, due to infection by a fungus known as *Actinomyces bovis*.

**HISTORY.**—This disease, which especially affects horned cattle and swine, and has long been known under various names as occurring in them, has during the last few years been proved to be not infrequent in man. Its parasitic nature was discovered in 1876 by Bollinger, who named the parasite *Actinomyces*, the ray-fungus. In 1877 J. Israel, and in 1879 Ponfick, described cases in the human subject, and showed their identity with the animal disease. Since that time numerous instances of the disease have been

observed; with chronic abscesses, especially in the liver, lungs, bones and periosteum, intestinal canal—especially the large intestine—and more rarely in other positions, *e.g.* in the brain, as in recent cases recorded by Dr. Delépine and others.

**GENERAL CHARACTERS OF THE DISEASE.**—In the lower animals actinomycosis frequently produces indurated masses of connective-tissue growth, which tend to become calcified; hence the name 'woody tongue' applied to the affection in cows. In man there is a tendency rather to the formation of chronic abscesses. Infection usually appears to occur by the alimentary canal or air-passages, the virus entering through carious teeth, the crypts of the tonsils, pharynx, &c., or more rarely by direct aspiration into the lungs. Most commonly it tends first to produce destructive changes, for example, in the jaws (upper and lower), the wall of the pharynx, or the mucous membrane of the alimentary canal. In the jaws a process of gradual necrosis may occur. From the upper jaw it is apt to spread to the periosteum in front of the vertebræ, or to the base of the skull, leading to chronic periosteal abscesses, followed by caries, sometimes with retro-pharyngeal abscess. It may next invade the tissues of the neck, forming deep-seated abscesses. Not infrequently in man, however, the liver is the first organ obviously attacked. It has been shown that a primary infection of the mucous membrane of the intestines, especially the large intestine, may occur, and not improbably the infection of the liver is secondary to this. Affection of the lungs may take place by direct aspiration of the virus, or possibly through infection extending from the mediastinum, which has been reached through the œsophagus. Infection of the brain and other parts may take place either through the lymphatics or through the blood.

**ANATOMICAL CHARACTERS.**—Four varieties of the chronic inflammatory process have been determined in actinomycosis:—

First, when upon a free surface, as the mucous membranes, the formation of masses of epithelium containing the fungus has been observed, together with subjacent ulceration.

Second, when occurring upon a free surface, as in serous cavities or within the periosteum, tumour-growths may occur, formerly mistaken (when periosteal) for osteo-sarcomas.

Third, the formation of masses of dense granulation-tissue or sarcoma-like tumours, which may become fibroid or calcified, but remain indurated. Allied with this there may be an infiltration with firm granulation-tissue.

Fourth, the production of chronic abscesses, which soften slowly, causing widespread infiltration and necrosis of the tissues, but are not usually accompanied by marked inflammatory reaction. In the pus from these abscesses, which is usually thick, yellow, and somewhat lumpy, may be found minute

yellowish granules or masses, varying in size from the most minute particles visible with the naked eye, to the size of about  $\frac{1}{10}$  inch in diameter, of yellowish colour, ovoid or rounded, and of slightly greasy feel. Under the microscope these are found to consist of masses of the ray-fungus.

On microscopical examination of prepared sections, especially in the firmer nodules, the characters can be better seen. Various modes of staining may be employed, for which the reader is referred to special technical works, gentian-violet, fuchsin, and methylene

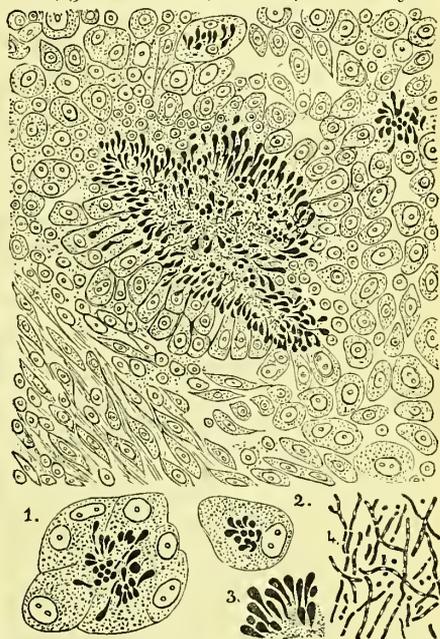


FIG. 1.—The wood-cut shows the characters of a minute centre of growth in an actinomycosis nodule from the tongue of a cow. The upper portion is sufficiently described in the text. The mass of fungus is represented as much less dense than in reality, otherwise no detail could be shown. (Magnified about 800 diameters.)

The lower four small figures show the characters seen in the same growth under a somewhat higher power. (1) A group of cells (? one cell in a state of division) containing a cluster of the clubbed filaments. (2) A single cell with actinomyces growing in it. (3) A group of clubbed hyphæ much larger than the others. (4) Filaments from centre of mass, showing bacillus-like characters, and false branching; resembling cladothrix.

blue being amongst the most suitable reagents. The structure is composed of granulation-tissue, either small or large-celled, frequently with branching and spindle cells, resembling spindle-celled sarcoma. Scattered through the granulation-tissue are deeply stained masses, which form the centre of more typically arranged masses of cells. The fungus itself, when seen in the smaller nodules, is usually composed of a central

portion containing coccus-like bodies, and delicate, branching, bacillus-like filaments. At the periphery of the mass the filaments are arranged side by side and radially, their outer extremities being swollen and bulbous-looking or club-like. Immediately surrounding these a zone of large epithelioid granulation-cells may be observed, the cells sometimes being arranged radially and resembling columnar epithelium. Still further out are epithelioid cells in layers; and beyond these are smaller cells, gradually merging into the surrounding granulation-tissue. Considerable varieties of structure are, however, observed, which need not be detailed here. The epithelioid cells may be scanty, or irregularly arranged, and of small size. In general arrangement these centres recall somewhat that of an elementary tubercle-follicle. The masses of fungi also present varieties. When of large size they frequently show zones of calcification. Club-shaped bodies may also be found scattered more irregularly through the mass. The branching, delicate threads may be numerous or scanty; and masses of coccus-like bodies may be observed.

Much discussion, which it would here be undesirable to detail, has taken place as to the nature of the fungus, and the exact relations of its different forms. The general belief now is that it belongs, or is closely allied, to the *cladothriceæ*, and that the several forms are various stages of its growth, the coccus-like bodies being spores, the filaments the earlier stages of its growth, and the club-like bodies swollen filaments or *hyphæ*—swollen in consequence of defective nutriment; some regard them, however, as possibly *asci*. The fungus has been successfully cultivated by Bostrom, and in its general characters appears closely to resemble the *cladothriceæ*.

For further details reference may be made to special works.

**MODE OF INFECTION.**—The general evidence at the present time renders it probable that the infection of actinomycosis is introduced by food, and especially by some varieties of grain on which the fungus grows. Grain grown on freshly cultivated ground has in some cases apparently led to an outbreak amongst cattle. In one case in the human subject, recorded by Soltmann, a child swallowed a head of wall-barley (*hordeum murinum*), which stuck in the œsophagus and was followed by the development of an actinomycotic abscess (Klebs, *Allg. Path.* 1887, p. 286). Direct transmission by contagion has been observed by Johnes and Ponfick; and by inoculation of material from abscesses (Fleming, *Actinomycosis*, 1888; also Crookshank, *Med. Chi. Trans.* vol. lxxii. p. 207). Organisms similar to the actinomyces grow on various kinds of grass.

**GENERAL COURSE.**—Actinomycosis is usually extremely chronic. When primarily

affecting the *lungs*, it tends to spread locally and involve the pleura, the periosteum of the ribs, and the tissues of the thoracic wall, forming sinuses which open externally. In some cases the fungus has been found in the sputum without definite physical signs in the lungs. In the *liver* the abscesses may also reach a considerable size, and tend to form adhesions and fistulous tracks through the abdominal wall. In the *neck*, as has been stated, a slow process of extension may take place to the base of the skull, the vertebrae and their periosteum, and the deeper tissues of the part, forming in some cases very large chronic abscesses, which when opened show little tendency to heal. Direct spread of the infection by the lymphatics has been observed, but the production of abscesses in the brain can scarcely be accounted for except by carriage through the blood-stream. The fungus, like other organisms, is frequently found, especially in the coccus and rod forms, within granulation-tissue cells and leucocyte-like cells in the local affection; and it is not improbable that these simpler forms may be carried throughout the body by wandering cells and leucocytes. The abscesses are sometimes unattended by fever, but abscesses in the lungs and liver have sometimes been accompanied by recurrent rigors.

**DIAGNOSIS.**—This in many cases can only be determined by microscopical examination of the discharges or the pus. A very chronic slowly-spreading abscess-formation of undetermined cause should lead to suspicion of actinomycosis. The absence of fever cannot be relied upon. In the microscopical examination of the sputum care must be taken not to confound the fungus with very similar organisms which may occur in carious teeth, &c.

**TREATMENT.**—Little can be said on this subject. Complete evacuation of abscesses, and (where possible) irrigation with dilute antiseptic solutions, would no doubt be indicated. From our present knowledge it is improbable that any internal remedy could be safely given in sufficient doses to destroy the fungus. The general treatment must rather be directed to the nutrition and support of the vital powers of the patient.

W. S. GREENFIELD.

**ACUPUNCTURE** (*acus*, a needle; and *punctum*, a prick).—**SYNON.**: Fr. *acupuncture*; Ger. *Nadelstich*.

**DESCRIPTION.**—Acupuncture is an ancient mode of treatment for the relief of painful affections, now but little used, consisting in the introduction of fine round needles through the skin, to a varying depth. It is said to have been introduced into this country from China or Japan, about 200 years ago. The needles used are about two inches in length, and are set in round handles, so that they can be introduced with a gentle, rotatory movement. Acupuncture is now employed

chiefly in lumbago and sciatica, in which affections it undoubtedly may give relief. The operation is thus performed. The patient being laid upon his face, tender spots are sought for—in lumbago over the erector spinæ, in sciatica along the course of the sciatic nerve. The needles are then pushed in vertically for a depth of from one and a-half to two inches, and allowed to remain for from half an hour to two hours. The number of needles employed may vary from one to six. In sciatica it is recommended, if possible, to make the needle actually penetrate the nerve. This result is known by the patient complaining of sudden pain shooting down the back of the leg. The mode of action is uncertain, but in sciatica it has been supposed that the puncture of the nerve-sheath allows the escape of fluid. Acupuncture has also been employed in painful neuritis following injury, but without effect. In a modification invented by Bausnscheidt, forty punctures, about half-an-inch in depth, were made in an area of the size of a crown-piece, by an instrument working by a spring. Oil of mustard, diluted with olive oil, was then painted on, which gave rise to an eruption like herpes. The term *acupuncture* is also applied by some to the introduction of needles into a cyst, in order to allow the fluid contents to escape, as in the treatment of ganglion, of hydrocele in infants, or of hydatid cyst of the liver. Puncture of the skin for the relief of œdema is sometimes called by the same name. For this purpose the ordinary three-cornered acupressure-needle is more convenient than a round acupuncture-needle, as the puncture resulting from it allows fluid to escape more readily. The number and situation of the punctures being determined upon, they should be made in rapid succession and of sufficient depth to allow a free escape of the fluid. **MARCUS BECK.**

**ACUTE.**—This word, when associated with a disease, signifies that such disease runs a more or less rapid course, and is generally attended with urgent symptoms. It is also employed to express intensity of a particular symptom, as, for example, pain.

**ADDISON'S DISEASE.**—**SYNON.:** *Morbus Addisonii*; Bronzed Skin Disease. *Fr. Maladie d'Addison*; *Ger. Addisonische Krankheit.*

**DEFINITION.**—In his original memoir on this subject, Dr. Addison wrote: 'The leading and characteristic features of the morbid state to which I would direct attention are anæmia, general languor and debility, remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of colour in the skin, occurring in connexion with a diseased condition of the suprarenal capsules.'

In reality, the general symptoms of the

disease, as given above, outweigh in importance any pigmentary change whatever; and it is quite possible for the disease to run its course without any unusual deposit of pigment in any part of the body.

Addison's disease might, however, be defined as a constitutional malady, characterised by great weakness and anæmia, with deposit of pigment in the skin and some other parts of the body, depending on chronic tuberculosis of the suprarenal capsules, and the extension of fibrous thickening to the adjacent nerve-ganglia and plexuses, causing their compression and atrophy.

**ÆTIOLOGY.**—The constitutional or general nature of Addison's disease must ever be borne in mind, though some of its factors are strictly local; but, though constitutional, it is not transmissible either (*a*) by contagion or infection, or (*b*) by inheritance. If, however, Addison's disease itself is never an inherited malady, it is in very many cases associated with a highly hereditary constitution, that of the tubercular or scrofulous type; and in such individuals accidents, like falls or blows, which would fail to make an impression on stronger men or women, may suffice to set the morbid process in motion. In not a few instances the bodies of the subjects of this disease have been found perfectly healthy apart from the morbid change in the suprarenal capsules characteristic of the malady; and, in a certain number of cases, local abscesses seem to have been the starting-point of the specific changes in the capsules themselves. Addison's disease is, moreover, essentially one of early adult life, the great majority of cases occurring between fifteen and forty. It is much more frequent in men than in women; and seems to be in a great measure confined to the working classes.

**SYMPTOMS.**—The earlier symptoms of Addison's disease are often so indefinite and so insidious, that it may not be until the disease is fully developed that the patient seeks medical aid, and by that time they are usually unmistakable. It is different when the malady apparently originates in a fall or a blow; but even such a starting-point as this may only be sought for late in the history of the disease.

The mischief may be said to commence in most cases with a feeling of general weakness and of being unwell; the discolouration of the skin usually appears later, but may be the first prominent symptom. In a small number of cases the onset may be acute, with loss of appetite, sickness, headache, pain in the epigastrium, sometimes also vomiting and diarrhœa. When the disease has attained its full development, the characteristics of the malady are most striking. Then the downcast, mournful look, the drooping shoulders and stooping gait, the arms hanging helplessly by the sides, and the slow

and listless movements of the patient are exceedingly impressive. If to this be added the darkening of the skin, the clear and pearly conjunctiva, and the breathlessness on exertion, we have almost all that meets the eye when such a patient presents himself. But to these, on inquiry, other important symptoms are easily added. The breathlessness will be found to be partly due to anæmia, partly to impaired innervation. From the same causes in part, but not entirely, we find a quick feeble action of the heart, readily giving place to palpitation. With these are usually associated pain and tenderness in the epigastrium and hypochondria, irritability of the stomach and nausea—giving rise to retching, and frequently obstinate vomiting. Such modifications of breathing as sighing, yawning, or hiccup, are frequent. Again, from the anæmia, there is a strong tendency to giddiness and syncope, which last increases as the disease wears on, and in many cases carries off the patient when raising himself or being raised in bed, for the purpose of taking food or performing other necessary functions. This is not the invariable mode of death, for nervous symptoms, such as coma or convulsions, may usher in the final scene. When the prostration is great, the patient may be for some time before death apparently unconscious, but this is simply due to unwillingness to make the slightest exertion, owing to his profound weakness.

Throughout the whole disease the bodily temperature is diminished ( $97^{\circ}$ – $98^{\circ}$  Fah.) rather than increased; and this is often markedly the case towards the close of the disease, though then it has been noted as high as  $100.8^{\circ}$  Fah.

From the above sketch it is plain that the two most prominent factors in the disease, as presented during life, are:—

1. General weakness and anæmia.
2. Abnormal deposit of pigment in various parts of the body.
  1. To the former of these is to be referred (a) the loss of muscular power, as evidenced by diminished muscular energy and force, both in the voluntary and involuntary muscles, for the heart's action is feeble and imperfect, and the bowels are usually confined. (b) At once a cause and consequence of the weakness and anæmia are loss of appetite, sickness and vomiting, though these, too, depend in part on other morbid changes; whilst, lastly (c), imperfect nutrition of the nervous system results, notably of the brain itself, whence arise vertigo, numbness, dimness of sight, deafness, tremors, and the like. The pain in the epigastric and hypochondriac regions is probably due to local causes.
  2. The deposit of pigment is peculiar and characteristic. It is not uniform in disposition, and varies greatly in tint. It may only amount to a light brown or smoky discoloura-

tion in certain parts, or it may assume the appearance of a dark olive-green hue, approaching to black in some situations, especially over the genitals and nipples. Elsewhere it is most abundant on the face (where it often seems to begin), on the neck, the backs of the hands, the folds of the legs, and along either side of the *linea alba*. A striking peculiarity is that the conjunctivæ are clear and pearly, and that the nails are never discoloured. On the other hand, there is a great tendency to the deposit of pigment where the skin has been irritated or the epidermis removed, as by a mustard poultice or blister; but the skin is always smooth and supple. In a typical case under the writer's care, blisters had been applied to the chest for the uneasy feeling experienced there; and the pigmentary tint was deeper here than in any other part of the body, save the genitals. Cicatrices affecting the whole depth of the skin do not seem to be so pigmented. The mucous membrane of the mouth not infrequently becomes the site of pigmentary deposits. These are not diffuse; but, when the lips are affected, they usually take the shape of smears or lines. On the insides of the cheeks blotches or irregularly-defined spots are most common, as well as on the sides and root of the tongue. The latter spots are commonly better defined than are the others, and somewhat resemble the small well-marked black spots occasionally observed in parts already pigmented.

The site of this pigmentary deposit is in the growing layer of the epidermis, the usual site of colour in all races of mankind, and which is usually known as the *rete mucosum*; but occasionally pigmentary granules are to be found deeper, in the cells of the true skin.

An interesting clinical fact was brought out by the late Dr. Greenhow, which will, probably, be noted in a considerable proportion of cases. This is the mode in which the disease progresses. Often it presents periods of remission, only to be followed by a more marked advance; but, notwithstanding these remissions, the progress is invariably towards a fatal termination. The time occupied in this progress varies much: it may be weeks or months, or it may be years. But in all well-defined cases the result is the same.

**PATHOLOGY.**—From the earliest description of the morbid state known as Addison's disease, the malady has been associated with disease of the suprarenal capsules. At first it was supposed that any form of disease affecting these organs must give rise to a similar train of symptoms, and some of the investigations carried on with a view to sustain this position sound absurd enough by the light of subsequent experience. Gradually it has been made clear that only one kind of lesion is accompanied by the specific symptoms just detailed. Briefly, the morbid changes are as follows:

The suprarenal capsules normally consist of two parts, a cortical and medullary, differing greatly in their structure. In Addison's disease both are superseded by a new structure, which is to be seen in various stages. In the earliest of these the capsules are invaded by a kind of translucent material, which is sometimes almost cartilaginous in its hardness, and which, when examined under the microscope, resolves itself into a kind of very finely fibrillated or trabecular connective tissue, with corpuscles like leucocytes freely congregated in the interstices of the meshwork or between the fibres. This material, when seen in bulk, is grey or greenish-grey, afterwards becoming red on exposure. With it is mixed up an opaque yellow substance, varying in amount, and apparently more abundant the more advanced the disease. In the earlier stages it presents the appearance of nodules embedded in the translucent material, but later almost the whole of this material may have disappeared, and the yellow opaque matter become converted into a thick creamy fluid, a putty-like substance, or even one or more cretaceous masses. This opaque material, then, is evidently, from its first appearance, indicative of fatty degeneration, and closely resembles in every respect what used to be known as yellow or crude tubercle. The more recent discovery of the tubercle-bacillus in several cases confirms Virchow's original view as to the essentially tubercular nature of the affection.

The exterior of the capsules presents certain important features. The capsules themselves may be large or small, according to the stage of the disease and the nature of their contents, but, even when they are small, it may be safely assumed that at one time they were enlarged. In all cases they will be found closely and strongly adherent to neighbouring structures. Some of these structures are of great importance: for example, the semilunar ganglia, and the vast plexus of nerves associated therewith, in which important changes have been found. These pathological conditions have been so often observed and so carefully noted, that they cannot be looked upon as accidental concomitants of the diseased process, but rather part and parcel of it, and in all probability as giving a clue to some of the most marked phenomena of the malady. Broadly it may be said that these changes consist in a great thickening of the connective tissue surrounding the nerve-fibres and the ganglion-cells, giving rise to something like compression and ultimate destruction of these elements. This occurs both in the cerebro-spinal nerve-fibres, and in those more intimately connected with the ganglionic system. The nerve-cells, moreover, are not infrequently deeply pigmented.

The view commonly held as to the actual pathology of Addison's disease is that its

phenomena are due to the nerve-lesions just described, and not to the destruction of the suprarenal capsules, though the exact mode in which they produce these effects is at present a mere matter of speculation. According to another view the destruction of the suprarenal bodies is the cause of the prominent symptoms, and this is supported by Dr. MacMunn, as the result of his spectroscopic observations on the blood and urine. He believes that the function of these bodies is to separate effete pigments and their accompanying proteids. When they are diseased, these elements are not removed, but circulate in the blood, the pigments or their incomplete metabolites producing pigmentation of the skin and mucous membranes, and appearing often in the urine as uro-hæmato-porphyrin; the proteids setting up toxic effects, and leading to further deterioration of the blood, with its consequences.

In making the section of the body after death from Addison's disease, one cannot help being struck with the amount of subcutaneous fat, especially over the abdomen, as contrasted with the difficulties of nutrition under which the patient suffered; yet a considerable quantity is almost always found. But more closely connected with this malnutrition are certain changes in the absorbent system along the digestive tract. These consist in enlargement of the solitary and agglomerated glands constituting Peyer's patches, and of the mesenteric glands; as well as in lymphoid growths in the mucous membrane of the stomach, which give rise to little projections, termed *mammillations*, on the walls of that organ, especially near the pylorus. Small ecchymoses are also not unusually found in the same situation. Of other organs it may be noted that the liver and spleen are often enlarged and hyperæmic, and the heart small and light in weight.

DIAGNOSIS.—There would have been less difficulty or doubt in the diagnosis of Addison's disease, had it been clearly enunciated from the first that a bronzed skin did not alone constitute the malady. The disease rests on a threefold basis—*general weakness, diseased suprarenal capsules, and bronzed skin*, the last being the least important of the three. There may be darkening of the skin from a great variety of causes, viz.: (a) exposure, and attacks of vermin (*morbus erroneum*, Greenhow; *Vagantenkrankheit*, Vogt); (b) wasting diseases, as chronic phthisis; (c) syphilis; (d) malaria; (e) liver-disease or jaundice if long-continued; &c.; but in none of these cases should there be any difficulty if the preceding dictum is borne in mind.

PROGNOSIS.—This is always unfavourable, but it is impossible to assign any definite period for the termination of the disease, since it often progresses irregularly, with periods of improvement followed by relapse.

**TREATMENT.**—From what has just been said, it is plain that not much is to be effected by treatment as regards the cure of Addison's disease; but much may be done by careful management to retard its progress and comfort the patient. As soon as the disease is discovered, the sooner the patient makes up his mind to an invalid life the better. Rest and careful dieting are the basis of the treatment. As regards diet, it may be briefly said that what the patient can take best suits best. As the stomach is so irritable, anything likely to upset it should be avoided. Hence, as a rule, it is better to give concentrated nourishment, as essence of meat (*not the extract*) or chicken, or raw pounded meat, when other things cannot be taken. It is also important to bear in mind that the stomach will often tolerate food cold or even frozen, when hot substances would be promptly rejected. In certain stages of this malady it may well be said that the physician's success will depend more on his knowledge of the cookery book than of the Pharmacopœia—not, however, that our pharmaceutical gifts are to be despised. For the profound depression stimulants will be necessary, but these may take the shape of ether, or spirit of chloroform, as well as of wine or brandy. For the irritable stomach, alkalis, with nux vomica and ipecacuanha, or calumba, are of great service. So, too, in another way, are light tonics and neutral salts of iron; but the stomach should not be clogged with too much medicine. The bowels should not be much disturbed, but, if an aperient be required, a mild one, as a small dose of castor oil, or the compound liquorice powder of the Pharmacopœia, will suit. If the stomach rebel against these, a wine-glassful of Hunyadi Janos water the first thing in the morning, followed by a cup of warm milk, may better agree with the irritable organ. When there is diarrhoea, a totally different line of treatment will be necessary. But in all things, and at all times, the grand rule is to save the patient's strength, to add to it if possible, and to resist the inroads of the disease, whatever shape these may assume. ALEXANDER SILVER. W. CAYLEY.

**ADENALGIA** (ἀδῆν, a gland; and ἄλγος, pain).—Pain in a gland.

**ADENTITIS.**—Inflammation of a gland. See the several glands.

**ADENOCELE** (ἀδῆν, a gland; and κύλη, a tumour).—A tumour connected with a gland.

**ADENODYNIA** (ἀδῆν, a gland; and δόδυνη, pain).—Pain in a gland.

**ADENOID** (ἀδῆν, a gland; and εἶδος, form).—Glandular: resembling the structure of a gland, whether secreting or lymphatic.

**ADENOMA** (ἀδῆν, a gland, and the

termination *oma*, adopted to indicate a tumour).—A morbid growth, the structure of which is of glandular nature. See TUMOURS.

**ADHERENT. ADHESIONS.** }—Structures are said to be *adherent* when they become abnormally united together, the morbid formations by which this union is effected being termed *adhesions*. These are most frequently met with in connexion with serous surfaces, being usually the result of an inflammatory process, but they may be observed in other structures. The adhesions vary considerably in extent, number, mode of arrangement, firmness, and other characters: they may merely consist of a few loose, slender, and delicate bands; or these bands may be thick and strong; or the contiguous surfaces may be blended and matted together to a greater or less extent, so that they cannot be separated without tearing or cutting them asunder, this last condition constituting *agglutination*. In structure adhesions consist mainly of connective or fibrous tissue, more or less perfectly developed, with a few new vessels.

**EFFECTS.**—Adhesions are often found at post-mortem examinations, which have been of little or no consequence during life, as, for instance, many of those which form in connexion with the pleural surfaces. If, however, they are extensive and firm, or if they occupy certain regions of the body, they may prove of serious moment. The principal evils which are liable to result from adhesions may be thus indicated:—1. They often bind parts together, and interfere with the movements of important organs, such as the lungs, heart, stomach, or intestines; in this way preventing the due performance of their functions. 2. When an organ is displaced in any way, as, for example, the heart by pleuritic effusion, it may become fixed in its new position by the formation of adhesions, its functions being thus disturbed. 3. It is highly probable that agglutination may lead to hypertrophy of an organ, e.g. the heart, by embarrassing its movements, and hence affecting its action. 4. On the other hand, atrophy or degeneration of structure may ensue, in consequence of the adhesions interfering with the due supply of blood by pressing upon the vessels, so that the nutrition of the tissues becomes impaired. In the young, also, their development may be checked. 5. Adhesions may involve important structures, such as nerves or vessels, pressing upon or destroying them, thus giving rise to symptoms of a serious nature. 6. Tubes or canals for the passage of secretions or other materials are sometimes narrowed or obliterated by adhesions. 7. When formed within the abdominal cavity, especially when they take the form of bands, adhesions may prove highly dangerous by compressing, constricting, exerting traction upon, or strangulating some portion of the

intestine, in either of these ways leading to intestinal obstruction.

It is frequently difficult or impossible to determine the existence of adhesions by clinical investigation during life; but the history of some past illness during which they were likely to be formed, the results of physical examination, especially in connexion with the heart and lungs, and the symptoms present, not uncommonly enable them to be discovered. FREDERICK T. ROBERTS.

**ADIPOCERE** (*adeps*, fat; and *cera*, wax).—SYNON.: Fr. *Adipocire*; Ger. *Fettwachs*.

**DEFINITION.**—A substance formed by a spontaneous change in the dead tissues of animals.

**DESCRIPTION.**—As seen generally in a dried state in museums, adipocere somewhat resembles spermaceti in consistence, but it is less crystalline in fracture, and is of a dull white or buff colour, the surface being sometimes marked by the outlines of blood-vessels or of other textures. Adipocere in the earlier stages of its formation, or when formed in a damp situation, is soft, and if rubbed between the fingers communicates a greasy feeling. The odour is peculiar and rather disagreeable.

**CHEMICAL COMPOSITION.**—Adipocere dissolves in ether, and leaves a delicate filamentous web; it burns with a blue flame, and yields a white ash. It is properly described as a soap, composed of margaric and oleic acids in combination with ammonium, the fixed alkalis, and alkaline earths; the relative proportion of the latter ingredients varies with the age of the specimen (the ammonia disappearing), and with the composition of the fluids in contact with which the adipocere has been formed. It is said that oleic acid predominates in adipocere formed from dead fish.

**MICROSCOPICAL APPEARANCES.**—When the flesh of animals in which this transformation has recently commenced is examined with the microscope, it is found to be composed of broken-down or disintegrated tissues, fatty granules or particles, together with a few acicular scales or crystals. The granules may be seen in what had been muscular tissue to assume somewhat the arrangement of the muscular fibrillæ, thus presenting an appearance resembling an early stage of fatty degeneration. In old and dry specimens of adipocere the crystalline scales form the great portion of the mass, and they may be observed preserving the outlines of the muscular fibres.

**ORIGIN.**—Adipocere has long been known. It is formed readily from the flesh of animals exposed to moisture, or placed in running water, in very dilute nitric acid, or in alcohol and water in the proportion of 1 to 6. It is often met with in inconvenient abundance in the specimen jars of the anatomist. The

bodies of men and other animals buried in peat moss have frequently been found completely converted into adipocere. Lord Bacon mentions it in the *Sylva Sylvarum* (cent. vii. § 678), and so also does Sir Thomas Brown in the *Hydriotaphia*; but attention was especially called to its presence when a vast number of bodies were removed (in 1786-87) from the *Cimetière des Innocents* at Paris to the Catacombs. Fourcroy found many of these bodies converted into what he named *adipocire*, a name since retained.<sup>1</sup> Gibbes (as did others) suggested the possibility of applying adipocere formed from the waste flesh of animals to some useful purposes, but the tenacity of the disagreeable odour and the presence of other difficulties have prevented these suggestions from being carried out (*Philosoph. Trans.* 1794, p. 95). With respect to the immediate changes which give origin to adipocere, chemists have differed in opinion. One class believes, with Gay-Lussac and Berzelius, that the compound results from the fat originally present in the tissues, and that the other components are completely destroyed by putrefaction. Another class which includes the names of Thomas Thomson and Brande, maintains 'that the fatty matter is an actual product of the decay, and not merely an educt or residue.' These opinions may, the present writer thinks, be reconciled by the better knowledge we now possess of the elementary composition of tissues. We know that the combination of fat and albumin constituting one of the earliest steps in the process of nutrition is traceable in the further development and formation of nearly every texture. When that combination is destroyed by a cessation of the processes of life, the tissues are, as it were, resolved into their primary elements. We may thus have adipocere derived not only from free fat, but from the elements of fat existing in and obtained from the decomposition of other tissues. Adipocere may thus be described as both an educt and a product. This opinion is confirmed by the researches of Bauer and Voit, who showed that fatty matter was derived from the metamorphosis of albumin in starved animals, to which phosphorus had been administered.<sup>2</sup>

**PATHOLOGICAL RELATIONS.**—The medical jurist has studied this substance with the view of determining the time and progress of its formation, and of thus ascertaining the

<sup>1</sup> A curious illustration of this process is incidentally mentioned by Mr. L. Field. The Illinois limestone, he says, proves to be composed of coral, in each cell of which a particle of oil is found sealed up—'the result of the decomposition of the animalcula.'—*Journal of the Society of Arts*, 1883, p. 869.

<sup>2</sup> The writer would desire to refer here to the remarkable analogy which exists between the change of animal matter into adipocere, and that which occurs in vegetable matter by its conversion into peat and coal.

probable period at which death had occurred. But hitherto no decided or satisfactory information has been obtained, owing to the varied circumstances which influence the progress of the change, in connexion not only with the condition of the body itself, but also with the character of its surroundings. The formation of adipocere has a further and a special interest for the pathologist. It was the study of this process which led the present writer to discover the analogy which exists between it and fatty degeneration in the living body, and thus to establish the pathological doctrine that fatty degeneration is the result of a retrograde metamorphosis, not a form of perverted nutrition. (See *Medical and Chirurgical Transactions*, vol. xxxiii.)

RICHARD QUAIN.

**ADIPOSI.**—A term which properly signifies either general corpulency, or accumulation of adipose tissue in or upon an organ. See **FATTY GROWTH**; and **OBESITY**.

**ADIRONDACK MOUNTAINS**, in New York State.—A series of ranges rising from a plateau 2,000 feet above the sea, with picturesque lakes between them. Highest altitude, Marcy, 5,337 feet. Commodious hotels on the shores of the lakes. Much frequented as a summer climatic resort. Recommended in phthisis. See **CLIMATE**, Treatment of Disease by.

**ADYNAMIA** } (ἀ, priv.; and δύναμις,  
**ADYNAMIC** } power).—Terms indicating serious depression of the vital powers. The noun is employed as synonymous with the '*typhoid condition*.' The adjective is applied to diseases in which the phenomena of this condition are prominent. See **TYPHOID CONDITION**.

**ÆGOPHONY** (αἴξ, a goat; and φωνή, voice).—A peculiar alteration of the resonance of the voice, as heard on auscultation of the chest, compared to the bleating of a goat. See **PHYSICAL EXAMINATION**.

**ÆTIOLOGY** (αἴτια, cause; and λόγος, word).—That branch of pathological science which deals with the causation of disease. See **DISEASE**, Causes of.

**AFFINITY**.—This term is the designation of a property by which elementary and compound substances unite with one another and form new compounds. It is, therefore, a property with which chemists are principally concerned. But the ideas suggested to the chemist by the term affinity are also, though less explicitly, excited in the mind of the pathologist and of the therapeutist by certain classes of facts frequently falling under their observation. The pathologist, for instance, knows that saline or earthy matter is very prone to accumulate in the midst of degenerated tissue in the walls of an

artery or of a cardiac valve, so as to give rise to a patch of 'calcification'; he knows that in a gouty patient urate of sodium is most apt to accumulate and form 'chalk stones' in the tissues around affected joints; he knows that, however it may be administered, arsenic in poisonous doses tends to produce inflammation of the alimentary canal, that strychnine acts with preference upon the nervous system, and that in ordinary cases of lead-poisoning this metal interferes especially with the nutrition of the extensor muscles of the forearm. Applications of the same notion in the department of therapeutics are equally familiar in respect to the action of many drugs. It may be regarded as an ascertained fact that iodide of potassium tends especially to influence the nutrition of the fibrous structures in the body, and that bromide of potassium has a no less certain action in modifying the nutrition of the nervous centres in many unhealthy states. Again, there is a whole class of substances which when taken into the system have, whatever their other actions may be, an undoubted effect in modifying the functional activity of the kidney. We have in nitrite of amyl and in nitroglycerine remedies possessing a remarkable influence over the unstriated muscular fibres of the arteries and bronchi, or else over the nerve-centres by which they are controlled. We have in woorara an agent which acts especially upon the motor side of the nervous system; and we have in digitalis an important remedy which, amidst its other effects, seems to have a decided power of improving the activity of the cardiac ganglia. The recent progress of therapeutics encourages us to hope that more and more of these specific effects of drugs will be accurately determined, so that the notion implied by the term *affinity* may, after a time, have a deeper meaning than at present for the practitioner of medicine.<sup>1</sup> See **ANTAGONISM**.

H. CHARLTON BASTIAN.

**AFFUSION**.—A method of treatment which consists in pouring a fluid, usually water, either cold or warm, upon the patient. See **WATER**, Therapeutics of; and **BATHS**.

**AFRICA, SOUTH**.—The portion of this continent lying between 22° and 35° south latitude, and 18° and 32° east longitude, and including Cape Colony, Natal, Bechuanaland,

<sup>1</sup> Dr. James Blake states that we must look elsewhere than to chemistry for the nature of the reactions between living matter and the reagents with which we may bring them in contact: and that this problem will be solved when the spectroscopic characters of the elements are better known. He holds that not only are toxic actions not chemical actions, but also that the whole direct action (of metallic salts) on the most important functions of living matter is a physical molecular action determined by the number and character of the harmonic vibrations of which the reagents are the seat. (*Comptes Rendus de la Soc. de Biologie*, 1890.)

the Orange Free State, and the Transvaal, thus embracing the region at present available for health-resorts, and excluding the recently acquired British and German territories.

The physical features of this tract of country consist of: (1) a low-lying coast of varying extent, characterised generally by a warm and moist climate, and being in many parts swampy and malarious; and (2) an inland region, rising in terraces to 6,000 feet and upwards. This more or less elevated district, ascending in plateaux as the interior is approached, and varying in altitude from 1,200 to 6,000 feet, affords excellent sites for health-resorts. The climate of tracts at between 1,900 and 3,000 feet is warm and dry; but the most suitable one for invalids is to be found at the altitude of 4,000 to 5,000 feet, on the vast rolling grassy plain, or *veldt*, of which the Orange Free State, the Transvaal, and the Karoo District of the Cape are the best examples. Here the climate is characterised by great sunniness, dryness, and liability to extremes of temperature, though the night record seldom falls below the freezing-point.

**Cape Colony.**—Cape Colony consists of eastern and western provinces, presenting varieties of climate according to elevation, of which the inland portions are far preferable. As the rainy season occurs in the winter in the west, and in summer in the eastern province, the wet season may be avoided by travelling from one to the other. The general meteorological features of the colony are great dryness; moderate heat; small rainfall, precipitated in heavy showers on a small number of days; and a variable amount of wind.

None of the *coast towns* are suitable for invalids, but in the neighbourhood of Cape Town, *Rondebosch*, *Claremont*, and *Wynberg*, situated on the wooded slopes of Table Mountain, can be recommended for temporary sojourn.

*Graham's Town* (1,000 feet), in the *eastern province*, 100 miles from the sea, has an equable and genial climate, with a summer heat which is tempered by rain, the larger proportion of which falls at that season. Mean annual temperature, 60° Fah., the summer mean being 63° and the winter 53°; the mean range, 15°. Rainfall 26 inches, distributed over 84 days.

*Ceres* (1,700 feet), in the *western province*, 85 miles by rail from Cape Town, has a somewhat moister climate than *Graham's Town*, and possesses an excellent sanatorium with many social advantages.

The higher plateau of the colony contains the following suitable stations:—

*Aliwal North* (4,348 feet), 280 miles by rail from the port of East London, with a fine, clear, dry climate, which in the writer's experience has proved useful to many cases

of phthisis. The rainfall is 18 inches, distributed over 61 days, but occurring chiefly in summer, and sharp frost is occasionally recorded in the winter nights.

*Tarkastad* (4,280 feet), *Dordrecht* (5,200 feet), and *Burghersdorp* (4,650) have similar climates, the last place being connected by rail with Aliwal North, and within easy access of several stations higher and lower in elevation. The rainfall is 11·39 inches, and the number of rainy days 41.

*Cradock* (2,850 feet) and *Beaufort West* (2,792 feet) are both accessible by rail, and, though at lower altitudes, enjoy very fine climates. At the former the humidity is 62 p.c., the average summer maximum 91° Fah., the rainfall 9·18 inches in 45 rainy days, showing great dryness of climate.

**Orange Free State.**—*Bloemfontein* (4,540 feet) is to be recommended, and undoubtedly the Orange Free State offers many advantages in its vast *veldt* and dry climate, but it still lacks railway communication, though this is projected. A drawback, too, is the occurrence of dust-storms to which it is liable. At Bloemfontein the average maximum for the six hottest months of the year is 82° Fah., the average minimum 55°, the humidity 55 p.c., the rainfall 16·97 inches, and the number of rainy days 70. Patients are able to sleep in the open air, except during a small part of the winter, when night frosts occur, and in the rainy season; and by day the outdoor life can be thoroughly carried out, either on the trek or while residing at a farm.

**The Transvaal.**—The Transvaal is at much the same elevation as the Orange Free State, but is said to have a moister climate. *Pretoria* (4,007 feet), the capital, and *Heidelberg* have been recommended as suitable stations for invalids.

**Natal.**—The colony of Natal, situated to the N.E. of the Cape Colony, has a tropical climate, and is distinguished for its luxuriant vegetation and moister climate. As in the Cape Colony, the coast district, including Durban, is not suitable for invalids, but the slopes of the Drakensberg Mountains, which separate Natal from the Orange Free State, are cooler and more salubrious.

Here *Howick* (3,400 feet), *Estcourt* (3,562 feet), and *Ladysmith*—all connected with Durban by rail—offer the invalid many advantages, in a dry climate, warmer than that of the Orange Free State, and fairly protected from cold winds; and they have proved of great benefit in bronchitis and asthma.

The mean annual temperature of *Pietermaritzburg*, the capital of Natal (2,090 feet), 71 miles from the sea, is 64·7° Fah., the winter being 60° and the summer 69°, whilst the rainfall is 30 inches, occurring chiefly in October and March.

C. THEODORE WILLIAMS.

**AGEUSTIA** (ἀ, priv.; and γεῦσις, taste).—Loss of taste. See TASTE, Disorders of.

**AGONY** (ἀγών, strife or struggle).—Agony implies bodily pain or mental suffering so intense that it cannot be endured, but excites a struggle against it. It is also applied to the final struggle that often precedes death. See DEATH, Modes of.

**AGORAPHOBIA** (ἀγορά, a market-place; and φόβος, fear).—SYNON.: Fr. *La peur des espaces*.—By these names a peculiar nervous complaint has been recognised, characterised by a feeling of alarm and terror, associated with a group of nervous symptoms, which some individuals experience when they are in a certain space. The condition may be developed rapidly or gradually, and the chief phenomena observed are as follows:—A sudden sensation is experienced, as if the heart were being grasped, while this organ palpitates violently; the face becomes flushed; the legs feel weak, tremble, and seem as if they would give way under the body. There may be sensations of itching, coldness, or numbness; or profuse sweating may occur. There is no true vertigo; the special senses are unaffected; and consciousness is not at all impaired. A curious impression is sometimes experienced, as if space were elongating itself out indefinitely. Persons who are thus affected are quite sensible of the foolishness of their fear, but cannot be reasoned out of it. During the attacks they feel a strong inclination to cry out, but hesitate to do so. They think that their dread is known to others, and many of them endeavour to conceal their feelings, lest they should be considered insane.

The circumstances under which the symptoms just described may be experienced are various. They may be felt, for instance, in the street, especially if the shops are shut; in public buildings, such as churches, concert-rooms, or theatres; in omnibuses, cabs, or other conveyances; on a bridge; or in looking at an extended façade or flying perspective. Most persons who suffer thus in the street feel better when with some one, or when near some object, such as a carriage, or even when carrying an umbrella or a stick. Occasionally, however, they shun other people, especially acquaintances.

But little is known as to the origin and nature of agoraphobia. The complaint is not regarded as idiopathic, but as sequential to some other condition. It occurs in males and females, and the individuals affected may be strong and in good bodily health, while they are often intelligent and well-educated. A history of hereditary nervous disorder can be traced in some cases, indicated by the occurrence of insanity or epilepsy in members of the family; and the patients themselves may present indications of a nervous temperament. Their emotions are often easily

excited; and they may be subject to nervous symptoms, such as headache, a feeling of heat in the top of the head, sparks before the eyes, occasional faintness, or motor disorders.

**TREATMENT.**—Special attention should be directed to maintain the general health in its widest sense, and particularly to give strength to the nervous system. Active mental occupation, and pleasing social and moral surroundings, will generally succeed in relieving and ultimately curing the malady.

FREDERICK T. ROBERTS.

**AGRAPHIA** (ἀ, neg.; and γράφω, I write).—This term is applied to defects of intellectual expression by writing rather than by speech. These defects may occur alone, or in association with defects of speech, according to the extent and situation of the lesion or nutritional defect in different cases. See APHASIA.

**AGRIA** (ἀγριος, wild).—This term signifies angry and severe. Willan describes a *Lichen agrius*, which is likewise termed *agrius*. It is a circumscribed inflammatory eczema, situated on the back of the hands. The qualities implied by agria are excessive pruritus, burning pain, thickening, fission, and copious exudation. See LICHEN.

**AGUE.**—A popular synonym for intermittent fever. See INTERMITTENT FEVER.

**AGUE-CAKE.**—A form of enlargement of the spleen, resulting from the action of malaria on the system. See SPLEEN, Diseases of; and MALARIA.

**AIKIN**, in south-west division of South Carolina.—A mild, bracing, dry climate, said to resemble that of Mentone in warmth and dryness, but with larger monthly temperature range. Soil, sandy. Recommended for pulmonary affections. See CLIMATE, Treatment of Disease by.

**AINHUM** (Nat., to saw).—This disease was first described by Dr. da Silva Lima of Bahia in 1867. It is peculiar to the African race, being found not only amongst the inhabitants of the West Coast of Africa, but also amongst the Hindoos of African descent, as well as amongst the slave population of South America. At its commencement, a groove or furrow is seen at the base of the little toe (the part almost invariably attacked), situated on its inner and inferior aspect, and corresponding to the digito-plantar fold. The furrow soon extends to the entire circumference of the toe; and, as it becomes gradually deeper, the latter is left hanging by a slender pedicle, which can only be brought into view by separating the walls of the furrow. The distal portion swells into an ovoid mass, about twice its natural size; finally some accident snaps the pedicle, and the toe drops off, in from four to ten years from the commencement of the disease.

The furrow is caused by a constricting band

of hardened and contracted skin—a local scleroderma, which leads to faulty nutrition and degenerative changes in the parts beyond.

Ainhum is not a painful affection in itself, but the extreme mobility of the little toe causes trouble and inconvenience, for which patients often seek relief in amputation.

Occasionally the sides and bottom of the furrow ulcerate. Not infrequently both little toes are attacked by the disease. Males are more subject to it than females. The microscope reveals only atrophic and degenerative changes. The cause of ainhum is entirely obscure. It has been cured by the early division of the constricting band.

A. SANGSTER.

**AIR, Ætiology of.**—See DISEASE, Causes of.

**AIR, Therapeutics of.**—Air is employed in the treatment of disease in many ways and for many purposes. It is used, firstly, as *the atmosphere*, a gaseous mixture of definite composition and with a variable pressure. Secondly, advantage is taken of air as a *vehicle* for other substances in the gaseous or finely divided state. And, thirdly, it is selected as a *medium* by which *the temperature* of the body may be readily and effectively influenced. In the first of these relations only—as pure air—will its therapeutics require to be considered in this article. The application to the body generally of air that has been warmed, or warmed and loaded with moisture, will be found described in the article on BATHS; whilst its administration to the respiratory organs, either in this form or as a vehicle for such substances as creasote, carbolic acid, alkaloids, and sulphurous acid, will be discussed under INHALATIONS.

**PRINCIPLES.**—The dual relation in which the air stands to the economy—as a definite compound of certain gases, and as an atmosphere with a certain pressure—is very frequently disturbed; and this disturbance accounts for some of the most familiar phenomena of disease. Alteration in the quality or quantity of the respired air, whether from the state of the atmosphere itself, or from derangement of the complex apparatus of respiration and circulation, is the cause of some of the most serious and distressing symptoms attending diseases of the chest. It might be predicted by the physiologist that under these circumstances relief would be afforded, at least to symptoms, by suitable alteration of the composition or volume of the air. The method of treatment thus rationally indicated proves to be readily practicable: the supply of air is unlimited; its composition may be altered at pleasure; its pressure may be increased or diminished; and such alteration will alter its chemical properties. We find accordingly that, ever since the discovery of the composition of the atmosphere, frequent trials have been made

of its value therapeutically. Oxygen was early recognised as its active constituent, and came to be administered, as it still is, in the form of inhalation. From time immemorial, indeed, advantage has been taken of the purity and certain other unknown qualities of the air for the prevention and treatment of disease; and the character of the atmosphere is naturally reckoned one of the most important elements of climate (see CLIMATE). More lately, use has been made of the powerful properties that air possesses when *physically* changed. Within recent years remarkable advances have been made, on the one hand, in the physiology of respiration and the relation of the circulation to the atmospheric pressure, and, on the other hand, in the pathology of diseases of the chest. Clearer views have been reached on the signification of various symptoms, and especially of dyspnoea in its different forms. At the same time observations upon the effects of compressed and rarefied air have been becoming more exact. Pursuing the physiological track, modern therapeutists have availed themselves of this knowledge, and revived the use of air physically altered in the treatment of diseases of the lungs, heart, and other parts of the body. This application they are now able to make with accuracy, and the success of the reformed system of aërotherapeutics appears clear.

The physiological effects of compressed or of rarefied air will manifestly be different according as it is admitted to the body as a whole, or only to a part of it. Familiar examples of the former condition are afforded by descent in the diving-bell, or ascent in the balloon; and of the latter by the action of the cupping-glass, and the effects of interrupted or frequently repeated respirations upon the pulse and system generally. Under the first circumstances the alteration of pressure is absolute; under the second it is relative, and capable of producing most important disturbances in the distribution of the vital fluids. The two methods of application must accordingly be separately discussed.

**GENERAL AËROTHERAPEUTICS.**—The effects of *compressed air* on the body as a whole have been studied in the *air-bath*. This is a mechanical arrangement in the form of an iron chamber, which can be filled with air at any pressure, whether above or below the normal, by means of steam-power.

The principal *physiological effects* of air condensed by three-sevenths of an atmosphere were ascertained by von Vivenot to be:—Pallor of the skin and mucous membranes; a sensation of pressure in the ears; diminished frequency of respiration, the act becoming easier; enlargement of the lungs, and increase of the vital capacity; depression of the cardiac force, and diminution of the size and strength of the pulse; rise of tempera-

ture; increased vigour of muscular action, secretion, and nutrition generally; compression of the gaseous contents of the intestines; and, perhaps, increased absorption of oxygen and excretion of carbonic acid. When the pressure is excessive, dangerous or even fatal symptoms may supervene. Frequent exposure to condensed air will induce considerable increase of the vital capacity; and most of the other effects, both physical and chemical, will tend to persist. In a word, it may probably be said that the air-bath acts on the system, first, by increasing the general mechanical pressure; and, secondly, by admitting an increased amount of oxygen. In employing the air-bath, the patient is kept in it for a period of two hours, at first daily, but after some weeks less frequently. The pressure, which is employed in different cases at one-fifth to one-half of an atmosphere above the normal, must be slowly raised on admission, and reduced on removal of the patient.

*Uses.*—The number of diseases in which the air-bath may be given with success is limited: (1) In certain forms of dyspnoea. It gives great relief in spasmodic asthma, and may also afford temporary relief in emphysema; but its prolonged use appears to be positively injurious, as it increases the pulmonary distension. (2) In hyperæmia and catarrh of the air-passages, including pertussis. (3) In imperfect expansion or threatened retraction of the chest, as in the subjects of phthisis and chronic pleurisy. Compressed air has also been extolled in some forms of cardiac disease, and in general malnutrition.

The effects of *rarefied air* admitted to the body as a whole do not demand description in this place, either in their physiological or in their therapeutical aspect. Artificially rarefied air is never employed in the form of the bath; and the natural supply in elevated regions, which has found favour as a means of treatment in phthisis, is a subject that belongs to CLIMATE and PHTHISIS.

**LOCAL AËROTHERAPEUTICS.**—When it is desired to bring compressed or rarefied air into contact with the *respiratory* surface only, other apparatus must be employed. Different forms have been in use for some years, respecting which it will be sufficient to state that the air contained in a portable gas-holder is compressed or rarefied by simple mechanical means, and thereafter brought into relation with the air-passages by an arrangement of tubes and valves. There are four possible methods of application: (1) inspiration of condensed air; (2) expiration into condensed air; (3) inspiration of rarefied air; and (4) expiration into rarefied air.

Another apparatus, called the *pneumatic cabinet*, has been introduced more recently. It consists of an air-bath, built of steel and glass, to accommodate both the patient and

the administrator. The disturbance of pressure within the chest is effected by rarefying the air in the cabinet by means of a bellows, and then allowing the patient to inspire from the external atmosphere through a tube from without. This procedure is in effect mainly inspiration of a relatively condensed air.

The *physiological effects* of the several methods of application may now be briefly stated.

*Inspiration of condensed air.*—Inspiration of air that has been condensed by one-sixtieth to one-fortieth of an atmosphere produces a sensation of extreme distension of the chest, accompanied by an actual expansion of the thorax and lungs, and an increased admission of air, so that inspiratory dyspnoea, if present, is relieved. At the same time the other thoracic contents are compressed, the systemic vessels fill, the arterial pressure rises, and the jugulars become distended. The lungs and heart will be comparatively anæmic. If the application of condensed air be frequently repeated, the vital capacity, the size of the chest, and the respiratory force may all be increased, and partial relief may be permanently afforded to dyspnoea.

*Expiration into condensed air* is most difficult of accomplishment, and the effect on the circulation does not differ essentially from that just described.

*Inspiration of rarefied air.*—Inspiration of air that has been rarefied by one two-hundred-and-fortieth to one one-hundred-and-twentieth—or even, after a time, by one-sixtieth—of an atmosphere, immediately causes the phenomena of inspiratory dyspnoea; the thoracic viscera are congested, and hæmoptysis may result, for the effect may be regarded as that of dry-cupping the pulmonary alveoli. The heart at the same time becomes full, and the jugulars collapse.

*Expiration into air* that has been rarefied by one-sixtieth of an atmosphere is attended with a sense of extreme compression of the thorax; at the same time there is actually a partial retraction of the lungs, an increase in the volume of expired air, and a corresponding diminution in the amount of residual air in the chest. Expiratory dyspnoea, if present, is relieved. While the lungs thus diminish in size, the other thoracic viscera are dilated—the heart and the pulmonary and other vessels within the chest being filled at the expense of those external to it, both arteries and veins. If the expiration into rarefied air be frequently repeated, the circumference of the chest will be diminished, while the vital capacity will be actually increased, along with increase of the inspiratory and expiratory force.

*Uses.*—The method of *inspiring condensed air* is obviously indicated in diseases where inspiratory dyspnoea is an urgent symptom. Spasmodic asthma, stenosis of the air-passages from anatomical causes, acute and

chronic bronchitis, and atelectasis, have all been successfully treated by this method. In croup, where urgently indicated, it is most difficult or even impossible to employ it. In threatened phthisis it is used prophylactically, and in chronic phthisis it may usefully develop the healthy portions of lung; but it is contra-indicated in pyrexial cases, and may prove dangerous by inducing hæmoptysis. In chronic pleurisy it may prevent or remove the effects of collapse and retraction of the chest-wall. The inhalation of condensed air should also be of use in certain forms of cardiac dilatation, especially that due to mitral incompetence. Improving, as it does, the general nutrition, it may be combined with other remedies for anæmia. In the administration of condensed air, a 'sitting' should last from ten to thirty minutes, once a day—seldom twice.

*Expiration into condensed air* is not used therapeutically.

*Inspiration of rarefied air* may be regarded as a means of exercising the inspiratory muscles. Like the atmosphere of great altitudes, it may therefore be employed in persons with badly developed chests; and even in phthisis it may, by increasing the amount of blood in the lungs, prevent caseation and promote absorption of the products. In disease of the right side of the heart, it would assist the flow of blood from the veins into the lungs, but it is not likely to be employed for this purpose.

*Expiration into rarefied air* promises to be the most successful and most extensively employed of all the methods. In it, according to Waldenburg, we have the physical antidote for emphysema, and in his hands the majority of such patients are said to have been either cured or radically benefited. It has also afforded great relief in some cases of bronchitis, where it increases expectoration.

Other local applications of the physical properties of the air, as seen in aspiration, cupping, Junod's boot, and inflation, are described elsewhere in this work.

J. MITCHELL BRUCE.

#### AIR IN CELLULAR TISSUE.—

See EMPHYSEMA, SUBCUTANEOUS.

#### AIR IN VEINS.—See VEINS, Air in.

**AIR-PASSAGES, Diseases of.**—See RESPIRATORY ORGANS, Diseases of; also LARYNX, TRACHEA, and BRONCHI, Diseases of.

**AIX-LA-CHAPELLE, Waters of.**—Thermal sulphur waters. See MINERAL WATERS.

**AIX-LES-BAINS, Waters of.**—Thermal sulphur waters. See MINERAL WATERS.

**AKINESIA** (ἀ, priv.; and κίνησις, motion). A synonym for paralysis of motion, whether partial or general. See PARALYSIS, MOTOR.

**ALASSIO.**—In Italy on the Mediterranean coast, between San Remo and Genoa. A mild, bracing, winter resort, sheltered by hills except on the south and east. Comparatively free from wind and dust. See CLIMATE, Treatment of Disease by.

**ALBINISM** (*albus*, white).—**DEFINITION.**—A state of whiteness or absence of colour of the integument and certain other tissues, consequent on defect of pigment-formation. The want of colour may be *complete or incomplete; partial or universal; congenital or accidental*. Partial albinism may be limited to a spot of small dimensions; or there may be many such spots of variable extent, dispersed over the surface of the body, and giving rise to the appearance which is denominated *pie'd or piebald*; whereas in universal albinism the defect of pigment is not restricted to the integument, but is especially remarkable in the iris and choroid membrane of the eyeball.

**GENERAL CHARACTERS.**—Persons and animals affected with albinism are called *albinoes*. It would seem more correct to limit the term albino to those in whom the absence or defect of pigment is universal, and demonstrable not only in the integument but likewise in the eyeball. In the true albino, therefore, the skin is white and pink and more or less transparent, and this both in the fairer and in the darker races of mankind; but in certain of the latter, where the pigmentary function is simply defective and not totally wanting, the colour of the skin may be grey or tawny, and more or less variegated and freckled. The hair, sometimes of a pure silvery or opaque white, may be diversely tinged with yellow or red; occasionally it is flaxen or possesses a greyish hue; and in some instances the whole body is covered with a white down. The iris is grey or pink, in accordance with the density of its fibrous structure, and the consequent facility of penetration of the colour of its vascular layer; or, as generally happens in the negro, it is blue. The pupil is contracted and brightly red, from the absence of the screen of protection usually afforded to the choroid membrane by its pigmentary layer; and for the same reason the rays of light penetrating the sclerotic and iris give a brilliancy of appearance to the fundus of the eyeball. The absence of pigment in the first place the excess of luminous rays penetrating the coats of the eyeball interferes with the correctness of his vision; his retinae are intolerant of light; he stoops his head, or droops his eyelids, to shelter his eyes; he sees with more comfort in the dimness of evening than in the light of the sun; he is near-sighted; and there is in many cases an oscillation of the eyeballs.

**ÆTIOLOGY.**—Albinism is met with among

all races of mankind and in every country, but is most common amongst those who are subjected to insalubrious conditions of climate and hygiene. For these reasons it is not uncommon among the natives of the marshy coast of Africa; among negroes who are transferred to unhealthy districts in South America and the West Indies; among the inhabitants of the western coast of South America and Mexico; in certain of the islands of the Indian Ocean; and even in the northern regions of Europe. When albinism is congenital, it has been assumed to be due to an arrest of development; but when accidental, its existence must be referred to exhaustion of chromatogenous or pigment-producing function. Arrest of development has been inferred from the occasional persistence in albinos of the *membrana pupillaris*, and of the *foetal* down of the skin; from the more frequent occurrence of the condition in females than in males; and from the observation that albinos are sometimes misshapen and feeble intellectually as well as physically. On the other hand it is well known that albinism is often associated with perfect physical strength and remarkable intellectual vigour. Among other causes to which it has been assigned are heredity, and debility, however induced.

**TREATMENT.**—The treatment of congenital albinism must consist in the application of those agencies which tend to strengthen and improve the general health. With regard to the special inconvenience resulting from the absence of pigment in the eyes, it has been observed that the difficulty felt in reading is greatly lessened by using screens or goggles made of some opaque material, such as aluminium, each perforated by a small opening, admitting only the rays of light from the object looked at. The treatment of accidental albinism will be considered elsewhere. *See* PIGMENTARY SKIN-DISEASES.

ERASMUS WILSON.

**ALBUMINOID DISEASE.**—**SYNON.:** Waxy, Lardaceous, and Amyloid Degeneration; Fr. *Dégénération amyloïde*; Ger. *Speckartige Degeneration* (Rokitansky); *Amyloide Entartung* (Virchow).

**DEFINITION.**—A peculiar form of degeneration, affecting certain organs, and constituting in its effects a distinct and general disease.

**ETIOLOGY.**—In the majority of cases albuminoid disease is preceded by long-continued suppuration, most frequently in the form of bone- or joint-disease; or else of destructive pulmonary phthisis, empyema, pyelitis, cystitis, and other affections, where there has been a constant drain of pus. In the absence of obvious suppuration, there is usually present an exhausting disease, as syphilis, ague, or some more obscure cachexia. It has been observed as a consequence of inherited syphilis. These antecedent con-

ditions must be regarded as the cause of the malady, and it is only in the rarest instances that no such cause can be traced. It is not easy to recognise the connexion, but it may be pointed out that a drain of pus involves not only a loss of highly organised protoplasmic material, but also of potassium salts, which are contained in large proportion in the solid elements of pus, and which salts, as we shall see, are deficient in the affected tissues.

**ANATOMICAL CHARACTERS.**—The organs affected are usually much enlarged, but sometimes they ultimately decrease in size. They are pale, being evidently anæmic, dense, dry, sometimes hard, and either generally or in certain spots translucent. In an advanced stage of the disease the parts appear as if soaked in wax, or other translucent material. If iodine, in alcoholic or aqueous solution, be applied to the affected parts, they are stained yellow, orange, or a deep mahogany brown, according to the degree of the morbid change. If the portions thus coloured be further treated with dilute sulphuric acid, a purplish black colour is produced. In fine sections stained with the aniline dye, methyl violet, the affected elements are coloured pink, while the rest of the tissue is blue. The contrast of colour thus produced is the best means of recognising the degeneration under the microscope. These characters depend upon the presence in the tissue-elements of a peculiar substance, allied to the albuminates, and containing, when approximately pure, about 15 per cent. of nitrogen. It is soluble in alkalis, not digested by pepsin, and not readily altered by putrefaction; it yields with iodine the characteristic colour just noted, which gave rise to Virchow's erroneous supposition of its being allied to starch, whence the name—*amyloid*. Opinions have differed as to whether this change should be regarded as an infiltration or a degeneration. The *albuminoid* material, however, being contained in the tissue-elements themselves, and not infiltrated between them, is probably not poured out by the vessels as such, but results from a transformation of the materials of the tissues. Moreover, minute analysis has never detected any trace of this substance in the blood itself. Chemical analysis of the affected organs shows a remarkable change in their mineral constituents, the potassium and phosphoric acid being very greatly diminished, as compared with healthy organs; while the sodium and chlorine remain normal, or are proportionately increased.

Albuminoid disease affects most frequently the liver, spleen, and kidneys. Next in order of frequency come the lymphatic glands, and the intestinal mucous membrane, especially its villi; more rarely the suprarenal bodies, the pancreas, the urinary mucous membrane, or the omentum are involved; and, quite

exceptionally, other parts, such as the thyroid body, the generative organs, the heart, and the lungs. In most organs the small arteries and their appendages (glomeruli of the kidneys, Malpighian corpuscles of the spleen) are the seat of the morbid change; but in some, such as the kidney, secreting cells may also be affected. In the liver the hepatic cells have been thought to be involved, but the change is really in the swollen and degenerated capillaries, especially those of the middle zone of each lobule. The diseased elements are enlarged, translucent, and structureless.

**SYMPTOMS, DIAGNOSIS, AND PROGNOSIS.**—The general symptoms of albuminoid disease are anæmia, debility, a cachectic appearance, and sometimes capillary hæmorrhage. The local symptoms are chiefly important in the case of the liver, spleen, and kidneys. Uniform smooth enlargement of the liver and spleen, which can be referred to no other cause, may be due to the albuminoid change. Where the kidney is affected, albuminuria, dropsy, uræmia, and a train of symptoms arise, which, regarded as a whole, differ from those of other kidney diseases. Diarrhœa is a frequent symptom, probably referable to the degeneration affecting the intestinal mucous membrane. The *diagnosis* is greatly confirmed by (1) the simultaneous occurrence of disease in several organs; (2) a history of suppuration, or of some cachectic disease, especially syphilis. The *prognosis* is extremely unfavourable, and, when the disease is far advanced, it is hopeless.

**TREATMENT.**—Though in advanced cases treatment can avail but little, there is reason to think that were the occurrence of the disease anticipated, or its presence earlier recognised, prevention, or even cure, might be possible. The only real means of prevention is to check suppuration; and there is no doubt that since the adoption of antiseptic methods in surgery, and of more radical treatment of diseased bones and joints, albuminoid disease has become less common, at all events in London. In all such complaints as chronic joint-disease, psoas abscess, syphilitic disease of bone, or prolonged empyema, the probability of this frequently fatal *sequela* should be borne in mind, and guarded against by a suitable regimen. The diet should not only be generally nutritious, but should include more especially abundance of nitrogenous food (albuminates), as well as the potassium salts, which the affected tissues lack. These are, indeed, largely contained in the juices of fresh meat, and also in the green parts of vegetables. Among drugs, nutrient tonics, of which iron and cod-liver oil are the type, must hold the first place; but the administration of potassium salts, as proposed by Dr. Dickinson, is also indicated. Of these we should be induced, on *à priori* grounds, to select those of which the local

action is least violent, and which cause little vascular depression, such as the bicarbonate or the citrate, or other organic salts.

J. F. PAYNE.

**ALBUMINS.**—**DEFINITION.**—Albumins are substances closely resembling egg-albumin, the chief constituent of white of egg or albumen. To distinguish between the white of egg and its chief constituent, the former is spelt albumen, and the latter albumin. Albumins constitute a subdivision of the class of albuminous bodies, which includes all substances having a general resemblance to albumen. See ALBUMOSE; and GLOBULIN.

**ENUMERATION.**—The subclass properly contains only two members, *egg-albumin* and *serum-albumin*; but the name *Bence-Jones's albumin* has been given to an albuminous body differing very considerably in its properties from the other two.

**CHARACTERS.**—Egg-albumin and serum-albumin are semi-transparent, yellowish, and structureless when dried. They are soluble in water; and this solution is coagulated by boiling. From the same solution they are precipitated by: (a) nitric acid; (b) salts of the heavy metals, for example, copper-sulphate; (c) acetic acid with potassium-ferrocyanide; (d) boiling with acetic acid and a neutral salt, for example, potassium-sulphate; (e) alcohol. Egg-albumin is distinguished from serum-albumin by the coagulum which it forms with nitric acid being insoluble in excess, while that of serum-albumin is soluble. Bence-Jones's albumin gives no precipitate with excess of nitric acid unless left to stand, or unless heated and left to cool, when it forms a solid coagulum. This coagulum redissolves on heating, and again forms on cooling. It is therefore an albumose (see ALBUMOSE). It may be separated from ordinary albumin by adding nitric acid, boiling, and filtering when hot. The ordinary albumin will remain on the filter while Bence-Jones's albumin will pass through, and will coagulate when the filtrate cools.

**MODIFICATIONS.**—By the action of acids and alkalis albumin may be converted into *acid-albumin* and *alkali-albumin* respectively, neither of which is coagulated by boiling.

*Acid-albumin* may be formed in two ways: First, by dissolving solid albumin in concentrated nitric or other mineral acid with the aid of heat. Secondly, by heating an aqueous solution of albumin with one of these acids very much diluted (1 in 500). Although soluble in very concentrated or very dilute acids, acid-albumin is insoluble in moderately dilute acids. Therefore, when the solution in concentrated nitric acid is diluted with water, a precipitate is formed, which redissolves when much water is added. And, conversely, when acid-albumin is made by

boiling a solution of albumin in water with very dilute nitric acid, the addition of more acid will throw down a precipitate, which redissolves if a very large excess of the concentrated acid be added, and especially if it be heated at the same time. On neutralising a solution of acid-albumin, a precipitate is thrown down, which dissolves very readily in excess of alkali.

*Alkali-albumin*, or *alkali-albuminate* as it is also called, is formed by dissolving albumin in caustic potash or soda; or by adding either of these to its aqueous solution and allowing this to stand, or heating it. This modification is not precipitated by heat, but is precipitated by neutralisation; the precipitate dissolving very readily in slight excess of acid. If alkaline phosphates are present in the solution, as they are in urine, alkali-albumin requires a slight excess of acid to throw it down, and is not precipitated by exact neutralisation, as acid-albumin would be under similar circumstances.

T. LAUDER BRUNTON.

**ALBUMINURIA.** — DEFINITION. — A condition characterised by the presence of albumin in the urine. Other albuminous bodies, not albumins, may be present in hæmoglobinuria, hæmaturia, pyuria, and spermatorrhœa.

**SYMPTOMS.**—Albumin may occur in the urine without any symptoms whatever, but its continuous loss leads to anæmia and changes in the circulation, which usually originate a series of symptoms. These are: a pallid pasty complexion, dry skin, and tendency to œdema of the cellular tissue, noticeable on the eyelids and ankles; derangement of digestion, flatulence, occasional nausea, and irregularity of the bowels; nervous disorder, shown by muscular weakness, languor, lassitude, vague pains about the loins, and headache; calls to make water during the night; attacks of difficult breathing; palpitation, and frequently accentuation of the second sound of the heart over the aortic cartilage, and reduplication of the first sound over the septum ventriculorum.

**TESTS FOR ALBUMIN.**—The two tests usually employed to detect albumin in the urine are—first, boiling; and, secondly, the addition of nitric acid; both of which produce a cloud or precipitate. If the urine is turbid, the albuminous cloud may not be noticed; and therefore such urine should be filtered before the application of either test, unless the turbidity, being dependent on the presence of urates, is removed by heat cautiously applied.

**Method of employing the test by boiling.**—With the object of saving time the urine is often boiled at once, but the results thus obtained are liable to several fallacies, which will be subsequently described. In order to avoid such fallacies the following method should be pursued:—Ascertain the

reaction of the urine; and, if it be alkaline or very strongly acid, add acetic acid in the one case, or liquor potassæ in the other, until its reaction is only slightly acid. Fill a test-tube to about one-third of its capacity with the urine, and hold it obliquely in the flame of a spirit-lamp, in such a manner as to heat the upper part of the fluid only, until it boils. If it be turbid from urates, it should be first warmed throughout until it becomes clear, and then the upper part only should be boiled. Finally, add a drop or two of acetic or nitric acid.

If albumin be present, it will form a cloud or a coagulum, more or less dense according to its amount. When there is much albumin, its quantity may be roughly estimated by allowing the urine to stand for a definite number of hours, so that the coagulum may subside, and then observing whether it forms a fourth, a third, or a half of the whole length of urine in the test-tube. A small quantity causes a cloud, but no distinct coagulum; and, if merely a trace be present, a faint haze only will be observed, which is best seen by looking through the test-tube at a dark object. The advantage of heating the upper part only of the urine is, that the lower portion, which remains clear, affords a standard by comparison with which a faint cloud in the heated part may be more readily detected.

*Fallacies of the test by boiling.*—The first fallacy is that albumin may be present, and yet no cloud or coagulum be produced on boiling. This may occur if the urine be alkaline or very strongly acid, because alkali-albumin or acid-albumin, which are soluble in water, may be formed. It is to prevent the formation of *alkali-albumin* that acetic or nitric acid should be added to alkaline urine before boiling. This addition of acid also causes the coagulum to separate more readily; and it should therefore be made when the urine is neutral. On the other hand, urine rarely or never contains sufficient acid to form *acid-albumin*, unless the patient has been taking mineral acids; and therefore the addition of liquor potassæ is not necessary except under these circumstances. The second fallacy of the test by boiling is, that a cloud resembling that of albumin may be produced, although the urine is free from this substance. This occurs when the acidity of the urine is too slight to hold the earthy phosphates in solution, the heat probably affecting the relation between the basic and acid phosphates which are normally in solution, whereby deposition of insoluble phosphate results, forming a cloud like that of albumin. The two clouds are readily distinguished by the addition of a drop or two of acetic acid, when if due to phosphates the cloud will disappear by solution, but if caused by albumin it will remain. If an excessive quantity of nitric acid be added, an albuminous cloud

may also clear up; for albumin coagulated by heat is soluble in strong acid, though only to a slight extent.

**Application of the nitric acid test.**—

Pour some urine into a test-tube, and then allow about one-fourth of its bulk of strong colourless nitric acid to trickle slowly down the side of the tube, so as to form a layer below the urine without mixing. Or the acid may be put in the test-tube first, and the urine poured on it. Both processes give the same result. If albumin be present, a haze or cloud will form close to the line where the liquids meet.

*Fallacies of the nitric acid test.*—1. Albumin may be present and yet escape detection, if the nitric acid is simply poured into the urine and mixed with it, as is sometimes done. For if there be too much or too little acid, acid-albumin is formed and dissolved; whereas, if the liquids form two distinct layers, as in the process already described, the acid gradually mixes with and shades off into the urine, so that, at a greater or less distance from the line where they join, it is certain to be of the proper strength to precipitate the albumin. 2. Albumin may be supposed to be present when it is not, from the formation of a cloud by the precipitation of acid urates. This cloud disappears on the application of heat; and another specimen of the urine tested by boiling gives no cloud. To avoid this fallacy, it is common to employ the test by boiling, in addition to that by nitric acid. 3. The third fallacy is not of common occurrence. It is due to the presence of fat or saponified fats in the urine. Urine containing these when simply boiled gives no cloud; but if nitric acid is added to it in the cold, or acetic acid when it is hot, the fatty acids are precipitated and form a cloud resembling albumin. This is distinguished by not being formed if along with dilute acetic acid some ether is added to the urine before boiling; the ether retaining the fatty acids in solution. If the precipitate produced by nitric acid be collected on a filter, and treated with ether, it will be dissolved, while an albuminous precipitate will not. Copaiba, which can be recognised by its odour, sometimes causes an opalescence in the urine, which is increased by nitric acid, but is removed by heat.

**Additional tests for albumin.**—When urine contains *mucus*, which would render the presence of an albuminous cloud obscure, a solution of ferrocyanide of potassium followed by acetic acid should be added: this will produce a cloud if albumin be present, whilst it rather clears up a turbidity due to mucus. A solution of pyro-phosphate of sodium also precipitates albumin. If a few drops of albuminous urine be poured into a test-tube containing one or two drachms of a saturated solution of picric acid, a precipitate is formed which does not dissolve on

boiling, and thus differs from that due to albumoses. A strong solution of trichloroacetic acid also gives a precipitate with albumin. These tests are sometimes useful in determining the presence of albumin in the urine in doubtful cases.

**QUANTITATIVE ESTIMATION OF ALBUMIN.**—

There are three methods in common use for this purpose. The first is easy but inexact. It consists in boiling the urine with dilute acetic acid in a test-tube, allowing the coagulum to subside for a definite number of hours, and then estimating the proportion it bears to the quantity of urine boiled—for example, a fourth, a third, &c. The second is the most exact, but is troublesome. It is like the first; but the urine is carefully measured before boiling, and the amount of coagulum is ascertained by collecting it on a weighed filter, washing, drying, and again weighing it. The third method is easy and tolerably exact. A tube of known length is filled with urine and placed in a polarising apparatus. From the amount of rotation which the polarised ray undergoes in passing through the urine, the amount of albumin it contains may be calculated. A fourth method has been recommended by Sir William Roberts. It consists in diluting the urine with water until it gives a haze on the addition of nitric acid, which does not become visible until between one-half and three-quarters of a minute after the acid has been added. This dilute urine contains 0.0034 per cent., or 0.0148 grain of albumin per fluid ounce; and from the degree of dilution required the amount contained in the urine may be calculated. A fifth method is that of Esbach. It is less accurate than Roberts's, but is convenient. It consists in precipitating the albumin from urine in a graduated tube by means of picric acid, and reading off the quantity of albumin precipitated after the tube has stood for twenty-four hours. The precipitant consists of 1 part of pure picric acid and 2 parts of citric acid in 100 parts of water. The tube is filled up to a mark with the urine, and about two-thirds of its bulk of the precipitant is then added; the exact amount necessary being indicated by another mark on the tube. The lower part of the tube is graduated with lines numbered  $\frac{1}{2}$  up to 7, and these indicate the parts by weight of albumin in 1,000 parts of the urine by measure. Thus, if the upper level of the precipitate after twenty-four hours stood at 2, the proportion of albumin would be 2 in 1,000, or  $\frac{1}{500}$  of a grain of dry albumin to the ounce of urine. The results given are rather too low, and the method gives the best results when the proportion of albumin is small, so that urines containing much ought to be diluted before the test is applied. It is unsuitable if quinine, anti-pyrim, or thallin is likely to be present in the urine

**PATHOLOGY.**—Albuminuria has been said to occur in consequence of various conditions: e.g. changes in the blood, changes in the circulation, changes in the kidney. Thus abstinence from salt, or a diet of eggs alone, is said to produce albuminuria by altering the constitution of the blood; and an alteration in this fluid is supposed to be partly the cause of the albuminuria observed in high fevers, scarlatina, diphtheria, and osteomalacia. The albuminuria of heart-disease depends on changes in the circulation; and that of nephritis on alterations in the kidney. In order to distinguish more clearly between the different kinds of albuminuria we may divide them into—(1) *false albuminuria*, in which some other albuminous body than serum-albumin is present; (2) *true albuminuria*, in which serum-albumin, frequently accompanied by globulin, appears in the urine. In *true albuminuria* there is always some change either in the circulation through the kidney, or in the structure of the kidney itself. In *false albuminuria* the albuminous body passes out through the kidney, without there being any alteration either in its circulation or structure.

(1) *False albuminuria.*—The chief albuminous bodies occurring in false albuminuria are hæmoglobin, egg-albumin, globulin, and Bence-Jones's albumin (albumose). Hæmoglobin occurs in the urine whenever blood is present in it (see HÆMATURIA), in which case it is contained in the corpuscles; or it may occur free (see HÆMOGLOBINURIA), the blood-corpuscles, while still circulating in the vessels, having undergone solution. This may result from the inhalation of arseniuretted hydrogen, or from the introduction of bile-acids or of a large quantity of water into the veins. Hæmoglobin is also found in the urine in paroxysmal hæmoglobinuria, but the cause of the solution of blood-corpuscles in this disease is unknown. Egg-albumin is excreted by the kidneys, and appears in the urine, whenever it is injected directly into the circulation or under the skin, or when it is absorbed unchanged from the stomach or rectum. When taken into the stomach it is usually completely digested before it undergoes absorption; but when taken in such large quantities that the whole of it cannot be digested, part of it is absorbed unchanged and is excreted in the urine. Thus a diet consisting exclusively of eggs, especially when continued for several days, produces false albuminuria, and large enemata of eggs have a similar effect in animals, and probably also in man. Bence-Jones's albumin is of very rare occurrence. It is found in osteomalacia. Like egg-albumin, it is excreted by the kidneys when it is injected into the circulation, or in large quantities into the intestine. It is almost if not quite identical with the hetero-albumose which Kühne finds to be one of the products of imperfect

digestion. It seems probable that those cases of albuminuria which appear to depend on imperfect digestion, are due to the passage into the systemic circulation of albuminous bodies, which have not undergone the proper transformation in the alimentary canal or liver. See ALBUMOSES; and ALBUMOSURIA.

(2) *True albuminuria.*—In true albuminuria there must be some change, either in the circulation or structure of the kidney, for serum-albumin differs from the other albuminous bodies just mentioned in not being excreted by the healthy kidney. Some regard the alterations in the circulation which produce albuminuria as of two kinds:—(a) increased pressure of blood in the renal arteries; (b) increased pressure in the renal veins. Increased pressure in the arteries may depend either on general high arterial tension, or upon an increased local supply of blood to the kidney, owing to dilatation of the renal arteries, such as follows division of their vaso-motor nerves. Experiments seem to show, however, that increased tension in the renal arteries does not produce albuminuria, and that the only change in circulation which will cause it is increased pressure in the renal veins. Congestion of the renal veins may be produced by ligature of the renal arteries; and when the flow of blood through the kidney is temporarily arrested by ligature of the artery, the urine secreted after the removal of the ligature is albuminous. Venous congestion of the kidney also occurs whenever the onward flow of venous blood is obstructed, either by a ligature on the renal veins; by the pressure of a tumour or of the pregnant uterus upon them or the vena cava; by disease of the liver obstructing the vena cava; or by disease of the heart or lungs, such as tricuspid or mitral regurgitation, or chronic bronchitis and emphysema. The temporary albuminuria sometimes observed after cold bathing may also be due to venous congestion; and it is probable that albuminuria consequent upon lesions of the nervous system, is due rather to the changes which these produce in the circulation, than to any direct action of the nerves upon the tissues of the kidney itself. The albuminuria observed after varnishing the skin is probably due to the retention of some substance which acts as a poison. The structural changes in the kidney which cause albuminuria are acute and chronic inflammation, waxy degeneration, and cirrhosis. See BRIGHT'S DISEASE.

**TREATMENT.**—(1) In *false albuminuria*, where hæmoglobin appears in the urine, the treatment indicated is to counteract the solution of blood-corpuscles; and for this purpose quinine is very often useful. When other kinds of albumin appear in the urine, and are probably due to imperfect digestion, the treatment is to give some artificial digestive fluid. Arsenic is also useful. Regarding those cases of osteomalacia in which Bence-Jones's

albumin occurs, we unfortunately know very little.

(2) In *true* albuminuria, depending on venous congestion, the obstacle to free circulation should be removed, if possible; and the congestion lessened, both by drawing the blood from the interior to the surface of the body, and by causing contraction of the renal vessels. The blood may be drawn from the interior to the surface by means of warm baths; but in some cases these prove injurious rather than useful, and the employment of a wet pack, which has a similar effect on the distribution of blood without exciting the heart, is to be preferred. Cupping over the loins is serviceable: it probably acts by causing reflex contraction of the renal vessels rather than by actually draining blood away from them. The tone of the renal vessels may be increased by the employment of digitalis (*see* DIURETICS); and this drug is useful even when no cardiac disease is present, although its good effects are still more marked when the congestion is dependent on disease of the heart. The constant drain of albumin from the body occasions anæmia, which not only produces many unpleasant symptoms, but tends to cause fatty degeneration of various organs, from which there is no reason to believe that the kidneys are exempt. The administration of iron, therefore, is the chief medicinal remedy in structural disease of the kidneys. It is useful by diminishing or removing the symptoms of anæmia, and the tendency to fatty degeneration consequent thereon; and also by increasing the tone of the vessels, thus diminishing the loss of albumin.

T. LAUDER BRUNTON.

**ALBUMOSES.** — DEFINITION. — Albumoses are proteid bodies derived by the action of digestive ferments (pepsin chiefly) upon albumins and globulins, and in their properties may be classed as intermediate between these proteids and the final product of digestion (peptone). They are formed during natural digestion in the stomach, and also in artificial digestion.

ENUMERATION.—Many varieties of albumoses have been described, collectively termed *proteoses*. The individual bodies differ somewhat, according as they are formed from albumins (albumoses) or globulins (globuloses): for all practical purposes the term albumose is the best to use. The forms of albumose important in medicine are *hetero-albumose*, *proto-albumose*, and *deutero-albumose*, the last being closely allied to peptone.

CHARACTERS.—1. *Hetero-albumose* ('Bence-Jones's albumin') is insoluble in water, but soluble in dilute saline solutions, from which it is precipitated by heat if no free acid is present. In some cases it is precipitated at a temperature of 43° to 50° C., and re-

dissolves on heating the liquid to the boiling point. Artificially prepared hetero-albumose is rendered partly insoluble by heating; but the coagulum is soluble in dilute acids and alkalis, thus distinguishing it from the coagulum formed on heating a solution of serum-albumin or serum-globulin. Hetero-albumose is also precipitated from solution by saturation with magnesium sulphate or sodium chloride.

2. *Proto-albumose* and *deutero-albumose* are soluble in water, are not precipitated by heat, and are thus sharply distinguished from hetero-albumose.

These three albumoses have one reaction in common (besides those given by all proteids), viz., that with a trace of copper sulphate and an excess of liquor potassæ they give a pink-red colour (biuret reaction), the colour given by hetero-albumose being the least marked. They all behave in a peculiar way to nitric acid: if added *drop by drop* to a solution of hetero- or proto-albumose, nitric acid causes a precipitate, which is soluble in excess of the acid, but is also soluble on heating, re-appearing on cooling, and so on. With deutero-albumose, however, nitric acid causes no precipitate, unless common salt be added until the liquid is nearly saturated with it: the precipitate which is then produced by the acid behaves like the nitric acid precipitates of proto- and hetero-albumose. (For other distinctive reactions *see* ALBUMOSURIA.) Albumoses are distinguished from peptones by the nitric acid reaction; as well as by their precipitation with acetic acid and potassium ferro-cyanide, and with neutral ammonium sulphate added to saturation. The characters of true peptones are that they are not precipitated by heat, by nitric acid under any condition, by acetic acid and potassium ferro-cyanide, or by saturation with neutral ammonium sulphate. This last test is the most distinctive. In the presence of ammonium sulphate to saturation they give the biuret reaction, and are thrown down by tannin.

SIDNEY MARTIN.

**ALBUMOSURIA.** — SYNON.: Propeptonuria.

DEFINITION.—A condition in which albumoses are present in the urine. The condition where peptones are present (peptonuria) is not at present distinguishable from albumosuria. Peptonuria will therefore be considered under this heading.

TESTS FOR ALBUMOSE OR PEPTONE IN URINE.—Urine containing albumoses or peptones shows ordinarily no special characteristics indicating their presence; unless, in some cases, frothing when shaken. It may be dark- or light-coloured, of high or low specific gravity, and with or without deposit. In one case Kühne found a whitish deposit, consisting partly of albumose; and in a similar case Bence-Jones found casts, ordinary

albumin being absent. As a rule, however, albumose and peptone are present in solution in the urine. In searching for these, coagulable albumin must be absent, or if present removed by filtration after coagulation. The following methods are, therefore, to be used:—Heat the top of the column of urine in a test-tube in the usual way, but without the addition of acid. If a cloudiness appears, it is due to the precipitation of coagulable albumin, of phosphates, or of hetero-albumose (Bence-Jones's albumin). Continue the heating to the boiling-point; the cloudiness, if due to hetero-albumose, will clear up, that due to phosphates and albumin will remain. Add a drop of acetic acid; the phosphates will be dissolved up, the coagulated albumin will remain insoluble. A precipitate in urine, then, caused by a moderate heat and redissolving on boiling is hetero-albumose. This may be verified by determining the exact temperature at which the precipitation occurs: hetero-albumose is precipitated between 43° and 50° C. (109.4° and 122° Fah.), serum-albumin or globulin at 73°–75° C. (163.4° and 167° Fah.), and phosphates only when near the boiling-point; this last precipitate also redissolves on cooling. If the urine contains albumin or globulin, it must be boiled after the addition of a drop of acetic acid, and filtered. The filtered urine must then be tested for albumose in the way to be described. The presence of hetero-albumose is indicated by the last test just mentioned; in addition, it gives a characteristic nitric acid test. If to the *cold* urine nitric acid be added drop by drop, a precipitate forms which dissolves on heating, reappears on cooling, redissolves on heating, and so on: this is perfectly characteristic of the body, since the similar precipitate of albumin or globulin does not redissolve on heating. By neither of these reactions is deuterio-albumose or peptone indicated, and urines containing these two bodies (one or both of them) are those usually classed as examples of 'peptonuria.' Their presence is shown by the following tests, which must be applied after hetero-albumose has been shown to be absent, and after coagulable albumin has been removed:—(a) A drop of dilute solution of copper sulphate added to the urine, followed by an excess of liquor potassæ, causes a pinkish-red colouration (biuret reaction); or a drop of diluted Fehling's solution may be added to the urine, and then an excess of liquor potassæ if necessary. If the deuterio-albumose and peptone are present in small quantity, this reaction is not obtained. (b) The urine is added drop by drop to a saturated solution of picric acid; both deuterio-albumose and peptone are precipitated, and are redissolved on heating, thus distinguishing them from mucin and from albumin.

The only accurate way of distinguishing

deuterio-albumose from peptone is to shake the clear urine in a test-tube with solid neutral ammonium sulphate. If the urine gives the two tests just described, and gives a precipitate with ammonium sulphate, it contains deuterio-albumose. If it gives the tests and no precipitate with the salt, it contains peptone, and the case is one of true peptonuria. It is best, in applying these tests, to evaporate the urine to a small bulk.

Proto-albumose has not as yet been found in urine.

*Quantitative Estimation.*—This is of but little clinical value. It may be done by precipitating a measured quantity of urine with a large excess (ten times its bulk) of alcohol, collecting, drying, and weighing the precipitate.

**PATHOLOGY.**—The presence of albumoses and peptones in the urine is a pathological, not a physiological, phenomenon. These bodies are formed during normal digestion by pepsin and hydrochloric acid in the stomach, and peptones are also formed in pancreatic digestion. But although they are thus formed in the gastro-intestinal canal, they are not found in the absorbent vessels of the stomach and intestine in any appreciable quantity; neither in the veins nor in the chyle-vessels. In the general systemic veins and lymphatics they are not present; and their occurrence in arterial blood in small quantities is extremely doubtful. Lastly, they are not present in the living tissues, nor in the fluids which bathe them. If they are found in the urine, they are therefore either derived from the gastro-intestinal canal, or are formed from some pathological condition in the tissues, or perhaps in the blood. With regard to their first source, it is quite reasonable to suppose that the metamorphosis (probably into serum-albumin) which albumoses and peptones normally undergo when passing through the intestinal wall, may not take place in certain diseases where absorption is deficient, and that they may thus pass into the circulation, and be excreted by the kidneys in the urine. This, however, is a mere conjecture, to explain the occurrence of 'peptonuria' (albumosuria) in some cases of chronic dyspepsia with dilated stomach. The second source of the origin of albumoses and peptones in the body is a complicated one. If these bodies are formed in any organ, tissue, or fluid of the body, they are absorbed into the blood, and then find their way into the urine. It is known that if albumoses or peptones are injected into the blood of an animal they are excreted in the urine; that hetero- and proto-albumose pass out in the urine mainly as deuterio-albumose; that deuterio-albumose passes out chiefly as peptone; and that peptone is unchanged as it passes from the blood into the urine. The same facts probably hold good for the human organism.

Bence-Jones's albumin (hetero-albumose) was first found in the urine of a case of

osteomalacia; and Virchow found a similar body in the diseased bones. This, then, is a clear case of excretion of the albumose from the diseased part, the excretion of a proteid which is outside the normal proteid metabolism of the body. A similar albumose has also been found in a case, not of osteomalacia, but of glycosuria (Gowers). In the case of all abscesses, whether acute or chronic, in empyema and purulent peritonitis, albumoses (or peptones) are found in the urine. Here the explanation is simple, since the pus contains albumoses, and these are simply excreted by the urine after absorption into the blood. In the case of abscesses and of osteomalacia there are collections of cells which undergo disintegration; the proteids the cells contain (consisting almost solely of coagulable albumin and globulin) becoming partly transformed into albumoses. Whether this transformation takes place by the agency of bacteria present in the pus, or of a digestive ferment set free by the dying cell, is not at present known. This disintegration and gradual death of exuded cells, with the formation of albumoses, probably explains the occurrence of albumosuria and peptonuria in phthisis, in pneumonia, in epidemic cerebro-spinal meningitis, in cases of cancer and other malignant growths, in the puerperal state, in typhoid fever, and in organic liver-disease. In phosphorus-poisoning there is also great disintegration of many tissues and organs, and albumosuria (peptonuria) occurs; in scurvy and acute infectious diseases (especially measles) also it is sometimes present.

One form of peptonuria has been ascribed to an origin in the kidney-cells in cases of chronic nephritis, where some of the coagulable albumin which is being continually excreted is changed into albumose or peptone. Peptonuria may indeed alternate with albuminuria in these cases, and peptone may be present as well as albumin. Peptonuria with albuminuria is not uncommon in advanced chronic phthisis.

Albumose and peptone are also found in the urine in many other conditions, in which no explanation of their presence is evident; such as in many forms of inflammatory eruptions of the skin, in pemphigus, in urticaria, and in nervous diseases—cerebral hemiplegia, or psychoses.

**SYMPTOMS.**—From the account given of the pathology of albumosuria and peptonuria, it will be seen that they are only to be regarded as symptomatic of other graver pathological conditions; and no symptoms can be very directly ascribed to the presence of albumoses and peptones in the body, as associated with the diseases which have been already enumerated. But there are certain physiological effects of these proteids which are important to recollect. When injected into the circulation of a dog, albumoses (and peptones to a

less extent) markedly reduce the blood-pressure, and produce coma and death, while the blood remains uncoagulated for a long time after death. These effects are not necessarily associated with, or threatened by, the presence of albumosuria in man; possibly because the amount of albumose present in the blood at one time is never sufficient to produce them. And when such appearances as coma and uncoagulated blood after death are observed in the diseases already enumerated, they are ascribed either to the result of the grave lesion on the body generally, or the brain in particular, or to the retained chemical products of the waste of morbid tissues. It may be that albumoses and peptones form one group of these waste products, aiding the production of coma and of post-mortem fluidity of the blood.

Another physiological effect of albumoses is that of producing fever when injected into the circulatory system. The fever is to some extent proportional to the dose, and is produced even when the dose is insufficient to cause coma and fall of blood-pressure, and in animals (such as rabbits) whose circulation and cerebrum are not affected by the poison. Whether the fever of chronic phthisis is partly due to the absorption of these albumoses is not yet settled; this is simply a suggestion based on the facts mentioned.

According to our present knowledge, it may be said that the chief clinical significance of a large amount of peptone or albumose in the urine is an extensive cell- (and proteid) disintegration in the body; in many cases indicating pus-formation.

SIDNEY MARTIN.

**ALCOHOL.**—**SYNON.**: Ethyl-Alcohol; Vinic Alcohol; Spirit of Wine. ( $C_2H_6O$ ).

Alcohol is the product of a process of fermentation induced by the action of a microscopic fungus, *yeast*, upon certain kinds of sugar, especially grape sugar, but also upon that derived from the different varieties of starch and, in the same manner, upon milk sugar. In this process a peculiar metamorphosis takes place, by which alcohol and carbonic acid are produced in considerable amount, together with very minute quantities of succinic acid, glycerine, and other bodies.

Alcohol may also be produced synthetically from its elements—carbon, hydrogen, and oxygen.

As alcohol is very volatile, boiling at  $172^\circ$  Fah. ( $78^\circ$  C.), it may be separated by distillation from the water with which it is at first combined. Other means must be resorted to, however, in order to separate the ultimate parts of this water, as a strong attraction exists between the two liquids.

Alcohol, diluted with about 95 per cent. of water, and subjected to the action of another microscopic fungus, is oxidised into aldehyde and acetic acid.

**PHYSIOLOGICAL EFFECTS.**—Applied to the skin, alcohol produces a sensation of coolness, due to its rapid evaporation; but, if the application be continued sufficiently long, *irritation* is excited. The latter effect ensues immediately if alcohol is brought into contact with a mucous membrane. Its strong attraction for water seems to be the chief cause of this action.

Alcohol is a powerful *antiseptic*, probably from the fact that it is capable, even when diluted, of preventing the development of septic germs, such as vibrios and bacteria, as well as of paralysing the activity of those already formed.

There is scarcely any other therapeutical agent the *internal action* of which varies so much according to the dose given. In *small quantity*, and slightly diluted with water, alcohol promotes the functional activity of the stomach, the heart, and the brain; whilst a like quantity, largely diluted, exerts but a limited influence upon these organs: if, however, the dose of alcohol be often repeated, it is readily assimilated, and, becoming diffused throughout the system, undergoes combustion within the tissues of the body, imparts warmth to them, and yields vital force for the performance of their various functions. Simultaneously with this consumption of alcohol, the body of the consumer is often observed to gain in fat—a circumstance due to simple accumulation, the fat furnished by the food remaining unburned in the tissues, because the more combustible alcohol furnishes the warmth required, leaving no necessity for the adipose hydrocarbon to be used for that purpose. A quantity of 100 cubic centimètres of alcohol *per diem* (about three and a-half fluid ounces)—equivalent to about one litre of Rhine wine of medium strength—is sufficient to supply between one-third and one-quarter the whole amount of warmth requisite for the human body during the twenty-four hours. The warmth so supplied cannot be measured by a thermometer, however, any more than can that furnished by the internal combustion of other hydrocarbons, such as the oils and sugars. The subjective impression of increased warmth usually experienced after taking a dose of any alcoholic liquid is deceptive, and is only due to an irritation of the nerves of the stomach, and to the increased circulation of blood through the cutaneous vessels, particularly those of the head.

The increase of the action of the heart after taking alcohol is well known, and has often been expressed in figures. Recent experiments have also established this fact with regard to the respiration. The quantity of the inhaled and exhaled air in healthy men was increased by 7 to 9 per cent.; in one instance, after taking champagne, by 15 per cent.

*Doses somewhat larger, but still sufficiently*

moderate not to cause intoxication, act, for the most part, in the same way; but, as an additional effect, they produce a distinct decrease of temperature in the blood, lasting half-an-hour or more. As far as the matter has hitherto been explained, this latter effect depends upon a directly depressing influence exerted by alcohol upon the working cells of the body, and upon a temporary paralysis of the vaso-motor nerves. The latter is followed, of course, by dilatation of the superficial vessels, particularly those of the head, in consequence of which a larger surface of blood is exposed, and the loss of heat by radiation into the air is increased, the temperature of the circulating fluid being thus lowered; whilst the combustion carried on by the cells being retarded, the generation of heat from this source is diminished. The quantity of carbonic acid eliminated is thus diminished, as is also the amount of urea excreted. After the organism has become inured to the action of alcohol, these effects upon the temperature of the blood are less distinctly, or in some cases not at all, marked.

The agreeable excitement at first caused by such doses of alcohol is succeeded by a reaction, characterised by lassitude and drowsiness, the latter condition usually lasting longer than the previous one of exhilaration.

The symptoms of intoxication produced by *larger doses* of alcohol are sufficiently well known. When the abnormal condition of excitement in the brain induced by this stimulant has been kept up, almost without intermission, for a length of time; or when it is suddenly withdrawn after the organ has been long subjected to it; the disturbance brought about is so great and persistent as to result in a complete overthrow of the reasoning faculties, and the condition known as *delirium tremens* ensues. At the same time that this pernicious influence is being exerted upon the cells of the brain, fatty accumulations may take place in other organs, particularly in the liver, heart, and connective tissues; the blood-vessels become diseased; and, in many instances, cirrhosis of the liver, kidneys, and meninges makes its appearance, as part of the general disorder of nutrition. The shrinking of connective tissue, characteristic of this last-mentioned complication, seems to depend upon the direct irritation caused by the presence of un-oxidised alcohol.

Under ordinary circumstances, and after the consumption of moderate quantities of alcohol, only slight traces of it are to be detected in the urine, and still less in the breath and perspiration. A long series of experiments, made by the writer's assistant, Dr. Bodlaender, on healthy men and animals, proved that altogether at the most 3 per cent. reappears. The feces do not contain any alcohol at all. As regards the breath, pure alcohol imparts no taint to the exhalations of the body; the ethers and fusel oil, on the

other hand, do so by reason of their being less readily combustible. It is very likely that alcohol is completely oxidised into carbonic acid and water during the process of assimilation; at least, no other secondary products resulting from its disintegration have as yet been detected.

Much importance is sometimes still attached to the experiments of Lallemand, Perrin, and Duroy in discussions on alcohol. They asserted just the contrary to the above. Anstie and Dupré in England had already proved their conclusions to be erroneous; the new experiments which Bodlaender carried out with great minuteness have removed every doubt on the subject.

**THERAPEUTICAL APPLICATIONS.**—There can be no doubt that a healthy organism, supplied with sufficient food, is capable of performing all its regular functions without requiring any specially combustible material for the generation of heat and the development of vital force. But the case assumes a different aspect when, in sickness, while the metamorphosis of tissue goes on with its usual activity, or with increased energy, as happens in many diseases, the stomach, refusing to accept or to digest ordinary food, fails to supply material to compensate for this waste. Here it is, then, that a material which can be most readily assimilated by the system, and which, by its superior combustibility, spares the sacrifice of animal tissue, is especially called for; and such a material we have in alcohol. *Small but oft-repeated doses* of alcohol, largely diluted with water, are generally well tolerated by the weakest stomach; and, thus given, the absorption and oxidation of the spirit goes on without difficulty or effort on the part of the patient's system.

According to the experiments of Dr. Frankland and others, the burning of 1.0 gramme of alcohol yields sufficient heat to raise the temperature of seven litres of water 1°C.; and the burning of 1.0 gramme of cod-liver oil suffices for nine litres. Now, in taking three tablespoonfuls of the oil daily, we yield about the same amount of warmth to the body as is given by four tablespoonfuls of absolute alcohol—the quantity contained in a bottle of light claret or hock. The oil, however, is digested and oxidised by the organs of the body with difficulty, while, for the assimilation of the alcohol, scarcely any exertion of the working cells is required. Thus, it can be demonstrated by calculation, as above mentioned, that heat-producing material, sufficient to supply nearly one-third the whole amount of warmth required by the body within twenty-four hours, is offered in a quantity of 100 grammes (about three-and-a-half fluid ounces) of alcohol. In this sense alcohol is a *food*; for we must regard as food not only the building material, but all substances which, by their combustion

in its tissues, afford warmth to the animal organism, and, by so doing, contribute towards the production of vital force, and keep up the powers of endurance. Alcohol, therefore, diluted with at least 90 per cent. of water (in any convenient form of beverage), may be given with advantage, in small but oft-repeated doses, in most of the acute and chronic diseases where it is desired to sustain the strength of the patient, but where at the same time the digestive organs, from any cause, refuse to tolerate a more substantial form of nourishment, at least in quantities that would answer the necessities of the case. In such cases it is certainly not sufficient to call alcohol merely a *stimulant*. If alcohol served here only in the quality of a stimulant, its effect would soon pass away, leaving the patient more exhausted than ever; for the human organism is so constituted that it cannot be driven to perform its functions by the application of measures that simply stimulate, without supplying some new force to take the place of that put forth by the organs of the body under the impulse of excitement. To take a familiar illustration, alcohol thus given stimulates no more than does the readily combustible coal which we put in small quantities upon a languid fire, to prevent its going entirely out.

Recent experiments in the writer's laboratory (Geppert) have contributed the following facts to this part of the question. Twenty to 75 cubic centimètres of absolute alcohol, taken in water or in the form of port, brandy, or sparkling hock by healthy adults, who were accustomed to take no alcohol or only a moderate quantity, hardly altered the quantity of the consumed oxygen, and likewise left unaltered or very slightly decreased the excretion of the carbonic acid.

The principal point in this result is the unchanged standard of the consumption of oxygen. We know that the consumed alcohol is burnt in the organism, a minute quantity excepted. The alcohol was thus unable to increase oxidation, as its adversaries have still continued to assert; it had not contributed to a more rapid wasting of the organism; nor had it made the regular equal march of this wasting irregular and slow. On the contrary, it had substituted itself simply as fuel in order to sustain the normal temperature of life, the existence of which is necessary to keep the whole machine in motion. Alcohol acts here, generally speaking, as if we had given the person experimented upon oil or sugar. A part of the disposable oxygen which would serve for oxidising other substances, serves for the combustion of the alcohol, and keeps them intact for the organism.

*Medium doses* of alcohol act powerfully upon the brain and heart, and are therefore serviceable as real stimulants in cases where it is desirable to excite the cerebral and circulatory systems to greater activity. We must

not forget, however, that while exciting this increased activity, such doses do not elevate the temperature of the body; on the contrary, where the effect can be measured, it is found that they depress it a little. By continuing to exhibit such doses, we can sometimes (in erysipelas, puerperal peritonitis, and similar diseases) lower febrile heat by alcohol where even quinine proves ineffectual. The consequences of this decline of fever-heat are an immediate restoration to consciousness, if delirium or stupor has been present; and, in any case, a general improvement in the feelings of the patient. Todd and his school, before the application of the thermometer, called this *the effect of stimulus*, while in reality the improvement is, to a great extent, due to the diminution of febrile disturbance. As fever patients can tolerate large quantities of alcohol without showing any sign of intoxication, it is allowable, and sometimes even necessary, to rise in the scale of doses beyond the limits ordinarily prescribed.

Of late years alcohol has been given during the night to hectic phthisical patients as a preventive against copious and exhausting attacks of sweating, and with a gratifying amount of success. Such patients certainly tolerate the remedy much better than has hitherto been generally supposed. It need hardly be said that, in cases of cardiac excitement, not resulting from fever, alcohol is at least to be used with caution.

MODE OF ADMINISTRATION.—One of the most important, but at the same time most difficult, points for decision is the exact nature and quality of the alcoholic drink to be prescribed or allowed to a patient who may require alcohol in some form. For *general use*, a pure claret, hock, or Moselle wine are the forms of alcohol most to be recommended. Cognac, champagne, old gin or whisky, and the heavier Southern wines, may also be used according to circumstances. But whatever drink may be selected, it must at least be free from fusel oil to such an extent that a healthy man, even after imbibing a considerable quantity, will not feel any other effects than those of a pure stimulus; that is to say, an agreeable exhilaration of spirits, neither accompanied by a sense of weight in the head, nor followed by that persistent overfilling of the cerebral vessels and dulness of ideas characteristic of the physiological effects of fusel oil.

The *fusel oils* (so called from their oily qualities) consist chiefly of propyl, butyl, and amyl alcohols, of which the last-named forms the largest proportion. In order to examine any specimen of alcohol with reference to its purity from these objectionable constituents, it is only necessary to rub a few drops between the palms of the hands for half a minute, by which rapid evaporation is caused, and then to smell the moist spot left on either palm. If the alcohol be pure, no odour whatever

should remain, as ethyl alcohol evaporates very quickly; amyl alcohol, on the contrary, is much less volatile, and, if present in the liquid, will not have evaporated, so that its peculiar and unmistakable odour will remain to attest its presence as an impurity in the specimen examined.

This test is not applicable to the more complicated liqueurs and wines, as these all contain certain odoriferous organic principles of their own, which might disguise the smell of the fusel oil. The inoffensive quality of any given preparation, as a wine or spirit, can only be relied upon when one knows by experience that it *is* pure; and then it should always be obtained, if possible, from the same source, so as to ensure uniform purity.

To facilitate the process of estimating the quantity of any particular beverage necessary to be administered in order to produce a given effect, a table is subjoined showing the percentage of absolute alcohol contained in average specimens of the different kinds of wine, beer, &c., in common use.

#### *Absolute Alcohol contained in—*

- Kumiss (a fermented liquor made from milk or whey). from 1 to 3 vol. per cent. Kefir is the same, but derived from the milk or whey of the cow, whilst kumiss comes from the milk or whey of the mare. Both contain much carbonic acid, which renders them easily digestible.
- German beer<sup>1</sup>: from 3 to 5 vol. per cent.
- Hock or claret: from 8 to 11 vol. per cent.
- Champagne: from 10 to 13 vol. per cent.
- Southern wines (port, sherry, madeira, &c.): from 14 to 17 vol. per cent.
- Brandy and the stronger liqueurs: from 30 to 50 vol. per cent.

For *antipyretic purposes* one will need to give an adult daily not less than the equivalent of fifty cubic centimètres (about two fluid ounces) of absolute alcohol, in divided doses within an hour or two. Taking this as a starting-point, the dose suitable for each individual case can be estimated accordingly.

The great quantity of carbonic acid contained in certain 'sparkling' wines acts upon the temperature of a fever patient much in the same favourable manner as the alcohol itself; and when alcohol is to be taken as a food, it would seem that the impregnation with carbonic acid facilitates its absorption.

All that has been stated thus far with regard to the use of alcohol in sickness applies to children as well as to adults. Of course no reasonable person would accustom healthy children to the use of alcoholic beverages; but, in cases of disease, really good and pure wine or brandy can be advantageously employed, even for infants, either as a *stimulant*, an *antipyretic*, or an *article of food*, according to circumstances.

For *external use*, alcohol has been super-

<sup>1</sup> English beer will contain a little more, but the writer has made no personal examination as to exactly how much.

seded by various more modern agents, of which carbohc and salicylic acids may be mentioned as the most important.

As regards the copious experimental literature on alcohol and its medicinal properties, the writer would refer to his *Vorlesungen über Pharmakologie*, Berlin, 1886, pp. 354-392, and to Professor v. Jaksch's and his own papers in the Transactions of the Congress for Internal Medicine, Wiesbaden, 1888, pp. 70-133. The two papers have also been reprinted in the same volume. C. BINZ (Bonn).

**ALCOHOLIC INSANITY.** — See ALCOHOLISM; and INSANITY.

**ALCOHOLISM.** — DEFINITION. — This term is applied to the diverse pathological processes and attendant symptoms caused by the excessive ingestion of alcoholic beverages. These are very different according as a large quantity is consumed at once or at short intervals, or smaller quantities are taken habitually; hence they are subdivided into those due to (a) *acute*, and (b) *chronic* alcoholism. To the *acute* forms of alcoholic poisoning belong acute catarrh of the alimentary mucous membrane, rapid coma, some cases of delirium tremens, and certain special forms of acute insanity; whilst to the *chronic* class are referred the prolonged congestions, the fatty and connective-tissue degenerations of the various organs and tissues, most cases of delirium tremens, nervous affections of slow onset and course, and the cachexiæ, which, in varying combinations, attend a continuously immoderate consumption of alcohol.

**ÆTIOLOGY.**—That ordinary vinic or ethyl alcohol, in any and every shape, is a sufficient exciting cause of such *chronic* affections is beyond a doubt; moreover, we find that the more concentrated the form in which it is taken, the more surely and rapidly are they induced, and that, although some beverages give a greater liability to certain forms of disease than to others, yet the ultimate tissue-changes produced by all are practically similar, and of a markedly degenerative character. The purest alcoholic fluids will also induce the *acute* forms; but some of the phenomena observed in the worst cases of alcoholic poisoning have been referred, with some probability, to admixture with fusel oil, essential oil of wormwood, cocculus indicus, and other substances, more deleterious even than ordinary alcohol itself. See ALCOHOL; and ABSINTHISM.

The *predisposing causes* of a sudden debauch, such as festive gatherings, example of companions, desire of relief from anxiety and melancholy, &c., scarcely require mention. Acute alcoholic coma is generally due to the rapid consumption of a large quantity, but occasionally it is caused by taking a smaller quantity in the presence of some special

condition, such as starvation, prolonged exposure to cold, or debilitating disease.

Chronic habitual drinking is undoubtedly *hereditary* in many cases; not that the ancestors have necessarily been drunkards, but that the family is of unstable nervous organisation, and that the neurotic taint which shows itself in some members in such affections as epilepsy, hysteria, or insanity, is manifested in others by an intense craving for alcohol. Sometimes a pernicious education, by fostering habits of indulgence in early youth, has led to subsequent excess; and the prescribing of stimulants has occasionally been productive of similar harm. In the experience of the writer the exhibition of large doses in fevers and acute affections has never done this—indeed, in several instances, a great dislike to stimulants has been produced; but the custom of recommending small quantities to young people and women, as a remedy in hysteria, hypochondriasis, neuralgia, and allied disorders, or to relieve debility *post partum*, or the fatigues incident to their daily life, cannot be too strongly protested against. The effect of *occupation* is very marked. Brewers, publicans, potmen, and others who trade in alcohol are, as a class, very intemperate; and so frequently are commercial travellers, owing to peculiarities in their mode of transacting business. Sedentary employments, being more monotonous, are more baneful than outdoor occupations. Mechanics drink more freely than agricultural labourers; whilst night-labourers, cabmen, sailors when on shore, brewers' draymen, navvies, pitmen, and puddlers, consume an enormous amount of alcoholic fluids. *Social influences*, such as domestic unhappiness, rate of wages, unhealthy dwellings, bad drinking water, or an intermittent supply, are important factors in the causation of drunkenness. Under some circumstances, alcoholic excesses do less injury than usual, for example, in persons whose employment leads to copious sweating, or necessitates abundant exercise in a keen air; and some constitutions resist the baneful influence of alcohol to a remarkable extent.

**PATHOLOGY.**—A large amount of ardent spirits acts on the nerve-centres as a narcotic poison, and causes rapid death by coma. Smaller quantities produce intoxication, accompanied with or followed by an acute congestion and catarrh of the alimentary canal, especially of the stomach and duodenum. Habitual dram-drinking, by altering the chemical composition of the blood, and checking the normal changes of its corpuscles, exerts an injurious influence on the nutrition of the tissues. This is increased by the lessened consumption of food, and by the alterations in the calibre of the blood-vessels, set up at first by a special action on their vaso-motor nerves, and afterwards maintained by degeneration of their coats, and fre-

quently of the heart itself. Moreover, alcohol probably interferes *directly* with the nutrition of the cell-elements of the various organs as it circulates through them; and it retards the elimination of effete materials—carbonic acid, uric acid, and urea.

**ANATOMICAL CHARACTERS.**—(a) *Acute Alcoholism.*—Dr. Beaumont thus describes the appearances which he observed in the stomach of Alexis St. Martin, after an excess of alcoholic stimulants:—‘Inner membrane morbid; considerable erythema, and some aphthous patches on the exposed surface; secretions vitiated.’ On another occasion, ‘Small drops of grumous blood exuded from the surface, the mucous covering was thicker than common, and the gastric juices were mixed with a large proportion of thick ropy mucus and muco-purulent matter slightly tinged with blood.’ The post-mortem appearances in a case of rapid coma in a patient at King’s College Hospital, after taking three pints of raw whisky, were:—Intense injection of the vessels of the pyloric end of the stomach and duodenum, with a peculiar blanching of the mucous membrane between them, giving rise to a vivid scarlet arborescent appearance on a white ground; two ounces of bloody serum in the pericardial sac, and about sixteen ounces in the right pleural cavity (the left being obliterated by old adhesions); double pneumonia of the lower lobes; extreme congestion of the kidneys; and engorgement of the large veins over the posterior part of the brain. Contrary to the usual statements, no alcoholic odour could be detected in the brain, and there was no increase of fluid in its ventricles. The heart, liver, and kidneys were fatty; but these changes were probably of older date. In similar cases Deverjé has noticed a bright red colouring of the pulmonary tissue; whilst Tardieu found pulmonary apoplexies in two cases, and meningeal hæmorrhages in five others. Death from *acute delirium tremens* leaves no marked characters: meningitis and coarse brain-lesions are extremely rare, whilst pneumonia is much more common. After repeated attacks, as well as in old drunkards, fatty degeneration of the viscera and various other chronic changes are found.

(b) *Chronic Alcoholism.*—The amount of fat in the blood is increased, or it becomes more visible. Chronic congestion and catarrh of the stomach, leading to atrophy of the gland-cells and an increase in the submucous connective-tissue, is very constant, but chronic ulcer is not frequent. The liver is at first enlarged from congestion, and may continue so from a subsequent infiltration with fat; but more frequently it shrinks, owing to cirrhosis. Lobar emphysema, chronic bronchitis, and hypostatic pneumonia are common. The heart is flabby, dilated, and presents fatty infiltration or even degeneration of its muscular tissue; but it may be

hypertrophied, probably as a result of co-existent disease of the kidneys. The arteries and endocardium are studded with atheromatous deposits; the capillaries are congested, and the veins varicose. The kidneys exhibit the fatty, or, more commonly, the granular form of Bright’s disease. The muscles are pale and flabby, and even in the bones formation of fat takes place at the expense of the bony texture. The nervous centres are atrophied and tough; the convolutions are shrunken; the nerve-cells and nerve-fibres are wasted; and an increased amount of serous fluid exists in the ventricles and subarachnoid space. The abnormal adhesion of the dura mater to the cranium, the large Pacchionian bodies, the opaque arachnoid, and the thickened pia mater, all testify to an exaggerated development of fibrous tissue. Occasionally hæmorrhage into, or softening of, the brain, consequent on the diseased state of its blood-vessels, is met with. The increase of connective tissue is especially marked in *spirit-drinkers*, and explains the emaciated appearance, prematurely aged look, sunken cheeks, and wrinkled countenance which they generally present. The *beer- and wine-drinkers*, on the contrary, are loaded with fat, not only in the viscera, but in the subcutaneous tissue and the omentum; and hence these subjects are corpulent, with oily skins and prominent abdomens, even when the face and extremities are wasted. Gouty deposits are also frequent. These differences, however, are not nearly so absolute as is maintained by many writers. Alcoholic paralysis is due to a multiple peripheral neuritis, leading to atrophy of the muscles supplied by the affected nerves. The presence of a variable amount of dropsy, a congested pharynx, chronically inflamed conjunctivæ, turgid capillaries, and occasionally papules of acne rosacea on the face, complete the morbid anatomy of the confirmed toper. The necropsy in *alcoholic insanity* discloses no specific characters.

**SYMPTOMS.**—1. *Acute Intoxication.*—In this state the successive and varying mental phenomena, the disorders of common and special sense, and of the motor apparatus, are well known. These are followed by uneasy sensations and tenderness in the epigastrium, vomiting or retching, headache and vertigo, with dimness and occasionally yellowness of vision on stooping and rising again. The tongue is furred, the appetite is lost, and there is a constant feeling of thirst. The urine is copious and pale, but afterwards becomes scanty and loaded with lithates. The countenance is sallow, and the general lassitude and depression are very marked.

2. *Acute Alcoholic Coma.*—In slight cases of this condition, prolonged drowsiness is the chief symptom: but in the more severe forms the patient is quite insensible; the power of motion is in complete abeyance;

the breathing is stertorous; the face is usually pale, the features remaining symmetrical; the pupils are generally dilated, though they may be contracted or unequal; the pulse is slow and laboured; the skin feels cold and clammy; and the temperature is low—in one case it fell to 92° Fah. There may be albuminuria; and occasionally the urine and fæces are passed involuntarily.

3. *Chronic Alcoholism*.—The earliest symptoms of this form are muscular tremors, especially on waking; disturbed sleep; noises in the ears; dull headache; occasional vertigo; and disorders of vision. If there be also a foul breath, slightly-jaundiced conjunctivæ, watery eyes, and flabby features, with or without papules of acne rosacea around the nose and mouth, the combination is very characteristic. Irritative dyspeptic symptoms—the *vomitus matutinus* of Hufeland, and the signs of commencing or actual cirrhosis, of Bright's disease, or of fatty heart, frequently co-exist.

*Alcoholic paralysis* in its milder forms is marked by distressing pains in the extremities, especially in the legs, increased on deep pressure, redness and œdema of the feet and hands, restlessness, insomnia, and a gradually increasing loss of power, beginning in the lower limbs and afterwards extending to the forearm and arms. The gait is peculiar and uncertain, with the legs wide apart; and support by sticks or by an attendant is often required whilst walking. The hands are irregular and jerky in their movements; and the reflexes, especially the patellar-tendon reflex, are diminished or lost. The peronei and extensors of the leg, the extensor muscles of the thigh, and subsequently the extensors of the forearm and the triceps, are markedly atrophied, and exhibit the 'reactions of degeneration.' See PARALYSIS, TOXIC.

In its most severe forms anæsthesia succeeds to hyperæsthesia, and complete paralysis of the above muscles, leading to a condition of talipes equino-varus of the feet, and wrist-drop of the hands, may come on; and gangrene has occasionally been noticed. Loss of memory and other psychical disturbances are common. Death is generally due to some cerebral complication, or to apncea from paralysis of the respiratory muscles.

4. *Delirium Tremens*.—This form of alcoholism occasionally supervenes on a single debauch, but it much more frequently affects the chronic drinker. It generally comes on during a drinking-bout, but this may have terminated before the attack commences. In some cases it is undoubtedly determined by prolonged abstinence from food, mental distress, surgical operation or injury, or the onset of an acute disease, along with the ingestion of alcohol; in other cases no cause but the last can be traced. The first stage is indicated by inability to take food;

marked anxiety and restlessness; tremor of the voluntary muscles; furred and tremulous tongue; cool skin, which is frequently bathed in perspiration; cold hands and feet; and a soft weak pulse. There is complete insomnia, or short periods of sleep are interrupted by terrifying dreams, and the patient's nights are tormented by visions of horrid insects, reptiles, and other objects pursuing him and eluding his attempts to escape from them or to seize them. Illusions of hearing are not uncommonly added; but the sense of smell is much more rarely involved. If there is no improvement, these troubles not only haunt his nights, but persist in the day-time; he becomes more incoherent, his mental alienation increases, and attempts at suicide are common. The pupils are now greatly contracted, but there is no intolerance of light. The pulse quickens, and is very feeble or even dicrotic; and the general symptoms become more marked. A prolonged sleep may occur in this stage, and the disease thus terminate. If it continue, the strength fails; the pulse becomes small, weak, and thready; the tremor increases; the tongue gets dry and brown in the centre; persistent comatose and subsultus tendinum come on; the patient talks incessantly, and picks at the bed-clothes; finally death is ushered in by a delusive calm, or takes place in a paroxysm of violence. The writer has known cases in which an attack of delirium tremens always began by several severe epileptiform fits.

5. *Alcoholic Insanity*.—The forms of insanity caused by alcoholism are *acute mania* and *melancholia*, *chronic dementia*, and *oinomania*. In the first, homicidal impulses, and in the second, strong suicidal tendencies, due to actual delusions and not to mere passive terrors, are added to the other signs of delirium tremens. Oinomania is a peculiar form of insanity, in which the patient breaks out into paroxysms of alcoholic excess, attended with violent, strange, or even indecent acts, due to apparently uncontrollable impulses. The attack lasts a few days, and is succeeded by a long interval of sobriety and chastity. These patients have generally some hereditary taint; and not infrequently evidences, though often slight, of a morbid mental state may be detected in the intervals, if very carefully looked for. See INSANITY, Varieties of.

COMPLICATIONS.—Most of these have been already described, but chronic drinkers are especially liable to pneumonia of a low type, and to acute phthisis. Delirium tremens is very rarely complicated with meningitis; acute alcoholic gastric catarrh may be followed by jaundice; and cerebral hæmorrhage may come on in a drunken fit. Temporary albuminuria is occasionally caused by the ingestion of large quantities of spirits, and even of beer.

DIAGNOSIS.—The diagnosis of *acute alco-*

holic gastric catarrh, of insanity from alcohol, and of oinomania depends on obtaining a true history. Acute alcoholic coma can only be diagnosed with certainty by emptying the stomach, and examining its contents. Mere odour of the breath is quite fallacious; and the writer attaches but little importance to the state of the pupils, or to the general features of the coma. Convulsions sometimes usher in the condition; and apoplexy may arise from the accidental rupture of a blood-vessel whilst a person is drunk. Opium-poisoning can only be satisfactorily eliminated by examining the contents of the stomach. Uræmic poisoning may be diagnosed by testing the urine, though here an element of uncertainty is introduced by the occasional occurrence of albuminuria in alcoholic cases; the presence of hypertrophy of the heart, of dropsy, of casts in the urine, or other changes typical of Bright's disease, must decide the question. Delirium tremens is occasionally separated with difficulty from some forms of insanity not caused by drink; but in the latter cases *delusions*, not mere terrors or hallucinations, are of primary importance. The delirium of acute fevers and pneumonia may be mistaken for delirium tremens; but the pyrexia, history of the case, and physical condition of the patient will guide to a correct diagnosis if the possibility of error be remembered. *Chronic* alcoholism has been mistaken for some chronic nervous affections, such as locomotor ataxy, chronic softening and multiple sclerosis of the nerve-centres, paralysis agitans, chronic tremors from metallic poisons, senile dementia, and commencing general paralysis. The correct history, and a careful examination of the patient, are especially important in diagnosing alcoholic paralysis from other nervous affections, especially from locomotor ataxy and lead-paralysis. In all these maladies, *special* symptoms are present, besides those common to them and to chronic alcoholism.

**Prognosis.**—In the acute forms of alcoholism the prognosis is favourable so far as the immediate attack is in question. In acute coma, the patient generally, but by no means invariably, rallies from the state of insensibility; but he may die from the super-vention of a very rapid pneumonia. The prognosis in delirium tremens is favourable in young subjects; but its gravity increases with every attack, and with the co-existence of disease of the viscera, especially of the heart, liver, or kidneys. Patients with marked symptoms of fatty heart, or in whom pneumonia sets in, but rarely recover. Chronic alcoholism may be temporarily arrested; but the ultimate issue is unfortunately, as a rule, only too certain, for the habit is in most cases too strong to be broken off, or even to be checked for any lengthened period. Mental impairment, persistent tremors, ataxy, and signs of coarse

brain-lesions, are especially significant of a speedy termination.

**TREATMENT.**—The *acute gastric catarrh* due to alcohol is most rapidly subdued by washing out the stomach with copious draughts of tepid water, and then giving a saline purge. All forms of alcohol should be rigidly abstained from; and the diet must be simple, and taken in a fluid form for a day or two. Passive exercise in the open air, or, if the patient be vigorous, a brisk ride on horseback, is very beneficial.

In cases of *acute coma* the stomach should be at once emptied by means of the stomach-pump. Cold affusion, followed by energetic friction and the application of bottles filled with warm water, so as to keep up the temperature, will generally revive the patient. Galvanism, in the form of the interrupted current, may often be employed with advantage. If the patient be strong, a smart purge, or, if weak, a milder one, will be all the after-treatment that is necessary.

*Delirium tremens* must be treated differently in the young and in the old. In first attacks in young, strong subjects, after a sudden debauch, complete abstinence from alcohol, light and easily assimilated food (milk diet), moderate purgation, and occasionally tartarated antimony in doses of from one-twelfth to one-eighth of a grain, carefully watched, have been most efficacious in the writer's hands. If the patient has two or three restless nights in succession, bromide of potassium (thirty grains), or chloral hydrate (twenty grains), may be given, separately, or better in combination, at intervals of four hours, until sleep is procured, or sulphonal (thirty grains); but as the disease is spontaneously curable, sedatives must not be pushed. An experienced attendant should be always present, but no form of mechanical restraint is permissible. In older cases, a mild purge should begin the treatment; and light but very nourishing food should be administered at short intervals. Milk, beef-tea, raw eggs beaten up with milk, strong soups, and such articles are to be given freely; when, by careful management and good nursing, a very severe attack may be tided over, and natural sleep will return in from three to five days. The early administration of sedatives is to be deprecated; but should the restlessness persist, in spite of careful and assiduous feeding, a full dose of laudanum (℥xxx.—xl.) at bed-time is of great value. In the absence of albuminuria, lung-complications, or any sign of failure of the heart's action, the writer prefers this drug to other hypnotics. If the opium alone fail, its combination with an alcoholic stimulant (brandy, whisky, or stout) often succeeds. If there be any tendency to syncope, or if pneumonia should come on, as well as in cases complicated with shock, *e.g.* surgical injuries, a free use of stimulants

is imperative. Hypodermic injections of morphine, and large doses of digitalis, are recommended by many authorities; but the writer has seen great harm attend their free exhibition. The cautious inhalation of chloroform vapour has occasionally cut short an attack by inducing sleep, but it much more frequently fails. Mechanical restraint is seldom, if ever, necessary, if the patient be properly nursed and attended to. All methods of self-destruction must be carefully guarded against. A padded room, when required and available, is of the utmost benefit.

The great desideratum in *chronic alcoholism* is to substitute an easily-digested and nourishing diet for the alcoholic stimulants, which can then be safely dispensed with altogether. The practitioner's judgment, and his knowledge of the *cuisine*, are very important in the management of these cases. Strong meat-soups, and good specimens of the concentrated preparations of meat, are of great value. The strictly medicinal treatment will consist in the administration of bitter tonics, such as nux vomica, quinine in small doses, calumba, or gentian; with carminatives, such as spirit of chloroform, armoracia, and capsicum. Alkalis, effervescent mixtures, and hydrocyanic acid are peculiarly useful if the stomach be irritable. The condition of the liver and bowels should be carefully regulated. Bromide of potassium is in general the best sedative to employ against the insomnia, though chloral hydrate is more certain; but the latter should only be given occasionally, lest the patient acquire the habit of frequently resorting to it. In long-standing cases, cod-liver oil, arsenic in small doses, and oxide of zinc have all done good, but they require a long and protracted administration. Phosphorus has been of no use whatever in the cases in which the writer has tried it; but small doses of the more easily assimilable preparations of iron are occasionally well borne, and are then most useful. The craving for drink, if urgent, may be checked by small doses of opium, but this drug must be exhibited with extreme caution. Judicious supervision, and, in inveterate cases, a residence in a proper asylum, are the only means from which any permanent benefit can be expected. Absolute abstinence from alcohol cures almost every case of alcoholic paralysis, unless it has become quite hopeless; and massage and mild currents of electricity are of service in restoring the atrophied muscles. The treatment of insanity induced by alcoholism will not differ from that recommended in other forms, except in an enforced abstinence from its cause.

JOHN CURNOW.

**ALEPPO EVIL.**—See DELHI BOIL.

**ALEXANDERSBAD,** in Bavaria.—

Chalybeate waters and hydropathic establishment. See MINERAL WATERS.

**ALGID** (*algidus*, cold).—A word implying extreme coldness of the body, used only when it arises in connexion with an internal morbid state, such as cholera, or a special form of malignant remittent fever.

**ALGIERS.**—Warm winter climate. Mean winter temperature 61° Fah., liable to rapid changes. Heavy rains not infrequent. See CLIMATE, Treatment of Disease by.

**ALIMENT.**—Food or aliment furnishes the elements required for the growth and maintenance of the organism; and, through its action with the other life factor—air, forms the source of the power manifested.

The aliment of organisms belonging to the vegetable class is derived from the inorganic kingdom. Under the influence of the sun's rays the elements of inorganic principles are appropriated in such a manner as to lead to the construction of organic compounds and meet the requirements of growth. This constitutes the main operation of vegetable life, and in it we have the source of the aliment of animals, which can only appropriate organic compounds, and which, either directly or indirectly, derive these compounds from the vegetable kingdom. As the solar energy employed in the construction of organic compounds, through the agency of the vegetable organism, becomes locked up in the compound formed, such compound represents matter combined with a definite amount of latent force. In the employment, therefore, of organic matter as aliment by animals, we have to look upon it not only as yielding the material required for the construction and maintenance of the body, but as containing and supplying the force which is evolved under various forms by the operations of animal life.

Aliment constituting the source from which the several elements belonging to the body are derived, it follows that, to satisfy the requirements of life, it must contain all the elements that are encountered. It is not, however, with the elements in a separate state that we have to deal, but with the products of nature in which they are variously combined.

The *alimentary products* as supplied by nature are resolvable by analysis into a variety of definite chemical compounds. These constitute the *alimentary principles*. Some are common to both animal and vegetable food, as, for instance, albumin, casein, fats, &c.; others are peculiar to either the animal or vegetable kingdom. Starch, for example, is met with only in vegetable, and gelatine only in animal products.

With reference to the alimentary principles, it must be understood that in no case do they exist in natural products in an isolated form, and no single alimentary principle is capable

of supporting life. Although, however, it is with the alimentary products as a whole that we are practically concerned, yet, regarded from a scientific point of view, a knowledge of their constituent principles is required, to enable us to assign to them their proper value as alimentary articles; and for the purpose of systematic consideration some kind of classification is needed.

CLASSIFICATION.—Prout classified the constituent principles of food into four groups, which he named (1) the *aqueous*; (2) the *saccharine*; (3) the *oleaginous*; and (4) the *albuminous*. This classification is defective, inasmuch as it omits from consideration saline matter, which is equally as essential to nutrition as any other part of an alimentary product. The saccharine and oleaginous groups also stand as primary and independent divisions, whilst physiologically they are related, and may be conveniently considered under a combined heading.

Liebig proposed a classification based on physiological grounds; and, taking into account only the organic constituents of food, grouped them under the heads of (1) *plastic elements of nutrition*; and (2) *elements of respiration*. His *plastic elements of nutrition* comprise the nitrogenous principles; and to these he assigned the office of administering not only to the growth and renovation of the tissues, but also to the production of muscular and nervous power. Believing that the source of these powers sprang from the oxidation of the respective tissues, he held that the exercise of muscular and nervous action created a corresponding demand for nitrogenous alimentary matter, which thus became invested with an importance that led it to be regarded as affording a measure of the value of an alimentary article. By recent experimental research this view has been found to be untenable. The nervo-muscular organs are now looked upon as holding the position of instruments, by whose agency the force liberated by chemical action is made to manifest itself under certain other forms; and what is wanted for the purpose is simply oxidisable organic material, which may be derived from non-nitrogenous as well as nitrogenous food. The *elements of respiration*, or, as they were afterwards more appropriately styled, the *calorific principles*, represent the organic non-nitrogenous constituents of food. Their destination, according to Liebig, was heat-production. It is now maintained, however, as stated above, that they play a part in connexion with nervo-muscular action; and it may be also said that they are to some extent concerned in tissue-development. From the considerations set forth, Liebig's classification loses the scientific value it was at one time supposed to possess.

The following grouping of the alimentary

principles, based on chemistry, furnishes a classification which involves no theoretical proposition, and is practically convenient:—

Food is primarily divisible into *inorganic* and *organic* principles.

The *inorganic* principles consist of water, and the various saline matters required by the system. They are as much needed for the support of life as the organic portion of food.

The *organic* principles are sub-divisible into *nitrogenous* and *non-nitrogenous*; and the *non-nitrogenous* are again further sub-divisible into *fats* and *carbo-hydrates*.

The *nitrogenous* principles contribute to the growth and nutrition of the various bodily textures, and furnish the active constituents of the secretions. They also undergo resolution in the system into urea, which is excreted; and a complimentary portion, which is susceptible of application to force-production. They are thus capable of administering to all the purposes fulfilled by the organic portion of an aliment.

The *fats* are applied to the production of heat and other forms of force. They seem also to be essential to tissue-development generally, besides yielding the basis of the adipose tissue.

The *carbo-hydrates* (starch, sugar, gum, &c.) contribute to the formation of fat, and are also applied indirectly, if not directly, to force-production.

There are a few principles, such as alcohol, the vegetable acids, and pectin or vegetable jelly, which do not strictly fall within either of the preceding groups. Alcohol occupies a chemical position intermediate between the fats and carbo-hydrates; whilst the others are more highly oxidised compounds than the carbo-hydrates.

All alimentary products in the form supplied by nature contain organic and inorganic principles, and the organic principles comprise more or less of the nitrogenous and non-nitrogenous divisions. The non-nitrogenous division, however, is not always represented, looked at broadly, by both fat and carbo-hydrate. Milk, which may be regarded, from the position it holds in nature, as furnishing a typical representation of an alimentary article, contains principles from each of the groups specified in the preceding classification. See DIET. F. W. PAVY.

**ALIMENTARY CANAL, Diseases of.**—See DIGESTIVE ORGANS, Diseases of; and the several organs.

**ALI WAL NORTH, in Cape Colony.**—See AFRICA, SOUTH.

**ALKALINITY.**—The reaction of human blood is always alkaline; and, though the normal degree of alkalescence has not yet been determined, it is probable that, like the temperature of the body, it is tolerably constant. In disease considerable variation,

no doubt, occurs, but still the blood is always found alkaline. Pettenkofer and Voit found the serum of blood acid in a case of leukæmia some few hours after death, but not during life; and Sir Alfred Garrod states that in chronic gout the serum may become somewhat neutralised, but never acid. F. Hoffman has also found that the blood retains its alkalinity with great obstinacy: he fed pigeons for a considerable length of time on food yielding only acid ash, but the animals suffered from blood-poisoning before the alkalinity of the serum was neutralised. The alkalinity of the blood is maintained by the constant passage into it of the alkaline salts of the food, and of alkaline carbonates derived from the oxidation of the lactic, oxalic, and uric acids furnished by the disintegration of the tissues. The blood is probably prevented from becoming too alkaline by the withdrawal of its alkaline salts by the alkaline secretions, namely, the saliva, the bile, and the pancreatic fluid; whilst the acid salts, which, if accumulated, would tend to depress its normal alkalinity, are removed by the acid secretions, namely, the sweat, the gastric juice, and the urine, and by the exhalation of carbonic acid from the lungs. It has been shown that the withdrawal of acid by one secretion has a decided effect on the reaction of other secretions: thus the saliva becomes more alkaline during digestion, when the stomach is pouring out the acid gastric juice; and Dr. Bence Jones has shown that during digestion the acidity of the urine is lessened. A similar relationship is also shown to exist between the elimination of carbonic acid by the lungs and the acidity of the urine, the latter falling as the former is increased, and *vice versâ*. The importance of a proper degree of alkalescence for the blood is obvious, when we consider that this condition increases the absorption-power of its serum for gases, and is necessary to maintain its albumin in the liquid state, whilst oxidation is always more perfectly performed in alkaline solutions.

C. H. RALFE.

**ALKALIS.** — DEFINITION. — Inorganic substances, which turn syrup of violets green, and turmeric brown; and restore the blue colour to litmus which has been reddened by acids; they combine with acids to form salts; and their carbonates are soluble in water.

ENUMERATION.—The only substances which correspond with the above definition are—Potash, Soda, Lithia, and Ammonia. The alkaline earths—Lime, Magnesia, Baryta, and Strontia—and the alkaloids, have a similar action on vegetable blues and yellows; but the carbonates of the former group are almost insoluble in water; whilst the latter contain carbon, and are therefore classed with organic substances.

PROPERTIES.—Ammonia is distinguished from the other alkalis by its volatility. The non-volatile alkalis are readily recognised by their spectra; and by the colour they impart to the blowpipe flame, potassium giving it a violet, sodium a yellow, and lithium a carmine colour. Potassium and sodium are present as constituents of the body in considerable quantities; ammonium exists to a smaller amount; and lithium probably in traces. Sodium is found chiefly in the blood, potassium in the muscles.

ACTION.—When applied to the skin, dilute alkalis and their carbonates act as rubefacients. Pure ammonia is a vesicant, and potash and soda have a caustic action. Both caustic potash and caustic soda absorb water from the tissues, and form a corrosive fluid, which destroys the parts around, as well as that to which the caustic has actually been applied. To prevent this effect they are sometimes mixed with lime, which absorbs the water. A mixture of potash and lime forms the Vienna paste. When inhaled, ammonia causes irritation of the respiratory passages, and increased secretion of mucus. This irritation excites reflex contraction of the blood-vessels and consequent rise of blood-pressure. When swallowed in quantity, the caustic alkalis and their carbonates produce symptoms of irritant poisoning. In the case of ammonia these symptoms may be accompanied by those of inflammation of the air-passages, caused by the irritant vapour. The best antidote is dilute acid, such as vinegar. In small quantities and diluted, alkalis increase the secretion of gastric juice before meals. After absorption into the blood they render this fluid more alkaline; while potash appears especially to accelerate tissue-change, and is accordingly classed among the alteratives. When injected directly into the blood, potash acts specially on the muscles, which it paralyzes. Ammonia stimulates the motor centres in the brain and spinal cord, the respiratory centre in the medulla oblongata, and the accelerating nerves of the heart. When injected into the veins it therefore causes convulsions like those of strychnine, and quickening of the respiration and pulse. Alkalis are chiefly excreted by the urine; and potash, soda, and lithia lessen its acidity, or render it alkaline. Ammonia is partly excreted unchanged, but a portion passes out in the form of urea and uric acid; and it does not render the urine alkaline like the others. Potash and lithia act as diuretics; soda to a less extent; and ammonia least of all. The diuretic action does not depend on any change in the blood-pressure. Potash and ammonia are diaphoretic. Potash lessens the tenacity of mucus.

USES.—Dilute solutions of potash and soda relieve itching in skin diseases. Caustic potash or soda is used to destroy warts; to cauterise poisoned wounds and ulcers;

to open hydatid cysts in the liver; and to establish issues. Ammonia neutralises the formic acid which renders venomous the stings of bees, ants, and mosquitoes, and is therefore applied to relieve the pain which they cause. The intravenous injection of ammonia has been recommended as an antidote in snake-poisoning; but the value of the remedy is not established. Mixed with oil, so as to form a liniment, ammonia is used as a rubefacient in sore throats, bronchitis, rheumatic pains, and neuralgia. It is inhaled to relieve headache; as a restorative in syncope and shock, when it raises the blood-pressure; and to facilitate expectoration in chronic bronchitis. Alkalis administered after meals act as antacids, and relieve heart-burn. When given before meals they increase the secretion of gastric juice, quicken digestion, and relieve weight at the epigastrium, pain between the shoulders, and flatulence. Bicarbonate of sodium is usually given for this purpose, but when the stomach is very irritable, liquor potassæ is preferred, as it is considered to have a sedative action on the mucous membrane. Alkalis appear to lessen the transformation of glycogen into sugar, and they are used on this account in diabetes. Liquor potassæ sometimes helps to reduce obesity. Alkalis are used in the treatment of scrofula, rheumatism, gout, and lithiasis; but in the two last-mentioned diseases lithia is considered the most valuable, whilst potash is preferred to soda, as the urate of lithium is most soluble, and the urate of sodium least so. The salts of certain organic acids, such as the acetate or citrate, may be employed as remote antacids to render the urine alkaline, as they undergo combustion, and are converted into carbonates in the blood. Alkalis are given to lessen the acidity of the urine in inflammation of the bladder or urethra, and potash is employed as a diuretic in dropsies. On account of its stimulating action on the heart and respiration, ammonia is administered in adynamic conditions and in chronic bronchitis.

T. LAUDER BRUNTON.

**ALKALOIDS and other ACTIVE PRINCIPLES.**—**DEFINITION.**—An alkaloid is a substance formed in the tissues of a plant or of an animal, having a definite composition as regards the proportions of the chemical elements of which it is composed, and capable of combining, like an alkali, with acids to form salts. All alkaloids contain nitrogen; and all, except conine, nicotine, and sparteine, contain oxygen.

Besides alkaloids there are other active principles found in plants, which have also a powerful influence on the animal economy, but do not possess all the chemical properties just stated.

**CHEMICAL COMPOSITION AND RELATIONS.**—These are briefly expressed in the above de-

finition. Thus morphine, for example, one of the alkaloids of opium, has always the chemical composition represented by the formula  $C_{17}H_{19}NO_3 \cdot H_2O$ , and it may unite with acetic acid to form acetate of morphine, just as potash may unite with the same acid to produce acetate of potassium. But the empirical formula  $C_{17}H_{19}NO_3$  represents only the percentage composition of the substance in the simplest numbers, and does not express how the atoms of the different elements are related to each other. For, just as ethylic alcohol, with the composition  $C_2H_6O$ , is believed by the chemist, from its behaviour towards other bodies, to contain a 'radicle,' or group of atoms,  $C_2H_5$ , having certain chemical properties resembling those of a base, such as potassium, K; and just as this radicle,  $C_2H_5$ , may replace one of the hydrogens of water, so as to form alcohol ( $C_2H_5 + H_2O = \frac{C_2H_5O}{H} + H$ );

so chemists have good reason for believing that alkaloids belong to the group known as *amines* or *amides*, which are really ammonia,  $NH^3$ , in which one or more of the atoms of hydrogen are replaced by a radicle; in other words, they are ammonia bases, combining with HCl without elimination of  $H_2O$ . Most alkaloids are derivatives of pyridine.

It is obvious that two or more alkaloids may resemble each other in percentage composition, and still be very different, both in their chemical *structure*, and, necessarily, in their physiological action. Thus strychnine ( $C_{21}H_{22}N_2O_2$ ), quinine ( $C_{20}H_{24}N_2O_2$ ), and cinchonine ( $C_{20}H_{24}N_2O$ ), differ only in a few atoms of carbon or of oxygen, more or less; but they have different physiological actions, showing that their chemical structure, which is not indicated in these formulæ, must also be different. The physiological action of an alkaloid may also be modified by combining it with another substance. Thus, as was pointed out by Crum-Brown and Fraser, compounds of strychnine with methyl, ethyl, and amyl, do not present the well-known physiological action of that substance, but one analogous to that of woorara.

**ENUMERATION.**—The alkaloids and other active principles most familiar to the physician are:—Morphine, Apomorphine, Narcein, Codeine, Thebaine, Narcotine, Papaverine; Atropine, Hyoscyamine, Hyoscyne, Daturine; Nicotine; Conine; Physostigmine or Eserine; Strychnine, Brucine; Quinine, Cinchonine, Cinchonidine; Beberine; Caffeine; Cocaine; Theobromine; Aconitine; Veratrine; Digitalin; Curarine; Muscarine; Santonin; Ergotin; Emetine; Pilocarpine; Salicin; and Strophanthin. For the alkaloidal substances formed in dead bodies and in animal tissues, see PTOMAINES.<sup>1</sup>

<sup>1</sup> Names of alkaloids are now made to end in *ina* (Latin) or *ine*—thus: morphina or morphine. The names of non-alkaloidal active principles terminate in *inum* Latin) or *in*—thus: digitalinum or digitalin.

**SOURCES.**—The majority of alkaloids are formed by plants. The function which they subserve in the economy of the plant is not known. Some plants produce only one alkaloid, while in others two or more are formed. A few of the alkaloids have been produced synthetically by the chemist.

**PHYSIOLOGICAL ACTION.**—Alkaloids have various degrees of physiological activity when introduced into the animal body. Many are slow in their action, and a large dose is required to produce any observable effect; while others act more rapidly, and are so potent that even a minute dose may destroy life. Compare, for example, narcotine, one of the alkaloids of opium, with nicotine, the alkaloid of tobacco. Twenty to thirty grains of the former have been taken by the human subject without producing any marked symptoms, while the twentieth part of a grain of the latter may induce symptoms so severe as to threaten death. It is also well known that alkaloids may have a different kind of action on different animals. Thus one-fourth of a grain of atropine will produce serious symptoms of a complex character in a dog, while three or even four grains may be given to a rabbit without causing any more marked effect than dilatation of the pupil. In considering the physiological actions of these substances, the following generalisations may, in the present state of science, be made tentatively:—1. As a general rule, the more complex the organic molecule, and the greater the sum of its atomic weight, the more intense will be the action of the substance. 2. Substances which split up quickly into simpler bodies produce rapid but transient physiological effects; whereas substances which resist decomposition in the blood or tissues may produce no appreciable results for a time, but when they do begin to break up, the effects are sudden and violent, and usually last for a considerable time. 3. Alkaloids have frequently a double action on different parts of a great physiological system; and their action in a particular group of animals will depend on the relative degree of development of the parts of the system in that group. Thus most of the alkaloids of opium have such a double action—a convulsive action resembling that of strychnine, due to their influence on the spinal cord or on the motor centres in the brain; and a narcotic or soporific action resembling that of anæsthetics, due to their influence on sensory centres in the brain. Hence, in animals where the spinal system predominates, as in frogs, these alkaloids act as convulsants; while in the higher mammals their principal action is apparently on the encephalic centres, which have now become largely developed.

Passing to the consideration of the action of the individual substances, we cannot do more than give, by way of example, a brief *résumé* of our knowledge regarding a few of them.

1. **Morphine** ( $C_{17}H_{19}NO_3$ )—an alkaloid of Opium. In the frog this substance has an action resembling that of strychnine. At first there is a state of agitation, followed by tetanic spasms; finally, all reflex actions, including those of the heart and of respiration, are paralysed. Pigeons have been found to possess a remarkable power of withstanding the influence of this drug—an ordinary sized bird requiring about two grains to kill it. Rabbits become partially somnolent, show a tendency to reflex spasms, and tolerate a large dose—say about one-half to one grain per pound weight of the animal. In the dog the intravenous injection of even one-tenth of a grain (for a small animal) causes agitation followed by sleep; the pulse and respiratory movements are slowed; the smaller arteries become (at least during one stage) contracted, so as to cause an augmentation of general blood-pressure; the pupil is contracted; and, if the dose be large, death may be preceded by convulsions. In the higher mammals morphine acts chiefly on the sensory apparatus, both peripheral and central.

2. Other alkaloids of opium have also been investigated:—(a) **Narcein** ( $C_{23}H_{29}NO_6$ ) is a pure hypnotic, causing profound sleep. Even in large doses it does not produce convulsions. (b) **Codeine** ( $C_{18}H_{21}NO_5$ ) has an action mainly like that of morphine. (c) **Thebaine** ( $C_{19}H_{21}NO_3$ ) causes tetanic convulsions, thus resembling strychnine. (d) **Narcotine** ( $C_{22}H_{23}NO_7$ ) is slightly narcotic, but strongly convulsant. (e) **Papaverin** ( $C_{20}H_{21}NO_4$ ) causes a somniferous action like that of narcein. It is evident, therefore, that opium, which may contain more or less of all of these substances, must have an action on the body of a very complicated character.

**Apomorphine** ( $C_{17}H_{17}NO_2$ ), a derivative of morphine, has none of the characteristic actions of that substance, but acts chiefly as a vascular depressant and as an emetic.

3. **Strychnine** ( $C_{21}H_{22}N_2O_2$ )—the alkaloid of *Strychnos nux vomica*. In the frog very minute doses cause convulsions of all the voluntary muscles, excited by peripheral irritation. These convulsions are due to the action of the poison on the spinal cord, as they persist after decapitation. In warm-blooded animals the reflex character of the convulsions is less evident; they have more of a tonic character, and chiefly affect the extensors. The exact *modus operandi* of the poison on the cord is unknown, but in some way or other it heightens its reflex sensibility. Death is usually the result of asphyxia from arrest in spasm of the respiratory mechanism, but it may result from exhaustion. **Brucine** ( $C_{23}H_{26}N_2O_4$ ), another substance found in *nux vomica*, appears to have an action like that of strychnine, but more feeble.

4. **Atropine** ( $C_{17}H_{23}NO_3$ )—the chief alkaloid of *Atropa belladonna*. In the frog it causes tetanic reflex spasms. Herbivorous animals,

as a rule, have a tolerance of this poison, so that its effects are best studied in carnivora. Even in these the action is somewhat uncertain. Respiration may be paralysed without general convulsions; the pulse is quickened, from paralysis of the inhibitory action of the pneumogastric nerve on the heart; and the arterial pressure is increased. After very large doses the arterial pressure may be diminished, with paralysis of all parts containing involuntary muscular fibre. Secretion is diminished. The pupil is dilated—apparently by a direct influence of the poison on the centres or nervous arrangements in the iris itself, as the effect may be observed even in an eye removed from the head. **Hyoscyamine**, and **Hyoscine**, the alkaloids of *Hyoscyamus niger*, and **Daturine**, the alkaloid of *Datura stramonium*, have actions allied to the action of atropine.

5. **Digitalin** ( $C_{27}H_{44}O_{15}$ )—the so-called active principle of *Digitalis purpurea*, probably a mixture of several bodies. A large dose causes slowing of the heart. If the dose be increased, the heart is arrested in diastole, and will not respond to direct excitation. With medium doses there is a period of acceleration of the heart, but this period may rapidly pass into that of slowness just mentioned. This effect on the heart has not yet been clearly accounted for, and it remains to be decided whether it be due to the influence of the drug on the terminations of the pneumogastric, or of the sympathetic, or on the intracardiac ganglia themselves. Coincident with the action on the heart, the smaller arteries are contracted, and the arterial tension is increased. *Digitalis* would appear to have little effect on involuntary muscle, but it exerts a potent action on voluntary muscle, which, after small doses, becomes feeble in contractile power, while large doses may abolish contractility altogether.

6. **Physostigmine**, or **Eserine**, ( $C_{15}H_{21}N_3O_2$ )—the active substance of *Physostigma venenosum*, or Calabar bean. As has been pointed out by Professor Fraser, this alkaloid has an action antagonistic to that of atropine. Sensibility and consciousness remain until death; the voluntary muscles are paralysed; involuntary muscles are said to show tetanic contractions; respiration is at first accelerated, and afterwards slowed; the vessels become alternately dilated and contracted; secretion, especially that from the lachrymal and salivary glands, is increased; and the pupil is contracted. Physostigmine appears to paralyse the anterior cornua of the spinal cord.

7. **Curare**—a resinous substance, containing an alkaloid, **Curarine**, of the composition  $C_{16}H_{15}N$ , obtained from certain parts of South America, and used by the natives of these regions as an arrow-poison. It is probably obtained from certain plants belonging to the genera *Strychnos* and *Paullinia*.

Its distinctive physiological action is abolition of the power of all voluntary movement, in consequence of its action, as was proved by Claude Bernard, upon the peripheral terminations of motor nerves—the 'terminal plates' of muscle. Respiratory movements are arrested as the result of paralysis of the muscles of respiration, but the heart may continue to beat for a considerable time. If artificial respiration be established, the circulation may be maintained for several hours, while the animal is completely under the influence of the substance. All the secretions are increased, and the mean temperature falls.

8. **Muscarine**—the alkaloid of *Agaricus muscarius*. It causes arrest of the heart's action in diastole, an effect which may be removed by the influence of atropine, thus affording an instance of physiological antagonism. In warm-blooded animals muscarine slows the heart's action; the blood-pressure falls; respiration is first embarrassed, and may be completely arrested; parts containing involuntary muscle are in a state of tetanic spasm; the pupil is contracted; and secretion is increased.

9. **Santonin** ( $C_{15}H_{16}O_3$ )—the neutral crystalline principle of *Artemisia maritima*. This drug may cause nausea, vomiting, hallucinations, vertigo, and a peculiar state of visual sensation—the field of vision usually appearing yellow, but sometimes violet. It is said that the stage of violet rapidly passes into that of yellow, and therefore it is probable that santonin may first excite the retinal fibres sensitive to violet (according to Thomas Young's theory of colour-perception), and afterwards paralyse them. In large doses, santonin causes loss of consciousness, tetanic convulsions, and death.

10. **Ergotin**—the so-called active principle of Ergot. It causes contraction of the smaller blood-vessels, contractions of the uterus, and slowing of the pulse; and the animal may die in consequence of arrest of the action of the heart.

11. **Quinine** ( $C_{20}H_{24}N_2O_2$ )—one of the alkaloids of Cinchona. In small doses quinine accelerates the heart in the warm-blooded animal; in moderate doses it slows it; and in large doses it may arrest it, and cause convulsions and death. Research shows that its action is essentially upon the central nervous system. It destroys all microscopic animal organisms, apparently killing vibrios, bacteria, and amebæ; but it seems to be without action on humble organisms belonging to the vegetable kingdom. It arrests the movements of all kinds of protoplasm, including those of the colourless corpuscles of the blood. It arrests fermentative processes which depend on the presence of animal or vegetable organisms, but it does not interfere with the action of digestive fluids.

12. **Nicotine** ( $C_{10}H_{14}N_2$ )—the alkaloid of Tobacco. It stimulates and then paralyzes

the secretory nerves of glands, and also the nerves of involuntary muscles. It causes cold sweats, a feeble circulation, and fainting; a large dose causes death by failure of respiration, attended by severe convulsions. The blood-pressure falls at first, but it may then rise, and the pulse-rate is rapid. Nicotine first stimulates and then paralyses the ends of the vagi in the heart.

13. **Cocaine** ( $C_{17}H_{21}NO_4$ )—from *Erythroxyton coca*. This is a local anæsthetic, acting on the terminations of the nerves of the skin and of mucous membranes. Its general action resembles that of caffeine, in small doses lessening fatigue, in larger doses causing weariness, deafness, and inability to think clearly. Its action is first on the cerebrum, then on the medulla, and lastly on the spinal cord. Very large doses may cause convulsions, of cerebral origin, or due to paralysis of the respiratory centre; the sensory columns of the cord are paralysed, while the motor are unaffected. Small doses raise the blood-pressure and quicken the pulse; large doses have the reverse effect. The secretions are generally diminished.

14. **Pilocarpine** ( $C_{11}H_{16}N_2O_2$ )—from *Pilocarpus pennatifolius* or *Jaborandi*. Pilocarpine stimulates the secretory nerves of glands, causing especially copious secretion from the salivary and sweat-glands. It also increases the secretion from the bronchial mucous membrane, from the glands of the stomach and intestines, and from the kidneys. It does not increase the secretion of bile, and its action on the mammary gland is uncertain. In large doses it may cause unsteadiness of movement, with a tendency to rotate, twitchings of muscles, shivering, and dyspnoea, showing an action on nerve-centres. In these circumstances the pupil is contracted, and there is indistinct vision, from spasm of the ciliary muscle. Large doses paralyse the vagus endings in the heart, and there may also be spasmodic contractions of the muscular walls of the stomach, intestines, and bladder. During profuse sweating the temperature rises. There may be death from sudden collapse.

15. **Strophanthin** ( $C_{16}H_{26}O_8$  ?)—the active principle of *Strophanthus hispidus*. It resembles digitalin in its general action: it stimulates all striated muscles; in large doses, it may arrest the heart in systole; and it also acts as a diuretic.

JOHN G. MCKENDRICK.

**ALLANTIASIS** (ἀλλᾶς, forced meat).—Sausage-poisoning. See POISONOUS FOOD.

**ALLEGHANY SPRINGS**, in Montgomery county, Virginia, U.S.A.—Calcic or earthy waters. See MINERAL WATERS.

**ALLEVAUD**, in France (Isère).—Sulphur waters. See MINERAL WATERS.

**ALLOCHIRIA** (ἄλλος, another; and χεῖρ, the hand).—A peculiar disturbance of cutaneous sensibility in which a tactile sensation is referred to another part of the body than the seat of actual impression; or to a corresponding part of the opposite side of the body. See SENSATION, Disorders of.

**ALOPECIA**.—See BALDNESS.

**ALOPECIA AREATA** (ἀλώπηξ, a fox).  
SYNON.: *Area*; *Alopecia circumscripta*; *Porriigo decalvans*. Fr. *Alopécie*; Ger. *Fuchsräude*.

**DEFINITION**.—A non-contagious atrophic disease of the hair, distinguished by the rapid development of more or less circular bald patches; in rare cases the baldness is general.

**SYMPTOMS**.—Alopecia areata is most common on the scalp, and is generally limited to that region, the occiput being the part most frequently affected. In some instances the beard and eyebrows suffer, and in very rare cases the whole of the hair is lost. Alopecia areata is usually confined either to a single round, perfectly bald spot, or to several spots irregularly scattered about the scalp; when, however, the disease attacks the occiput, it is often roughly symmetrical. The same remark applies to the eyebrows and eyelashes, and to all very severe cases, which have a strong tendency to become symmetrical. A characteristic feature of the disease is the rapidity with which the hair falls off over a limited area, leaving very few, if any, stumps on the bald patch, which is bounded by hair of apparently natural growth. When, however, the hair round the area comes out very easily, it may safely be predicted that the spot will increase in size. The part affected is sometimes pinker than the surrounding skin, but much more commonly it is of the same colour, or paler. There is sometimes a slight atrophy of the skin, as well as of the hair, so that the area has a shallow, cupped character; this is comparatively rare, and therefore cannot be due simply to the loss of hair.

**ÆTIOLGY AND PATHOLOGY**.—The causes of alopecia areata are not known. The immediate origin of the disease is probably some nerve-disturbance, leading to atrophy of the roots of the hair and sometimes of the skin. This view is confirmed by the fact that there is occasionally, though rarely, a temporary loss of sensibility over the area; and even when this is not the case, the skin is less sensitive to irritants than is normal. Sometimes the loss of hair is preceded by neuralgic pains, or tenderness on pressure, but more commonly there are no subjective sensations. The disease is rather more common in childhood and youth than in middle life, while it is seldom seen in old age; the writer has, however, met with one case in a man

over seventy years of age, who completely recovered. Alopecia is probably equally common in males and females, though on this point there is some difference of opinion. The old idea that the disease was due to a parasitic fungus has now been given up, in consequence of the complete failure of all attempts to demonstrate the fungus; moreover many of the clinical features of the disease, especially its great liability to recur after complete absence for perhaps years, is not at all in accordance with a parasitic origin. There is certainly sometimes a family tendency to alopecia. In extreme cases of alopecia areata, in which all the hair is lost, the nervous origin of the disease is generally admitted.

With regard to its morbid anatomy, it may be said that it is often difficult to find stumps, but when present they are very characteristic. They are also sometimes valuable for the purpose of differential diagnosis, since they are unlike the stumps of ringworm, being straight and thicker at the free end than at the point of insertion, which is contracted into a sort of neck, ending in a small, rounded, atrophied root. A very slight amount of traction removes them entire; in this respect they differ much from ordinary ringworm stumps.

**DIAGNOSIS.**—Ringworm is the only disease which is liable to be mistaken for alopecia areata. The difficulty arises thus: in common ringworm, either from treatment or from some other unknown cause, the hair sometimes falls rapidly off instead of breaking as it usually does. When the hair comes out in this way, with root attached, a smooth bald spot is left, exactly like alopecia areata; under these circumstances, unless a stump can be obtained for examination, the diagnosis is very difficult. The presence of other cases of ringworm in the same house, and the history of the patient, may be a guide to diagnosis.

Dr. Crocker mentions a very interesting and instructive series of cases of 'eight children in one family, who, while at the seaside, had each a few small perfectly bald spots on their heads; they were quite bare from the first, and never larger than half-an-inch in diameter.' After a time the governess contracted the disease, which was believed by her doctor to be alopecia areata, and not contagious; she, however, slept with her sister, who afterwards showed similar spots. Dr. Crocker says: 'The hairs round were loose: there were no short hairs, but one pulled out of the border showed distinct fungus-elements, indistinguishable from *tinea tonsurans*. In no case were there more than three spots, and they were all small. In one child there was a history of a red ring on the side of the cheek.' The writer has himself met with three similar examples. The first was in a school of about forty-five girls, where there were upwards of twenty girls affected with

small bald spots on the head, mostly free from stumps; there were, however, in a few cases a sufficient number of stumps full of ringworm fungus to make the diagnosis certain. The medical officer to the school correctly diagnosed ringworm, although the appearance of most of the children would have deceived anyone had the cases occurred singly. The second instance was in a boys' school, in which sixteen or seventeen cases of bald spots on the head occurred. The writer saw in the first instance only two boys, who were sent to him before it was known that any others in the school were affected. Both boys had small bald spots like area, but on one of the spots were ringworm stumps. The writer recommended that all the boys in the school should have their heads examined; this was done, and then many others were found to have bald spots. He subsequently saw one or two other boys from the same school with bald spots, which he had no doubt, under the circumstances, were ringworm. In this instance the disease had been called alopecia areata, and pronounced not contagious. The third case was in a family of children.

There is one point especially worthy of notice in all these curious outbreaks of *bald ringworm*: the spots were for the most part small, and by no means always circular at the margin, like area. On the contrary, some of the spots were angular, and others narrow and long like scars; indeed the shapes of the bald spots were in most cases not like those of typical area, although in every other respect there was a very close resemblance, so that it would be almost impossible to diagnose an isolated case as *tinea tonsurans*, unless some of the affected hairs remained. A favourable point in these cases is that when the hairs have all been shed, the risk of further contagion is much diminished; on the other hand, the disease spreads very rapidly *at first*, for every hair that is shed is a centre of infection.

**TREATMENT.**—The prognosis in most cases of alopecia areata is favourable, but the disease is very tedious. The treatment is partly local and partly general; the local treatment consists in the application of stimulating remedies to the bald patch, of which the best is cantharides. The acetum cantharidis, diluted with one or two parts of spirit, and painted on daily with a camel's-hair brush, answers very well. The lotion used should not be strong enough to blister, because its daily use cannot then be continued. Of ointments, the chrysophanic acid is the most useful, but it is disagreeable. The general treatment should consist of suitable tonics, and wine or beer, with plenty of rest and change of air. Continued physical fatigue appears in many cases to favour the development of alopecia areata.

ROBERT LIVEING.

**ALPHOS** and **ALPHOIDES** (ἀλφός, white).—Terms signifying white and white-looking, associated with the whiteness of the discs of common psoriasis: hence *lepra alphos* and *lepra alphoides*. See PSORIASIS.

**ALPHOSIS** (ἀλφός, white).—Whiteness, or the process of turning white. See ACHROMA.

**ALTERATIVES**.—DEFINITION.—Medicines which gradually restore the nutrition of the body to a healthy condition, without producing evacuations, or immediately exerting any very evident action upon the nervous system.

ENUMERATION.—The principal alteratives are—Nitric and Nitro-hydrochloric acids; Chlorine and Chlorides; Iodine and Iodides; Sulphur and Sulphides; Potassium and its salts; Mercury and its salts; Phosphorus and Hypophosphites; Antimony; Arsenic; Taraxacum; Sarsaparilla; Hemidesmus; Guaiacum; Colchicum; and Mezereon.

ACTION.—Healthy nutrition depends on the digestion of the food, its assimilation by the tissues, the decomposition of the tissues during the exercise of their functions, and the removal of their waste products being performed in a proper manner—in due proportion one to another. If the food is not properly digested, as in dyspepsia; or is not properly assimilated, as in diabetes; if the tissues break up too rapidly, as in fever; or if the waste products are not properly removed, as in some cases of kidney-disease, nutrition suffers. Digestion and excretion may be improved by tonics, purgatives, and diuretics; but alteratives seem to exert their action upon assimilation and tissue-change. The digestion of food is effected by means of ferments, such as those of the salivary glands, stomach, pancreas, &c. Some also of the changes, such as the conversion of glycogen into sugar, which the food undergoes after absorption in the liver, and even certain so-called vital actions—such as the coagulation of the blood—are produced by a similar agency. It is not improbable that the histolytic changes in the tissues are also effected by ferments. They do not depend upon oxidation, for although during health the products of tissue-decomposition are oxidised as fast as they are formed, yet under certain circumstances the tissues are split up so rapidly that the products which they yield are only partially oxidised. This is seen in poisoning by antimony, arsenic, and still more markedly by phosphorus, where such tissues as the muscles become decomposed, yielding nitrogenous substances, such as leucin, tyrosin, or urea, and fat. The former are excreted in the urine; while the last, instead of undergoing combustion, accumulates in the place formerly occupied by the muscular tissue, which is accordingly said to be in a state of fatty degeneration. It is

possible then, although by no means certain, that alteratives influence nutrition, either by modifying the activity of ferments, or by altering the susceptibility of the tissues to their action.

Mercurials in purgative doses, taraxacum, nitric and nitro-hydrochloric acids, probably act by modifying the digestion of the food in the upper part of the small intestine, or by affecting the changes which it undergoes in the liver after absorption. Potash has probably an action on the muscles. Antimony, arsenic, and phosphorus especially affect the nervous and cutaneous systems. Mercury has a peculiar power of breaking up newly-formed fibrinous deposits, and particularly syphilitic growths. Iodine, iodides, and probably chlorides, act upon the lymphatic system and promote absorption.

USES.—Purgative doses of mercurials, taraxacum, nitric and nitro-hydrochloric acids are useful in cases of frontal headache, general malaise, and depression of spirits, associated with symptoms of so-called biliousness, or with the appearance of urates or of oxalates in the urine. Potash and colchicum are employed in the treatment of gout. Phosphorus and arsenic are used in cases of nervous debility, as well as in nervous diseases, such as neuralgia and chorea, in which antimony is also serviceable. Arsenic is also given in diseases of the skin; and antimony in inflammation of the mucous membrane of the bronchi. Mercury in alterative (that is, small) doses, which are absorbed into the circulation without purging, is used to break up newly-deposited fibrinous masses, as in iritis, pericarditis, &c., and to counteract the effect of syphilitic virus upon the soft tissues in the secondary stage of this disease. Iodine and iodides act on the lymphatic system, and are useful in removing glandular swellings. By stimulating the absorbent system they may also assist in the removal of the fibrinous deposits and syphilitic growths disintegrated by mercury. The iodides are sometimes given in the secondary, but are still more valuable in the tertiary, stage of syphilis.

T. LAUDER BRUNTON.

**ALVENEU**, in Switzerland.—Sulphur waters, and chalybeate water, with iodine and common salt. See MINERAL WATERS.

**ALVEOLAR**.—A word used in pathology as descriptive of any morbid growth which consists of small cavities or spaces (*alveoli*), usually occupied by contents, and bounded by walls formed of cells or fibres. Alveolar cancer is the most familiar application of the term, being a synonym for colloid cancer. See CANCER.

**AMAUROSIS** (ἀμαύρωσις, a darkening or dulling of the sight).—DEFINITION.—This term cannot be strictly defined. Literally, it means an *obscurity of vision*, a state of blind-

ness, in the popular sense of the term, whereby nothing more is learnt than that the patient cannot see well enough for practical purposes, and is thereby unfitted for the usual occupations of life. Besides this, it is always tacitly understood that an external observation of the organ of vision, during the life of the patient, does not reveal any ostensible cause of blindness. It is further understood that the use of glasses is no remedy in amaurotic cases. It is rather the kind, than the degree, of blindness that is called amaurotic; but it must be observed that lesser degrees of blindness, of the amaurotic type, are generally, vaguely and indefinitely, called *amblyopic*.

**ÆTIOLGY.**—The causes of amaurosis have been more recently specifically attributed to morbid conditions of the percipient nervous apparatus of the eye. All cases are excluded in which, in the present state of science, and using the ophthalmoscope, we can see any morbid condition. But very few cases are now, in the statistical tables of the chief eye-hospitals, included under the head *amaurosis*. Some few cases seem likely, at least for some time to come, to be called by this term. The ophthalmoscope has enabled us more accurately to classify a large majority of the cases formerly called amaurotic. Many new names are thus employed whereby we gain more definite information. If only, as in some of them (e.g. 'white atrophy'), we have substituted the name of a particular ophthalmoscopic sign for an indefinite symptom, at least we can speak more accurately of the part that is or has been diseased—of the retina, or of the ocular end of the optic nerve.

In a large majority of the cases commonly classed as those of amaurosis, it is found ophthalmoscopically that there is 'white atrophy' of the optic nerves. The 'discs' are nearly or quite bloodless—white, not pinky-white; and the nerve-fibres going to the retina, being more or less wasted, there is some excavation of the discs, perhaps so much that the lamina cribrosa, in one or both, is exposed to view, while the retinal vessels are somewhat diminished in size. The causes of this condition are, most commonly, intracranial tumours or other diseases which induce pressure upon the optic nerve, or lead to an extension of inflammation, followed by œdema or double optic neuritis (descending), these terminating in the atrophy and amaurosis. The nerve-disease is often due to syphilis. But some cases of white atrophy occur in which there has been no precedent neuritis. Of such 'tobacco amaurosis' is an example, in which, unless smoking be given up, by an idiosyncrasy of the patient, he soon becomes blind. But nerve-atrophy or inflammation should be no longer called amaurosis—they have obtained a better nomenclature. The preceding stages of the diseases causing them, if, as is rarely the case, unaccompanied by any definite

ophthalmoscopic signs, and yet producing a considerable amount of blindness, may, for want of better knowledge, at present be called *amaurotic*. Other such cases include those reported as snow-blindness; those in which blindness has been produced by a lightning-flash near the eye or by a blow on the eye without other mischief resulting; disuse of an eye in children, as in some neglected squint cases; irritation from some branches of the fifth nerve (dental caries, &c.); anæmia after excessive losses of blood; suppression of menses; blood-poisoning by tobacco, lead, quinine; uræmia; and some cases of cerebral apoplexy. Embolism of the central artery of the retina occurs, but it is easy of diagnosis with the ophthalmoscope, and therefore should not be called amaurotic. The writer does not think there are any cases of *long-standing* blindness that show no ophthalmoscopic changes.

**SYMPTOMS AND DIAGNOSIS.**—There is one symptom of amaurotic blindness, affecting both eyes, which is noteworthy, as constituting, *primâ facie*, a general distinction between it and the other cases of blindness not of nervous origin: the gait and general aspect of the patient are peculiar—he is hesitating and hopeless-looking. He no more tries to see objects. He holds up his head; the eyes are open and turned upwards. He feels his way with his feet, and his hands are extended before him. He does not look towards you, or at anything in particular. But amaurosis does not by any means imply a similar state of vision in both eyes, nor that the blindness is to be taken in the ophthalmological sense, *i.e.* wanting perception of light. It would be well if any less degree of imperfection of vision, without evident cause, might be called *amblyopic*, but the two eyes must be considered separately.

To diagnose the absence or presence of a power of perception of light, the patient should be placed opposite to a bright light, such as a gas-lamp, and near to it, but not so near that he can feel the heat of it; the light is then turned up and down, and it is fully exposed and obscured, and the patient is asked many times, in quick succession, if he sees light or not. Any other blindness than this of absolute amaurosis, or originating in any other diseases than those of the percipient nervous apparatus of the eye, is never so great as to prevent the perception of light. If the patient can distinguish light from darkness, we next test whether he can see shadows of some small objects—of the hand, or of one finger only, passed between him and the burning light, or the light of the window only. If he can see to count fingers, his blindness is insufficient to indicate what is called amaurosis. Another point in the diagnosis of amaurosis is that, ophthalmoscopically, the appearance of the fundus of the eye is normal, or such as, independent of errors of

refraction, we find in other cases compatible with standard vision, or at least with a fair amount of useful vision. This will allow of a considerable latitude, and will not include any slight or imaginary hyperæmia or anæmia of the optic disc, any physiological excavations of the same, or congenital opacities of the retina. The pupil of the affected eye is, if the other be perfectly excluded from light or vision, nearly always dilated to almost the greatest extent, though atropine dilates it yet more fully; and it is fixed, being insensible to light.

**PROGNOSIS.**—After a due consideration of the cases thus classed together—and they are very unlike in fact, and often very obscure—we may say generally, that if the blindness be of one eye only, sudden and recent, the prognosis is hopeful; but if both eyes are affected, and the disease, whatever it may be, is of steady progress and of long standing, it is very serious. The cases of amaurosis are very rare indeed in which vision is perfectly restored; most of them end fatally to vision, or would so end but that the disease is sooner fatal to life.

**TREATMENT.**—This must necessarily be varied according to the cause of the amaurotic condition. For instance, if there is intracranial disease, treatment directed thereto must be followed out; and should there be indications of syphilis, iodide of potassium and small doses of mercury must be given for some time. When amaurosis depends on any injurious habit, such as smoking, this must be relinquished. Large doses of strychnine and iron are useful in advanced white atrophy.

J. F. STREATFEILD.

**AMBLYOPIA** (*ἀμβλυωπία*, dim-sightedness).—Obscurity of vision. See AMAUROSIS.

**AMBULANCE** (*ambulo*, I move about).  
SYNON.: Fr. *Ambulance*; Ger. *Feldlazareth*.

**DEFINITION.**—A term adopted from the French word, which signifies the movable hospital of an army.

The use of an ambulance is to convey from place to place those who are sick, wounded, or otherwise incapable of moving.

**DESCRIPTION.**—An ambulance consists of a *vehicle* or caravan, upon or within which one or more *stretchers* can be laid or fixed.

*Stretchers.*—It is important that stretchers should be of uniform pattern, so as to admit of easy transference from one carriage to another. Each stretcher consists of two poles kept apart by metal traverses, supporting a canvas stretched between. The length of a stretcher (7 ft. 9 in.) is often inconvenient when conveying a patient through passages or up staircases. To obviate this difficulty, the poles of stretchers used for civil ambulances should be made of metal tube capable of being telescoped.

The *vehicles* or carriage-frames are of two kinds: (1) those for *hand ambulances*; and (2) those for *horse ambulances*.

1. *Hand ambulances*, or litters, of which there are several varieties, consist of a skeleton carriage, firmly supported on easy springs, and provided with two, three, or four wheels.

Upon a framework thus formed, the stretcher fits, and is immovably fixed by straps or bolts. The carriage is made with a view to convenience of size and lightness of structure, consistently with strength. Facility in turning should be secured. A complete covering or hood protects the patient from exposure. In all ambulances special care should be taken, by springs and other contrivances, to prevent the suffering caused by jolting.

2. *Horse ambulances* are used for (a) civil, and (b) military purposes.

(a) The first of these, as used in towns, are drawn by one horse. They usually consist of coaches which are unlined, so as to allow of washing and complete disinfection. One or two doors are placed behind, through which a stretcher or bed may slide on to a shelf, occupying about one lateral half of the interior of the coach. A seat for an attendant completes the fittings.

Ambulances for civil purposes should be easily obtainable at hospitals, police-stations, and other recognised centres.

(b) *Military ambulances*, more commonly termed ambulance waggons, consist of a wood-framed body provided with a cover, curtains, and hood of canvas extended on a skeleton-framed roof. They carry likewise a reservoir for water. These ambulances are drawn by two or more horses. In the English army they are arranged to carry two patients on stretchers, and five others less seriously injured, two in front and three behind. In the German army they accommodate four stretchers—two on the floor of the waggon, and two on a plane above—besides five other persons slightly wounded.

*Ambulance Field Hospital.*—Captain Tomkins, of the Victoria Rifles, and the writer have designed an ambulance waggon which supports on its exterior a folding double tent 35 × 22 feet. Twenty folded stretchers, each convertible into a bedstead, one foot high by two feet two inches wide, line the sides of the waggon. There are also provided the requisites for twenty beds, surgical instruments and appliances, a furnace for cooking and for warming the tent in winter, together with rations for three days. This arrangement obviates the necessity for assistance from the main army.

For this Hospital the Empress Augusta Prize and Gold Medal for prompt assistance to the wounded on the field of battle were awarded.

A. T. NORTON.

**AMBULANT** (*ambulo*, I move about). Moving, unfixed, shifting. Applied to diseases that shift from one part to another, such as erysipelas; in connexion with blisters applied in succession to different parts of the body, also called 'flying blisters'; and to moving or 'field' hospitals, as distinguished from fixed or 'base' hospitals.

**AMBULATORY** (*ambulo*, I move about).—A term used in connexion with mild or latent cases of acute diseases, in which the patient continues to walk about, or work, up to a late period of the attack, as in typhoid fever—*typhus ambulans s. ambulatorius*. See TYPHOID FEVER.

**AMELIE-LES-BAINS**, in France (Pyrenees).—Thermal sulphur waters and climatic health-resort. See MINERAL WATERS, and CLIMATE, Treatment of Disease by.

**AMENORRHŒA** (*â*, priv.; *μήν*, a month; and *ῥέω*, I flow).—Absence of the menstrual flow during any portion of the period of life when it ought to be present. See MENSTRUATION, Disorders of.

**AMENORRHŒAL INSANITY**.—See INSANITY.

**AMENTIA** (*â*, priv.; and *μένος*, the mind).—An obsolete term for dementia. See DEMENTIA.

**AMNESIA** (*ἀμνησία*, forgetfulness; Fr. *Amnésie*; Ger. *Gedächtnisswache*).—A general term signifying loss or defect of memory; commonly, however, used in reference to one particular class of defect of memory, viz., that for words (*amnesia verbalis*). Sometimes there is a mere forgetfulness of proper names; sometimes a substitution of wrong words; at other times the pronunciation of a meaningless jargon, not at all representing words. The production of these defects is due to disease in different parts of the brain, owing to the memories of words being threefold—auditory, visual, and kinæsthetic. See APHASIA.

**AMŒBA COLI**.—A species of amœba, found in connexion with ulceration of the colon and with tropical abscess of the liver. See ENTOZOA.

**AMPHORIC**.—A peculiar hollow metallic sound, elicited occasionally by percussion, but more commonly heard in auscultation. Amphoric breath-sound resembles that produced by blowing into an empty glass or metallic vessel with a narrow neck (*amphora*). See PHYSICAL EXAMINATION.

**AMYGDALITIS** (*amygdalæ*, the tonsils).—A synonym for inflammation of the tonsils. See TONSILS, Diseases of.

**AMYLOID DISEASE** (*ἀμυλον*, starch). The name given by Virchow to albuminoid disease, from the belief that the material characteristic of this morbid condition is of

the nature of starch or cellulose. See ALBUMINOID DISEASE.

**AMYOTROPHIC** (*â*, priv.; *μῦς*, a muscle; and *τροφή*, nutrition).—Associated with muscular wasting. See SPINAL CORD, Diseases of.

**ANÆMIA** (*â*, priv.; and *αἷμα*, blood).—SYNON.: Spanæmia; Hydræmia; Oligæmia; Aglobulism. Fr. *Anémie*; Ger. *Anämie*; *Blutarmuth*.

**DEFINITION**.—Deficiency of blood in quantity, either general or local; also, deficiency of the most important constituents of the blood, particularly albuminous substances and red corpuscles.

This definition is purely pathological, and the condition thus expressed presents many varieties, anæmia in the widest sense of the term including oligæmia, oligocythæmia, hydræmia, and spanæmia, as well as chlorosis (see CHLOROSIS, HYDRÆMIA, SPANÆMIA, OLIGOCYTHÆMIA; AND BLOOD, Morbid Conditions of). From the clinical point of view, anæmia is a condition of system in which impoverishment of the blood, whether from want or from waste, is associated with symptoms of imperfect discharge of the vital functions.

**ÆTIOLOGY**.—The causes of anæmia are generally multiple and complex. First, the supply of blood to the body may be insufficient, and that from a variety of causes, of which the chief are:—derangements of alimentation, including insufficient food, constipation, and morbid states of the lymphatic and blood-glands; such defective hygienic conditions affecting the formation and nutrition of the blood as want of light, air, and muscular exercise; prolonged exposure to the influence of certain poisons, as lead, mercury, and malaria; and, lastly, interference with the free circulation of the blood by cardiac or vascular disease, such as valvular disease or dilatation of the heart and aneurysm of the aorta. Secondly, the consumption of blood may be increased by hæmorrhage; by profuse discharges, such as suppuration, catarrh, and albuminuria; by rapid growth and development; by frequent pregnancy and superlactation; by excessive muscular exertion; and by the presence of pyrexia, or of new growths, which rob the system of nutritive material. In a third group of cases of anæmia both the supply and the consumption are at fault. Thus derangement of the organs and of the whole process of sanguification is frequently associated with profuse discharges from various parts; and in acute febrile diseases, malignant diseases, and the 'chronic constitutional diseases,' such as syphilis, tuberculosis, Bright's disease, albuminoid disease, Addison's disease, and others, the cause of the anæmia is extremely complex. But the majority of the cases of anæmia that are

regarded and treated as such, fall into the class to which the name of *idiopathic* has been applied. In such cases the anæmic condition is due, not to any disease so called, but to disturbance of nutrition generally—that is, of the healthy relation between the demands of the system and the supply of nutrient material. This condition occurs chiefly in children and young women, at the period of bodily growth and of the development and early activity of the sexual functions; and when, as so frequently and unfortunately happens, the air, light, food, occupation, and nervous relations of the individual are all more or less unhealthy.

**ANATOMICAL CHARACTERS.**—The blood suffers three principal changes in declared anæmia, namely: (1) deficiency in amount (oligæmia); (2) deficiency in red corpuscles or hæmoglobin (oligocythæmia, aglobulism); and (3) deficiency in albuminous constituents (hypalbuminosis). Of these oligæmia is the simplest, and perhaps never occurs alone; it is speedily complicated with aglobulism, which is a very early and common, as well as the most obstinate, change in the blood. Hypalbuminosis is the most advanced and perhaps the most serious alteration of the three (*see* BLOOD, Morbid Conditions of). The blood is scanty and pale; has a diminished specific gravity; and coagulates slowly and loosely, or in aggravated cases not at all, settling into three layers—consisting respectively of red corpuscles, white corpuscles, and plasma. The body presents certain changes directly due to the state of the blood. Whether the anæmia be local or general, the corresponding parts are blanched and ‘bloodless.’ The cells of the tissues become atrophied and degenerate, in consequence of, and in proportion to, the interference with their plastic and functional activity respectively; and the so-called ‘anæmic’ form of fatty heart, liver, kidneys, and other organs, is the result. If death occur suddenly from acute anæmia, the heart is found empty and contracted.

**PATHOLOGY.**—When the volume of blood in the body has been reduced by repeated small hæmorrhages, the phenomena that supervene, whilst they express the want of blood as a whole, and of its several constituents, are chiefly referable to the loss of two of these constituents—the albuminous substances and the red corpuscles or hæmoglobin, that is, of the oxidisable and the oxidising materials. The pathology of hypalbuminosis and aglobulism is fully discussed in the article on the morbid conditions of the blood, to which the reader is referred. The same effects will be produced by a drain of the liquid part only of the blood, or by poverty of the blood from any of the causes enumerated above, whether of the nature of waste or of want; inasmuch as loss of plasma speedily affects the nutrition of the red cor-

puscles. These phenomena constitute the symptoms of the anæmic condition whatever may be its cause; their relative prominence naturally varying according to an immense number of circumstances.

**SYMPTOMS.**—The subjects of anæmia are usually girls and young women. Their general appearance, which is striking, is one of pallor, debility, and variable loss of feminine fullness. The visible parts of the surface are pallid, often with a tinge of dusky brown on the eyelids and the backs of the hands; the clearness of the complexion varies with the normal pigmentation of the body; the skin is soft, satiny, and rather loose. The mucous surfaces also are blanched; the sclerotic is pearly blue. The loss of flesh may be moderate, or it may be considerable. The extremities are cold, and the legs and lower eyelids are often œdematous. Pyrexia is occasionally present, the temperature rising as high as 102° Fah. or even more. Bodily strength is reduced; muscular force is diminished, while myalgia is common; an air of languor and want of vigour pervades the whole demeanour; and the patient is sleepy, dull, and depressed. The subject of anæmia generally complains of weakness, various pains about the body and head, and marked shortness of breath on the least exertion. The last symptom is unaccompanied by other evidence of respiratory derangement; in character the breathing is regular, and short or even panting. The symptoms referable to the circulation consist chiefly of palpitation on exertion; a tendency to faint; and pain or distress over the cardiac region. The physical signs indicate cardiac enlargement of variable degree. A rough systolic murmur is usually heard over the pulmonary artery; a soft systolic murmur in the aortic base, in the mitral area, possibly over the whole præcordia, and even at the scapular apex; the second sound is often universally accentuated. Over the cervical vessels a systolic murmur is commonly audible, followed by a sharp sound; in the neck a loud venous hum. The cervical vessels may throb; the radial pulse is small, weak, and of variable but usually increased frequency and suddeness. There is a tendency to hæmorrhages, especially epistaxis; and petechiæ are occasionally observed. The digestive system is markedly affected, as shown by loss or perversion of appetite; an anæmic, often bare, but variable tongue; dyspepsia, nausea, and sickness after meals or on rising; and constipation, which is present in the majority of cases, and is frequently prolonged and severe. The menstrual functions are almost always deranged: amenorrhœa is common, in some form; menorrhagia is rare (except as a cause of anæmia); dysmenorrhœa is frequently associated; and leucorrhœa is the rule. The urine varies greatly. Headache, tinnitus aurium, and other cerebral symptoms

are common. There may be swelling of the optic disc. Blood drawn from the finger presents aglobulism. See HEMAGYTOMETER.

**COURSE AND TERMINATIONS.**—The course of anæmia in this form is essentially slow and progressive, unless it is checked; the *duration* is perfectly indefinite. The course of the symptomatic form will naturally vary with its cause. Idiopathic anæmia rarely terminates fatally; and, when it does so, the event may be referred, with few exceptions, to some complication. Occasionally, however, it progresses steadily to death. See ANÆMIA, PERNICIOUS.

Intercurrent diseases may be expected to be severe in an anæmic condition, in proportion to its degree.

**DIAGNOSIS.**—Anæmia is generally recognised with the greatest ease, and the chief question of diagnosis relates to its *cause*. The first point to be determined, therefore, is whether it is not *symptomatic* of some more grave state, such as tuberculosis, syphilis, albuminoid disease, or some other of the many possible causes of poverty of blood. Having settled that the anæmia is *idiopathic*, we must next exclude two diseases with which it may be confounded, namely chlorosis and leukæmia. Chlorosis, in which the plasma is not considered to be altered, and which possesses otherwise a special pathology, is expressed by the yellow tint of skin, by the absence of wasting and of dropsy, as well as by other features (see CHLOROSIS). The diagnosis of pernicious anæmia is fully given in the article on that subject. Leukæmia is recognised by examination of the blood and spleen. The starting-point of the blood-change in idiopathic anæmia can only be discovered by investigation of all the facts of the case.

**PROGNOSIS.**—The prognosis of anæmia is favourable as regards life. In simple anæmia from loss of blood, the patient may be assured of speedy and complete recovery. In idiopathic anæmia, however, this promise can be given only when the cause can be removed or avoided. Under favourable circumstances and judicious treatment, improvement will begin almost immediately; and health should be restored after a few weeks or months.

**TREATMENT.**—The treatment of anæmia, when it is symptomatic of some more grave condition, such as Bright's disease or phthisis, does not require notice here. When blood has been lost in serious quantity, without other injury of consequence, it will be naturally restored if but sufficient time be given, and interference otherwise avoided. Attention to the ordinary rules of health, abundance of food and air, and moderate exercise, will surely, if slowly, restore the patient, without the administration of a single drug. Even in this case, however, treatment may be of great service, by arresting, if necessary, the cause of the anæmia, such as menorrhagia or

epistaxis; and by assisting nature, if the condition should threaten at any time to become intensified by its own effects.

But before the blood can be restored in the large and ill-defined group of cases known as idiopathic anæmia, the unhealthy influences under which the patient is placed, and the functional and other derangements, which are usually accountable for the imperfect sanguification, must be discovered and corrected. Each case must be treated on its own merits, routine being avoided. Where the ætiology is complex, treatment must be equally general, and the whole system of life will have to be reformed. On the other hand, in the rapidly growing child and youth, and still more in girls at puberty, the great demand for nutritive material must be duly considered, and every obstacle to its supply removed. When other than direct discharges are draining the blood they must be checked. Lactation may have to be forbidden; and leucorrhœa and spermatorrhœa will sometimes demand local treatment.

The removal of the cause being thus made the first element in treatment, means must next be adopted for the restoration of the blood. To accomplish this, it will be necessary to bring the alimentary tract and the organs of sanguification into a healthy state. Dyspepsia and constipation require immediate treatment. The best plan is to begin with a brisk purge, and to follow this up with a course of iron and aloes (in various combinations) every night, the pill form being generally preferred, so as to secure one, and only one, daily evacuation of the bowels. Therewith a bitter and alkaline stomachic mixture may be given before meals for a few weeks. The food must be carefully ordered, so that it shall not only supply the albuminous elements that are specially deficient in the blood, but be retained, digested, and absorbed.

The process of sanguification may be successfully assisted by means of drugs. Iron is the sovereign remedy for aglobulism; and, practically speaking, it speedily becomes a question in the medicinal treatment of a case of anæmia in what form iron is to be given. The compound iron mixture of the pharmacopœia, perhaps, answers more frequently than any other; but, on the one hand, when there is constipation, as is so often the case, combinations of aloes and iron in pill, or a mixture containing the protosulphate with purgative saline sulphates, will be more suitable for a time; on the other hand, when there is a tendency to discharges, the persalts with bitters, combined, if necessary, with sulphate of magnesium, will answer the purpose better. Compounds of iron with quinine or strychnine should be given in cases where less marked anæmia occurs in older subjects, with nervous depression and general want of vigour. In special cases the ferrum

redactum, saccharated carbonate, or the vinum ferri (with compound decoction of aloes in constipated subjects), ammonio-citrate of iron, or Blaud's pill may be ordered. Some patients cannot take iron except in the effervescing form; others only if it is presented to them highly diluted—for instance, as a few drops of the tincture of the perchloride in a glass of water. To meet this difficulty, as well as to secure the benefit of many other recuperative influences, a course of chalybeate waters may be the best means that can be devised (*see* MINERAL WATERS). The addition of arsenic to the iron is invaluable in many cases; in other instances arsenic alone, given as Fowler's solution, proves eminently successful after iron has failed. According to some authorities, manganese assists the action of iron. Cod-liver oil is sometimes prescribed with success. Other symptoms must be treated on ordinary principles. Uterine complaints demand special attention; and bromides, ergot, opium, and other sedatives and astringents are indicated where excitement and excessive discharge are present.

Whilst these dietetic and medicinal measures are being carried out, it is impossible to insist too strongly upon attention to bodily and mental hygiene: the use of the tepid morning bath; the proper disposal of time in relation to exercise, education, and amusement; healthy clothing, or rather 'dress'; the avoidance of unwholesome excitement; early hours, and sufficient sleep. In a large number of cases change of air fulfils many of these conditions. On the other hand, in acute anæmia, as well as in severe cases of every kind, rest in bed is imperative for a few days. Time is an essential element in the cure. A frequent change in the form of the medicinal remedies is also advisable.

J. MITCHELL BRUCE.

**ANÆMIA, LYMPHATIC.**—A form of anæmia which is associated with a peculiar affection of the lymphatic system. *See* LYMPHADENOMA.

**ANÆMIA, PERNICIOUS.**—*SYNON.*: Fr. *Anémie pernicieuse progressive*; Ger. *Progressive perniciöse Anämie*.

**DEFINITION.**—A progressive hæmolytic process affecting both sexes, especially in middle life, characterised by fatty degeneration of the heart and retinal hæmorrhages, and in nearly all cases terminating fatally.

Pernicious anæmia includes two varieties: the primary, essential, or 'idiopathic anæmia' of Addison; and the deuteropathic, symptomatic, or secondary anæmia of German authors. The clinical symptoms in the two varieties are, on the whole, alike. Whilst it is possible that pernicious anæmia is really not a disease *per se*, but a group of symptoms, yet for clinical purposes these symptoms are constant enough to constitute a distinct disease.

**ÆTIOLOGY.**—Pernicious anæmia occurs

about equally in men and women, unless we include all cases of fatal or severe anæmia secondary to uterine hæmorrhage. In women the disease is most frequently met with between the ages of 20 and 40 (the period of child-bearing), whereas in men it occurs most frequently between 40 and 60. The youngest age on record is 7, the oldest is 68. Although the disease is more common among country people than town-dwellers, this cannot be ascribed to their respective occupations.

Among the better classes the disease is uncommon; when it does occur, no cause can be ascertained, excepting, in a few instances, mental worry, grief, or fright. It occurs with especial frequency in countries the inhabitants of which are poor and insufficiently supplied with food, particularly nitrogenous food, and who live in small, overcrowded, badly lighted, and ill-ventilated houses. On this account a great number of cases have been recorded by Swiss observers—Biermer, Quincke, and others. Repeated pregnancies, especially in women who are badly nourished or suffer from ordinary bloodlessness, are also a cause of pernicious anæmia. It seldom affects primiparæ. Excessive lactation, severe puerperal or other hæmorrhages, dyspepsia, and the uncontrollable vomiting frequently met with during pregnancy, are considered by many authors among the causes of the secondary form of this disease. Some German authorities include parasites, such as the *Ankylostoma duodenale* and the *Bothriocephalus latus*. Malaria and yellow fever have also been antecedent to this form of anæmia; and Müller has lately called attention to the connexion of constitutional syphilis with this disease.

**ANATOMICAL CHARACTERS.**—The appearances of the organs after death from this disease are the result of the anæmia. The skin may be pale, or presents a lemon-yellow colour. As a rule the body is not emaciated; and on section a thick layer of subcutaneous fat, of canary-yellow colour, is usually found. The muscles are of good colour. The heart and the great vessels contain but little blood, and that of a pale colour and often uncoagulated. The heart itself presents well-marked fatty degeneration, having the characteristic 'tabby cat' appearance. The fatty degeneration affects the ventricles more than the auricles, the left ventricle more than the right, and the muscili papillares more than any other part. This is the most constant of the morbid anatomical conditions. The cavities are sometimes dilated; the valves normal. Flecks of fatty degeneration are commonly seen affecting the intima of the large arteries, and the same change probably occurs in the capillary vessels, explaining perhaps the occurrence of petechiæ and retinal hæmorrhages. Small ecchymoses occur in the brain, serous membranes, and elsewhere. Passive effusions into the serous cavities are not uncommon.

All the organs of the body, with the exception of the spleen, look very pale, as if their vessels had been washed out with water. The liver is generally fatty; not much enlarged. The spleen may be normal, small, or somewhat enlarged, but never attains great dimensions, as in leukæmia. The kidneys are pale, often showing slight fatty change. The mucous membrane of the stomach and intestines often exhibits hæmorrhagic erosions; and atrophy of the tubular glands of the former has been described.

The marrow of the bones has been found normal in the majority of cases, but in some the yellow marrow has been replaced by red—probably a result of the disease rather than the cause of it. This indicates perhaps an effort on the part of this blood-elaborating tissue to take an unusually active part in the formation of red corpuscles, to compensate for their excessive disintegration.

**SYMPTOMS.**—The following are the symptoms which are, as a rule, present in most of the well-marked cases of pernicious anæmia. The patient suffers from weariness, lassitude, headache, vertigo, and dizziness, with faintness, palpitation, and breathlessness on exertion. These symptoms make their approach in so slow and insidious a manner that the patient is unable to fix the date of their onset, and probably will not seek medical advice until he is incapacitated for mental or bodily work.

The aspect of the patient is characteristic: the mucous membranes are pale; the skin is usually of a light lemon-yellow colour, 'like white wax changed by age.' The conjunctivæ are white, though the presence of yellow fat beneath might lead a careless observer to believe there was slight jaundice. The patient is seldom emaciated; indeed the subcutaneous fat may be in considerable quantity. There is often slight œdema of the lower eyelids and of the feet, but these symptoms are generally late in appearing. The intellect is usually unimpaired, but answers to questions are slow, apathetic, and delivered in a low tone of voice, sometimes interrupted by long-drawn sighs. Sleep is occasionally, but not usually, heavy, insomnia being a more constant symptom. The tongue is pale, smooth, and dry; the breath offensive, with a cadaveric odour in severe cases. Stomatitis and bleeding gums are sometimes met with. In many cases dyspepsia, flatulent eructations, vomiting, and a painful sinking feeling at the epigastrium are prominent symptoms. These gastric disturbances are attributed by certain authorities to irritation of the cerebral centres by anæmia, by others to atrophy of the gastric glands. Usually there is anorexia and nausea, but some patients eat well or even to excess. Constipation or diarrhœa may occur, but usually the stools are of normal colour. Dyspnœa is sometimes the

first symptom complained of. The breathing is generally accelerated, and, in advanced cases, is deep and noisy. The laboured breathing increases with the diminution of hæmoglobin, but not so rapidly as other symptoms, and is often absent as long as the patient is at rest. The action of the heart is regular and quick. Sometimes there is visible evidence of dilatation, the impulse being diffused, and the area of præcordial dulness being increased laterally. Systolic murmurs are heard over the cardiac region, sometimes loudest at the base, sometimes at the apex. A venous *bruit* in the neck is generally present. The pulse ranges between 100 and 120, and is regular, compressible, not small, often jerky, and possibly dicrotic.

One of the most striking clinical features of pernicious anæmia is the supervention of hæmorrhages. Epistaxis, menorrhagia, sometimes cerebral hæmorrhage, and towards the close of life petechial hæmorrhages and purpuric eruptions, may occur; but by far the most important are retinal hæmorrhages, because they serve as a diagnostic symptom of the greatest value.

The condition of the optic disc is shown in the accompanying figure (fig. 2). The

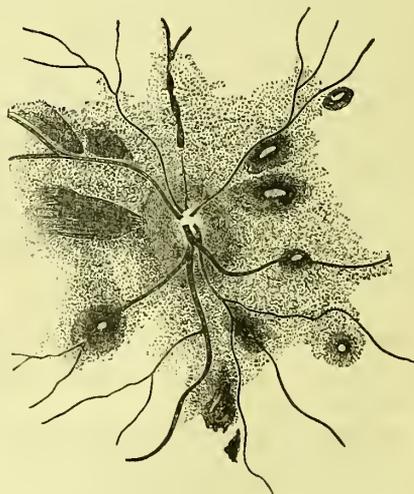


FIG. 2.—Retinal hæmorrhage. After QUINCKE.

fundus is pale, the arteries are narrow, the veins broad; the hæmorrhages are generally multiple, and flame-shaped, from their situation in the layer of nerve-fibres. They appear as linear striæ, rounded spots, or patches, clustered around the optic disc, and may have whitish or yellowish centres, due in part to leucocytes, in part to degeneration. These hæmorrhages may be quickly absorbed, often lasting only a few weeks. There may be no disturbance of vision, unless the hæmorrhage is situated in the maculæ luteæ. In all the six cases observed by the writer retinal hæmorrhages have been present.

More or less pyrexia is the rule in pernicious anæmia. It may be continuous for months, the evening temperature reaching 100°–101° Fah.; or it may be intermittent with sudden exacerbations. Occasionally the temperature may rise to 105° or 106° Fah. On the other hand, it may fall even as much as 10° below normal previous to death.

The state of the urine requires careful consideration. The quantity may be normal, except at the close of life, when it is often considerably diminished. It seldom contains albumin and never sugar. It is often of a deep colour, containing excess of urobilin, and there may be a direct association between the depth of colour and the hæmolytic process. In other instances, however, it is quite pale. The amount of nitrogen excreted *per diem* may be less than normal; but, as a rule, it is more than can be accounted for by the nitrogenous ingesta. Consequently it must be derived from the excessive waste of blood and tissue; but an incomplete oxidation of the nitrogenous substances leads to an excretion of urea and a deposition of fat. Uric acid often occurs in considerable quantities; indican, pathological urobilin, and pentamethylene-diamine have also been found in the urine, suggesting a morbid process in the gastro-intestinal tract. Free iron has been discovered in the urine by Dr. William Hunter.

*Blood.*—Notwithstanding its name, the blood in pernicious anæmia presents no changes absolutely characteristic of the disease. When drawn, it appears pale and watery, and the specific gravity is often greatly reduced. Examined microscopically, it invariably shows a considerable diminution in the number of red corpuscles, which may be reduced to 500,000 per c.mm.— $\frac{1}{10}$  of the normal or even less—in the last stages of the disease. Generally there is a striking variation in the form and size of many of the red corpuscles, as shown in the accompanying figure (fig. 3). It will be seen that some are much larger than normal (macrocytes); these are nucleated. Many, again, are pear-shaped or have an irregular contour (poikilocytes). And, finally, there are to be seen numbers of small, imperfectly developed corpuscles (microcytes). The corpuscles are usually of good colour. There is no leucæmia, but granular masses are common. Hayem holds that the presence of nucleated red corpuscles is a grave omen. There is a great diminution of hæmoglobin in the blood—sometimes in proportion to the falling-off in numbers of the corpuscles; but, owing to many of the corpuscles being more highly charged with colouring matter than normal, the hæmoglobin value of the blood may be excessive in relation to the number of the corpuscles.

The spleen and liver are in some cases enlarged, especially the former. There is also evidence of affection of another impor-

tant structure connected with the elaboration of the blood—namely, the bones. These

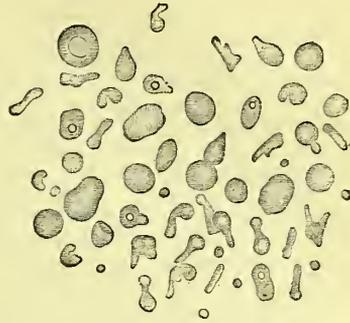


FIG. 3.—Microscopical appearances of the red blood-corpuscles in pernicious anæmia. After QUINCKE. Magnified 750 diameters.

may be tender, and the sternum is especially liable to be so affected.

*COURSE, DURATION, TERMINATIONS, AND PROGNOSIS.*—Of 110 cases collected by Dr. Sidney Coupland, the total duration of the symptoms in 52 was from 1 to 6 months; in 24 from 6 to 12 months; in 25 from 1 to 2 years; in the remaining 12 cases they lasted for periods of over 2 years. Of 130 cases of primary idiopathic anæmia collected by Dr. Pye-Smith, 30 are said to have recovered. Death is usually the result simply of anæmia, and special complications are exceptional. The prostration and weakness increase; the headache, vertigo, and vomiting become more distressing; the breathing becomes hurried, laboured, and deep ('air hunger'); the cardiac palpitation is more marked; and the patient usually falls into a deep, drowsy, lethargic state, either lying impassive, or tossing about in restless delirium, but ceasing to take interest in anything. Occasionally the patient remains conscious to the last, unable to sleep, and feeling utterly miserable and dejected, in consequence of the distressing headache and vomiting. Death may occasionally occur suddenly from syncope.

Some authors dispute the possibility of true pernicious anæmia being cured, and urge that those cases which recover cannot be considered 'pernicious.' The prognosis is always grave, if not absolutely bad; and even when a patient improves under treatment, and the condition of the blood would give hopes of a cure, a guarded opinion should always be given; for a fresh exacerbation of pyrexia, with hæmolysis, and a return of the symptoms in an aggravated form, generally occur without any apparent cause.

*PATHOLOGY.*—The term 'idiopathic anæmia' in our medical nomenclature is an indication that our pathological knowledge of this disease is still involved in obscurity. The examination of the blood and urine during

life, taken in conjunction with changes found in certain organs and tissues after death, has thrown some light upon the disease. In health there is a constant equilibration between hæmogenesis and hæmolysis. The pigment of the urine—urobilin, and the pigment of the fæces—stercobilin, are both iron-free derivatives of hæmoglobin. It was first shown by various observers that the urine of pernicious anæmia contains an excess of *urobilin*, and the writer has been able to confirm this in two cases. Quincke has shown that there is an excess of free iron in the liver, and to a less degree in the pancreas and kidney. These organs sometimes appear slaty-black at the necropsy, from the action of sulphuretted hydrogen upon the free iron. If sections are placed in solution of ferrocyanide of potassium acidulated with hydrochloric acid, they turn blue, owing to the formation of Prussian blue. The following is an analysis made for the writer by Mr. Vasey, F.C.S., showing the amount of free iron contained in the organs.

Organ	Weight in grammes	Percentage of ferric oxide in organ	Total ferric oxide in the whole organ	Total ash in the whole organ	Percentage of ferric oxide in whole organ
Anæmic liver .	1240	.29	3.60	1.05	27.20
"    kidney	282	Traces	—	1.25	—
"    spleen	141	Traces	—	.90	—
Normal liver .	—	Traces; along with distinct traces of copper	—	1.16	—

It has been said that this is merely the iron which has been administered medicinally, but the accompanying wood-cut (fig. 4) re-

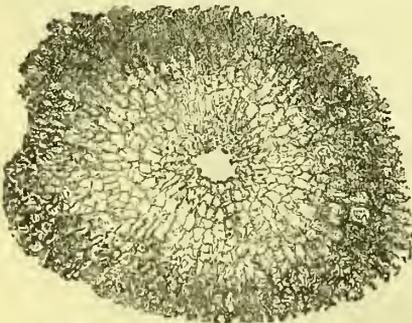


FIG. 4.—Section of the liver after treatment with ferrocyanide of potassium solution and hydrochloric acid. The dark shading indicates the deposit of iron in the portal zone. Magnified 75 diameters.

presents a section of the liver from a patient who had not taken iron for six months.

Although no specific organisms have been found in the blood or organs in this disease,

yet many of the symptoms—viz. the gastrointestinal disturbances, the peculiar cadaveric odour of the breath, the irregular attacks of pyrexia, and the presence of indican and other bodies in the urine—suggest the absorption by the portal circulation of some poison connected perhaps with the growth of micro-organisms. It has been shown that the blood in pernicious anæmia, as in many septic diseases, after being drawn, allows hæmoglobin crystals to form spontaneously. When arsenic is administered, the blood is said to lose this property, suggesting an explanation of the value of the drug in the treatment of the disease. Again, if putrid serum be added to healthy blood, crystals of hæmoglobin form. It is possible therefore that some septic poison sets up a hæmolytic process in the portal circulation, and that the hæmoglobin is broken up in the liver—into urobilin, which escapes by the urine, and free iron, which remains in the cells and capillaries of the portal zone. The urobilin, which passes from the hepatic veins into the general circulation, gives rise before its excretion to the lemon-yellow colour of the skin and fat (urobilin jaundice).

Dr. Delépine considers that the liver possesses a ferrogenic function, and suggests that in pernicious anæmia there may be an exaggeration of this physiological process. It would thus be analogous to diabetes in relation to the glycogenic function of the same organ.

It is necessary to add that some authors do not believe this to be a disease *per se*, but only a grave form of chlorosis or a pseudo-leukæmia of the myelogenic variety.

DIAGNOSIS.—The principal diagnostic features of pernicious anæmia are the following: (1) the patient is *middle-aged*; and, except in the case of women, there is nothing to account for the onset of the disease; (2) the absence of any organic disease; (3) the severe and progressive anæmia; (4) the great diminution of the corpuscles and colouring matter of the blood; (5) alteration in the shape and size of the corpuscles; (6) signs of fatty degeneration of the heart, without arterial degeneration or valvular disease; (7) retinal hæmorrhages; (8) irregular and occasional pyrexia; (9) no emaciation, but frequently rather an excess of subcutaneous fat; (10) lemon-yellow coloured skin and high-coloured urine.

The diseases for which pernicious anæmia has been mistaken are Bright's disease, ulcerative endocarditis, tubercnlosis, Addison's disease, cirrhosis of the liver, cancer of the stomach and malignant disease of other internal organs, chronic ulcer of the stomach, parasitic affections, such as ankylostoma duodenale, and pseudo-leukæmia myelogenica. It ought not to be confused with simple idiopathic anæmia or chlorosis, as it occurs at an age when these diseases are less likely to be met with.

**TREATMENT.**—Iron preparations are absolutely useless in pernicious anæmia, and cases have been recorded in which they have proved injurious. Dr. Byrom Bramwell introduced the employment of arsenic, and most of the recorded cases of recovery have been treated with this drug. Although it is not a specific, it does good in nearly all cases, for if it does not cure the patient it generally prolongs life. *Liquor arsenicalis* should be given in 3-minim doses, rapidly increased to 10 minims, three times a day; and if it cannot be tolerated, on account of gastric disturbances, it may (according to Hayem) be administered subcutaneously. Hayem also recommends the inhalation of 15 litres of oxygen a quarter of an hour before the two principal meals; and after meals a one per cent. solution of hydrochloric acid. It is necessary to give a nutritious diet, very easy of digestion, such as milk, beef-tea, eggs, scraped raw beef and meat-juice. If vomiting be very severe, nutrient enemata may be used. Transfusion has been tried in some cases, but it is doubtful whether it has ever done more than prolong life for a short time. Defibrinated human blood should be used, and not more than half a pint should be injected at once.

FREDERICK W. MOTT.

**ANÆSTHESIA** (*ἀ, priv., and ἄισθάνομαι, I feel*).—Anæsthesia literally means absence or loss of sensation, which may be general or local. The word is, however, more especially employed to signify loss of tactile sensibility, as distinguished from insensibility to pain, or *analgesia*. It is further used to indicate the condition induced by the action of anæsthetics upon the system. See ANÆSTHETICS; and SENSATION, Disorders of.

**ANÆSTHETICS.**—**DEFINITION.**—The name given to a series of agents which are employed for the prevention of pain, but more especially applied to those used in surgical practice.

**HISTORY.**—The idea of annulling pain in surgical operations is a very old one. Compression of the nerves and blood-vessels, and the inhalation of the vapour of carbonic anhydride and mixtures containing it, were practised at an early date. In the sixteenth century ether was probably the active ingredient of a volatile anæsthetic described by Porta. The use of anæsthetics was, however, little understood and rarely practised. Even the suggestion of Sir Humphry Davy, that nitrous oxide should be used in minor operations not attended with loss of blood, was of little practical value, on account of the inefficient apparatus then available. In 1845 Horace Wells inhaled laughing-gas so successfully that he may be said to have introduced the practice; but he appears to have so often failed to produce the desired effect that this agent fell into disuse on the introduction of ether in 1846 by Morton, after

some communications on its properties from a chemist named Jackson. In 1847 chloroform was used by Simpson, and quickly superseded ether almost all over Europe. At the present time the comparative safety of ether has caused this anæsthetic again to be preferred by many surgeons in this country.

**ENUMERATION.**—The three agents just mentioned—namely, nitrous oxide, ether, and chloroform—are those chiefly in use, and they have each advantages in particular cases. Experiments made with other agents, such as amylene, tetrachloride of carbon, ethidene-dichloride, and bichloride of methylene, have not shown that they possess sufficient advantages to counterbalance the defect of requiring special management in their administration. This list of anæsthetics might be still further increased, for in order to produce insensibility it is only necessary to reduce the supply of arterialised blood to the nervous centres, or to introduce into the blood a substance which deprives it of its power of oxygenating the tissues.

**MODES OF USE.**—Anæsthesia may be produced for surgical purposes:—(1) by numbing the part to be operated on by means of cold; (2) by intercepting its nervous communication; (3) by arresting the activity of the nervous centres concerned in sensation. Thus anæsthetics may be *local* or *general* in their action.

**Local Anæsthesia** may be induced by cold. The most convenient plan is to blow a jet of anhydrous ether spray upon the part, as suggested by Dr. Richardson, and thus to freeze it. The surface to be frozen should be dry, and hence the difficulty of freezing the gum of the lower jaw, on account of the saliva. A mixture of equal parts of pounded ice and common salt contained in a bag of muslin is effective, but less easily applied. This plan is adapted for opening abscesses and boils, and for the extraction of a few teeth; but the process both of congelation and of thaw is painful. Chloroform applied locally is said to cause numbness, but it is very little used except inside the mouth, and then it owes its soothing effects to the quantity of chloroform vapour which is inhaled. Compression of nerve-trunks for inducing anæsthesia is never practised at the present day. Hydrochlorate of cocaine in a 4 per cent. solution, applied either by pipette, camel's-hair brush, or spray, is largely used in operations on the eye, naso-pharynx, and other mucous membranes. Its use hypodermically requires great caution, as its constitutional action is still imperfectly known. In neuralgia it should be applied in the vicinity of the nerve affected.

**General Anæsthesia** is at present rarely obtained in any other way than by inhalation, although successful attempts have been made to induce the condition by subcutaneous

and intravenous injection of chloral or morphine.

**SUBJECTS FOR ANÆSTHETICS.**—We may say generally that any person fit for a severe operation is a fit subject for an anæsthetic; but no one is so free from danger that care in watching its effects can be dispensed with. The cases requiring the greatest vigilance are not the young and delicate, for whom a small dose suffices, but the strong, who inhale deeply and struggle much. Ether is probably better adapted than chloroform for those suspected to suffer from fatty degeneration of the heart. Many of the deaths under chloroform have occurred in intemperate drinkers, and the presence of alcohol in the system undoubtedly intensifies its effect.

**PRECAUTIONS.**—Before commencing inhalation the following particulars should be attended to. The patient must not have recently taken a full meal; he should lie comfortably, in a horizontal position if possible, unless when gas or ether is given for a short operation; and the dress should not be tight. When the administration is begun, he should be encouraged to breathe regularly and freely. The pulse as well as the respiration must be watched. If the vapour excites either swallowing or coughing, it is more pungent than is requisite, and its strength should be diminished. Most patients are at first afraid of breathing, and some hold their breath for half a minute. The vapour should not be removed on this account, but care should be taken, by holding the inhaling apparatus farther off, to prevent the vapour becoming too strong in the interval. After volition has been abolished, any pause in the breathing should be noted, and more or less fresh air given. Further directions will be given in describing the anæsthetics specially.

**SPECIAL ANÆSTHETICS.**—**Nitrous Oxide.** Protoxide of nitrogen, nitrous oxide, or laughing gas is now prepared wholesale, and sold condensed into a liquid in strong iron bottles. The gas, whether supplied thus or from a gasometer, should be inhaled from a bag having such free communication with the face that it will readily be supplied even in panting respiration. A long tube, however large, is objectionable, as the gas is less mobile than common air.

The special aim in giving gas should at first be to exclude air, and to exchange the atmosphere within the air-passages and lungs for one of pure gas. The patient should not merely be told to breathe slowly and deeply, but be shown how to do so, about fifteen times in a minute. The inspiration should not be jerking, and the expiration should be complete. It is a special merit of laughing gas that no harm can come of inhaling too freely at first. The gas-bag must be kept filled either by pressure on the gasometer, or by turning the screw tap of the gas-bottle. This may be

done by the hand of an assistant, or more conveniently with the foot of the administrator. The writers' plan is to fix the bottle vertically and turn the screw by pressing the foot against an iron plate with spikes on its upper surface, and a square hole fitting the tap on its lower surface. It is imperative that the face-piece or mouth-piece *should fit accurately*, and the air-pad is almost essential to effect this in a great many cases. It should be warmed if the india-rubber is stiff. After five or six good respirations there is no need to supply fresh gas with each inspiration. The expiratory valve should be kept closed, and the inspiratory valve opened. Care must always be taken that the supply of gas is sufficient to replace any that is lost by absorption into the blood or by leakage. This is more easily effected if the gas-bag is made of thin india-rubber, so as to distend easily and contract gently with the movement of breathing. It should be sufficiently filled, so that if the mouth-piece does not fit, the gas would escape instead of air entering and becoming mixed with it.

Lividity of the skin will not help us to know when the patient is fully under the influence of the gas, neither will insensibility of the eyelids, nor yet the state of the pupils. The breathing should become stertorous or interrupted, or the pulse very feeble, or convulsive twitchings should occur, before the face-piece is removed. A little air may be admitted by raising the face-piece, if the operation is not upon the face, and by doing so every fourth or fifth respiration anæsthesia may be kept up for several minutes. The effect of a single full inspiration may be to bring the patient into a state of excitement, and the continuance of the gas *without* air brings on convulsive movements, so that it is not well adapted for any operation lasting as much as five minutes, and requiring steadiness.

Patients are sometimes so unsteady that it is found to be almost impossible to make the face-piece fit. In such cases the best plan is to cover the patient's eyes and let him breathe air, merely preventing him from rising from the chair or bed, and not speaking till he is conscious, and as soon as he becomes so to recommence the inhaling as if nothing had happened. A violent patient often becomes perfectly rational in less than two minutes.

Sickness and headache ought not to result from the inhalation of gas; but if the use of it is prolonged, or if the patient is kept for several minutes in a semi-conscious state, breathing a little air with the gas, both these symptoms may occur. The recumbent posture, quiet, and warmth to the feet, constitute all that is likely to be required in the way of treatment.

**Ether.**—Ether, sulphuric ether, ethylic ether, vinic ether, or oxide of ethyl, was first used for anæsthetic purposes in 1846. Before its use was well understood in England,

chloroform was brought forward as a more convenient agent, and much less unpleasant to the patient. Ether is less liable to become dangerous to life, as it does not, under ordinary circumstances, depress the action of the heart. The best kind is the *Æther Purus* of the Pharmacopœia, of sp. gr. 720.

Although it is not difficult to destroy dogs suddenly with ether, it is believed by many writers on the subject that in man it can only prove fatal by causing asphyxia, and that the signs of this condition are so easily seen and remedied that practically this anæsthetic is quite safe. The writers are not of this opinion, believing that sometimes when narcosis is far advanced, the glottis will allow ether vapour to pass of sufficient strength to stop the heart. Such cases, however, are very rare indeed. Ether is extensively administered by pouring an ounce at a time upon a very large cup-shaped sponge, which, if cold from previous use, is dipped into hot water and squeezed as dry as possible. It is to be expected that the patient will resist breathing when this is held over his face, but after a minute's struggling he becomes unconscious, and easily managed. Compared with the method of giving ether timidly, so as to let the patient remain delirious for several minutes, this may be a good plan; but there is no necessity for giving the ether so strongly if *we diminish the access of fresh air*. The administration of nitrous oxide from which air is at first excluded, and to which it is afterwards admitted very sparingly, has taught us how slight the after-effects are from the asphyxia so induced. Cones of leather or pasteboard lined with felt, and having a small opening at the apex, are better than sponges; but they should be larger. They may be made more effective and economical by placing a thin india-rubber bag over the apex of the cone, so that more of the expired atmosphere may be breathed again.

Morgan's inhaler is very efficient. The ether is poured into a tin chamber as large as a hat, containing sponge. This is covered by a sort of diaphragm, which rises and falls with respiration as the patient breathes into and out of it by means of a tube and face-piece. There are no valves. Anæsthesia results partly from asphyxia, and partly from the action of the ether. The amount breathed depends on its temperature, and on the freedom of respiration. If the respiratory movements are slight, as in young children or in persons suffering from emphysema, the amount of ether supplied is apt to be too small. Ormsby's inhaler is an improvement upon Morgan's. The sponge for ether is contained in a cage near the face-piece.

With the view of regulating the strength of the ether vapour, the late Mr. Clover contrived the following apparatus, which is made by Mayer and Meltzer. It consists of an oval

india-rubber bag fifteen inches long, at the one end connected with the face-piece, at the other with the ether-vessel. Within the bag is a flexible tube also leading from the face-piece to the ether-vessel. By turning a regulator the patient is made to breathe into the bag either directly or indirectly through the tube and ether-vessel, or partly one way and partly the other. The more the regulator is turned towards the letter E, the more ether-vapour he takes. By turning it back again the amount of vapour is diminished. The ether-vessel contains a reservoir of water, which prevents the ether becoming too cold from evaporation. When in use, it may be conveniently suspended by a strap from the neck of the administrator.

At first the regulator allows the passage from the face-piece into the bag to remain open, and the bag should be filled by pressing the face-piece more firmly against the face during expiration than inspiration. By degrees the regulator is turned towards the letter E, and thus the passage to the inner tube is opened, and the air breathed through it carries ether vapour from the vessel into the distal end of the bag. When the regulator allows half the inspired air to pass through the ether, the vapour is strong enough to induce sleep in two minutes, usually without exciting cough. As the act of swallowing is excited by a smaller quantity of ether than that of coughing, it should be watched for, and the regulator very slightly turned back should it occur.

This same apparatus may be used for giving laughing-gas, all communication with the ether-vessel being cut off by turning a stopcock, and by attaching the tube leading from the gas-bottle to a mount near the bag.

By far the least unpleasant and the quickest way of preparing a patient for a surgical operation is to use gas and ether combined; the change from gas to ether being made by turning the regulator above described as soon as the patient is sufficiently under the influence of the gas to disregard the flavour of ether. The supply of gas should be stopped as soon as the ether is introduced; but if subsequently the patient is allowed to become conscious, the gas may be given freely as at first, in order to cause him to sleep again. The writers have found less sickness and more rapid recovery from the unpleasant taste of ether than when the latter is given alone. The chief difficulty is to prevent the unsteadiness of the patient, which results from the panting character of the breathing. To lessen this, the ether must be given as strong as possible without producing irritation of the throat, and the operator should wait until the influence of the ether has increased to the production of deep stertor. Air being then admitted with every fourth or fifth inspiration, the breathing soon becomes as regular as it is under ether when given in any other way.

On recovering from the inhalation of ether, patients are often in a state of intoxication for a period corresponding to the time and extent of the etherisation. The eyes should be covered, but the mouth and nose left free; and the room should be kept quiet, with a brisk fire, and the window more or less open.

**Chloroform.**—Chloroform was brought forward by Simpson in 1847. It should not be made from methylated spirit; and, when a drachm is poured upon blotting-paper, it should evaporate without leaving an unpleasant odour. It is the most convenient of all anæsthetics, and the most easy to administer. Unfortunately, when given beyond a certain strength, it has a tendency to produce cardiac syncope or asphyxia, and it is not improbable that some persons are particularly liable to be affected.

Some authorities think it desirable to give alcoholic stimulants before administering chloroform; others partially narcotise the patient with morphine or chloral. No doubt these agents assist the action of the chloroform, but if from any accident an excess of chloroform should be given, they interfere with the means of recovery, and for this reason are not to be recommended. There is less objection to the inhalation of a mixture of chloroform and ether, or of these agents with alcohol; but such mixtures, if kept for some length of time, alter their relative proportions, owing to the escape of the more volatile ingredients. Even the change from the administration of chloroform to that of ether, if made suddenly, is not free from danger, for, when a person is partially under the influence of chloroform, the glottis allows a high percentage of ether to pass; and, if the lung-circulation be slow, as is likely to be the case, the blood may be so highly charged with ether as to depress rather than stimulate the heart.

Chloroform, therefore, should be given gradually. The object should be to keep down the proportion of chloroform-vapour rather than to give abundance of fresh air. In preparing for an operation requiring perfect stillness, six to eight minutes should be allowed for the process. Sponges or lint saturated with chloroform, and held close to the mouth, are dangerous, from the possibility of liquid chloroform falling on the lips or into the mouth. In midwifery practice a piece of linen or blotting-paper, sprinkled with chloroform and placed at the bottom of a tumbler, is a convenient plan of administration, care being taken to prevent any liquid chloroform from settling at the bottom. In general surgery a handkerchief or towel may be folded into a small cone, open at the apex, into which not more than a drachm should be poured at first, and fifteen minims at a time afterwards. A better plan is to roll and tie a piece of lint into a compress the size of a walnut. A drachm to a drachm and a-half

of chloroform should be poured upon this, which is to be held about an inch in front of the patient's upper lip, the hand and compress being covered with a towel, which should gradually be drawn over the patient's face. This plan gives considerable command over the supply of chloroform, for, when the chloroform, having cooled by evaporation, is given off too slowly, the vapour can be increased by warming the compress in the palm of the hand. When it is becoming dry it ceases to feel cold, and warning is thus given that fresh chloroform is needed. This should be supplied half a drachm at a time. In doing this the towel should still be left over the face of the patient, in order to keep him breathing a slightly chloroformed atmosphere. The movement of swallowing should be looked for, and regarded as evidence that the vapour is stronger than is necessary. If any sound like hicough indicates laryngeal obstruction, the chloroform and towel should be removed, and, if the sound continue, the chin should be raised as much as possible from the sternum. Laryngeal obstruction arises from two main causes, viz., spasm of the glottis, and falling down of the epiglottis. The first is excited by the pungency of the vapour, and also by reflex action when certain nerves are injured, notably when a ligature is tightened upon a pile. The epiglottis covers the larynx every time we swallow, but the muscles coming from the chin raise it again directly. In deep narcosis these muscles are sluggish, and cannot act thus if the position of the chin places them at a disadvantage. If raising the chin fail to open the air-passage, the tongue must be pulled forward. In doing this the head should be kept back. Depressing the chin renders a partial obstruction complete. A laryngeal sound indicating obstruction is of little consequence if the pulse is good, since, although the breathing be imperfect, sufficient fresh air continues to be breathed; but if the pulse flags, or if it appears that the amount of chloroform in the air-passages is excessive, not a moment should be lost in seizing the tongue with forceps.

When a patient is delirious and struggling, extra care must be taken that the chloroform be not too strong, because he inhales deeply, carrying the vapour almost to the air-cells of the lungs, and, when he next closes the glottis and strains, the pressure of the air and vapour within the lungs is increased, and the chloroform enters the blood very quickly. The compress should be held at least two inches off the mouth, although the towel may still cover the face. Directly any stertorous noise is heard, a breath or two of fresh air should be allowed, and no more chloroform given till the pulse beats well and the respiration is free.

Skinner's apparatus—a cap of 'domette' flannel stretched over a frame—is a much

better apparatus than a towel; but its virtue is simplicity, and it has little pretension to exactitude. Snow's apparatus is very efficient, portable, and economical. The addition of a water-jacket to equalise the temperature was a great improvement; but it should be provided with a thermometer.

The safest and least unpleasant mode of giving chloroform is by means of the apparatus fitted with a large bag of air containing not more than thirty-three minims of chloroform in a thousand inches of air. The apparatus is, however, too complex to be generally adopted. The advantage of more precise measurement of the strength of chloroform vapour than is afforded by towels or napkins will appear when we consider the several circumstances that alter it when so given.

The *strength of vapour* given off from a known quantity of chloroform is influenced by:—

1. The extent of surface of chloroform.
2. The temperature of the chloroform, which is constantly changing.
3. The temperature of the air of the room, of the patient's face, and of the administrator's hand.
4. The distance at which the chloroform is held from the patient's face.
5. The rapidity of the current of air.
6. The height of the barometer.

Moreover, when the chloroform mixture is of *known* strength, its effect is increased by high barometrical pressure; by low temperature of the blood; by deep or quick respiration, and especially by muscular efforts when the glottis is closed; and by slow movement of the blood through the lungs.

On the other hand, it is lessened by low barometrical pressure; by high temperature of blood; by superficial or slow respiration; and by rapid circulation through the lungs.

Under ordinary circumstances danger from these causes is easily averted with moderate care, for they do not often concur to produce the same effect. But if a patient, fatigued with struggling, takes a very deep breath just as fresh chloroform has been poured upon the towel, and then closes his glottis and makes another struggle—the barometer being high at the same time—it is evident that blood unduly charged with chloroform will gain access into the coronary arteries, and depress the cardiac ganglia. Death has occurred so rapidly under these circumstances, that it has been thought to be the result of shock from the operation.

Chloroform lessens, if it does not entirely prevent, the shock of an operation; but it is to be feared that, if chloroform be given freely for this purpose, a dangerous amount of it may be administered.

If a severe operation is about to be performed, the chloroform should be given in the same gradual manner as in a slighter

one, but continued to the point of fixing the pupils and producing stertorous breathing; and, when the chief shock is expected, two or three breaths of pure air should be admitted, so that, if the pulse fail, there may not be an excessive amount of chloroform-vapour in the lungs.

*Compounds of Chloroform.*—Under this head comes *Methylene* (*Bichloride of Methylene*), which contains a variable quantity of chloroform. Its chemical characters and physiological effects are similar to those of a mixture of chloroform, ether, and alcohol. It narcotises quickly, but not safely; and, as the amount of chloroform in it is not always uniform, it is better to mix, in small quantities at a time, one part of alcohol, two of chloroform, and three of ether, and to keep the bottle so well corked that the ether is not likely to evaporate and leave chloroform in excess. The word ACE fixes the proportions in one's memory.

A mixture of one part of chloroform with four of ether is convenient for a brief operation, as this produces much less excitement than ether alone. On the whole the writers object to keeping mixtures of this kind ready-made; and it is probable that the plan of giving at the outset sufficient chloroform to abolish consciousness, and subsequently administering ether, will be found safer than mixing them together in the liquid state.

**Bromide of Ethyl** ( $C_2H_5Br$ ).—This substance has been recommended and moderately used as an anæsthetic. Its merits are that it exhilarates and produces rapid anæsthesia. Its effects pass off quickly, without any tendency to depress the action of the heart. The objections to the use of bromide of ethyl are that it is liable to decompose, leaves a strong smell and taste of bromine, and sometimes produces headache and sickness. Two deaths from it in America have been reported. Its use has been abandoned in England.

**Dichloride of Ethidene** ( $C_2H_4Cl_2$ ).—This substance is obtained by distilling aldehyde and pentachloride of phosphorus. The effects of this agent are intermediate between those of chloroform and ether. Its odour is usually preferred to that of either of the former. The writers have used it very extensively; and in minor cases, in which the operation has lasted only five minutes, the recovery is rarely attended by sickness or excitement. In the more prolonged cases it was found to cause vomiting, but this ceased much more rapidly than when chloroform had been given. Its effect upon the heart when given in large doses is depressing, and the pulse should be carefully watched during its administration.

**AFTER-TREATMENT.**—Quietude, or conversation of an encouraging or soothing character, is desirable during the half-minute of recovery from gas. The eyes should be covered, unless

the view is tranquil as well as pleasant. If gas be given until there are intermissions in the breathing, or its administration continued for several minutes with a small allowance of air, there may be headache and even vomiting: still no other treatment than repose is needed.

After the inhalation of ether a taste will remain. This may be got rid of by washing out the mouth, and gargling with warm fluids; while the vapour remaining about the patient and in the room may be removed by heating the surface by means of hot bottles, and making a bright fire.

When the system has been long or profoundly under the influence of chloroform or ether, nausea and vomiting are likely to ensue. The writers have found warmth, fresh air, and abstinence from food efficient in relieving these symptoms. Hot tea and coffee, taken from a feeder without raising the head, and afterwards beef-tea and jelly, are sufficient for twenty-four hours, unless the patient wishes for something solid. The rule then should be to give little, or less than asked for. Ice relieves thirst, and serves to postpone the necessity for giving solids.

**TREATMENT OF DANGEROUS SYMPTOMS.**—Anæsthetics in excess destroy life by stopping the action of the heart, or the respiration; generally both are affected.<sup>1</sup> When laughing-gas is given to animals till the breathing has ceased, the heart continues to beat long afterwards, and artificial respiration rapidly restores them. Ether-vapour, given almost pure through a tracheal tube, will arrest the action of a dog's heart in sixteen seconds; but if it be administered as rapidly as possible with a cloth, without opening the trachea, the breathing fails before the heart, and the hæmodynamometer shows adequate pressure in the vessels whilst the breath is gasping, and for several seconds after it has ceased.

With chloroform the hæmodynamometer indicates diminished pressure directly the animal ceases to struggle, and the heart sometimes stops before the breathing. In case alarming symptoms should arise, the first effort should be directed to lessening the amount of the anæsthetic in the lungs, by pressing the trunk with both hands, and squeezing out as much air as possible without causing a shock. If, after this has been done two or three times, the air does not readily re-enter the chest, the obstruction is to be overcome either by lifting the chin or drawing out the tongue; and other artificial movements of the chest must be carried on (*see* ARTIFICIAL RESPIRATION). If pallor be noticed whilst breathing is going on, the recumbent posture and elevation of the feet

are immediately required (*see* RESUSCITATION). Nelaton's plan of inverting the body has often been followed by recovery; but, considering the impediment to inspiration from the weight of the abdominal viscera, the writers are of opinion that the pelvis should never be many inches higher than the head. Nitrite of amyl—by reason of its effect in dilating the vessels of the skin—has been recommended, but without careful physiological inquiry, and upon very small clinical experience. Hypodermic injection of ether is also useful.

Electricity might be expected to prove the best agent to assist the action of a feeble heart. Mr. Clover's experimental observations were not favourable to its employment; and certainly artificial respiration should not be delayed one moment in order to apply electricity.

Insufflation is not to be depended on. The condition would be rendered worse by distension of the stomach, which cannot always be prevented by pressing the larynx against the spine.

Laryngotomy may be required in cases where air cannot be made to enter the chest, in spite of throwing the head backward, and removing the chin away from the sternum.

Hot-water injections may be of use, but there can be no necessity for brandy whilst artificial breathing is being carried on. Afterwards, if swallowing is difficult, brandy may be added to the enema.

Friction of the limbs in the direction of the heart is unnecessary, provided the feet are slightly raised. Where there has been great loss of blood, the limbs should be bandaged firmly, from the fingers and toes upward, as in Esmarch's plan for saving the blood of a limb about to be amputated. In warm weather, or if the body is warm, a towel dipped in cold water may be flapped against the chest, but harm would result from cooling the body generally. Bottles of hot water and hot blankets should be applied as soon as the breathing is restored, and a brisk fire should be kept up, in order to favour the ventilation of the chamber.

J. T. CLOVER.

G. H. BAILEY.

**ANALGESIA** (ἀ, priv.; and ἄλγος, pain).—Absence of sensibility to painful impressions. *See* SENSATION, Disorders of.

**ANAPHRODISIA** (ἀ, priv.; and Ἀφροδίτη, Venus).—Absence of sexual appetite. Sometimes used to express impotence. *See* IMPOTENCE; and SEXUAL FUNCTIONS IN THE FEMALE, Disorders of.

**ANAPHRODISIACS.**—DEFINITION.—Medicines which diminish the sexual passion.

ENUMERATION.—The agents employed as anaphrodisiacs are:—Ice, Cold Baths—local and general; Bromide of Potassium and Ammonium; Iodide of Potassium; Conium;

<sup>1</sup> On this subject reference should be made to 'An Address on Experiments on Anæsthetics,' by Dr. Lauder Brunton, *Trans. Medical Soc. of London*, vol. xiii. p. 261, and *Brit. Med. Journ.*, 1890, ii. p. 531, for report of experiments by Dr. MacWilliam.

Camphor; Digitalis; Purgatives; Nauseants; and Bleeding.

**ACTION.**—The erection which occurs in the genital organs during functional activity is due to dilatation of the arteries in the erectile tissues, and is regulated by a nervous centre situated in the lumbar portion of the spinal cord. From this centre vaso-inhibitory nerves pass to these arteries, and cause them to dilate whenever it is called into action. It may be excited reflexly by stimulation of the sensory nerves of the genital organs and adjoining parts, or by stimuli from the alimentary canal. It may also be excited by psychical stimuli passing to it from the brain. Some anaphrodisiacs may act by lessening the excitability of the nerves of the genital organs, as the continuous application of cold, and probably, also, bromide of potassium; some by diminishing the excitability of the genital centres in the spinal cord and brain, as bromide and iodide of potassium, and conium; others by influencing the circulation, as digitalis. There are also adjuvant measures, of a hygienic and moral character, which greatly assist and may even replace anaphrodisiac medicines, such as a meagre diet, especially of a vegetable nature, the avoidance of stimulants, and the pursuit of active mental and bodily exercise. Everything tending to stimulate the genital organs, or to increase the flow of blood to them or to the lumbar portion of the spinal cord, should be avoided, such as warm and heavy clothing, or pads about the hips or loins; and a hard mattress should be used in place of a feather-bed. Everything likely to arouse the passions, such as certain novels, pictures, theatrical representations, &c., should also be shunned.

**USES.**—Anaphrodisiacs are employed to lessen the sexual passions when these are abnormally excited in satyriasis, nymphomania, and allied conditions. As such excitement may sometimes depend on local irritation of the genitals, in consequence of prurigo of the external organs, excoriations of the os uteri, or balanitis; or on the presence of worms in the rectum or vagina; these sources of excitement should be looked for, and, if present, should be subjected to appropriate treatment.

T. LAUDER BRUNTON.

**ANARTHRIA** (*án, neg.*; and *ἀρθρία*, the limbs).—**SYNON.**: Fr. *Anarthrie*; Ger. *Gliedlosigkeit*.—A term applied to those defects of speech (not strictly aphasia) which consist in a merely blurred, or more or less unintelligible articulation. The patient does not suffer from forgetfulness of words, but from a difficulty in their utterance. See **APHASIA**.

**ANASARCA** (*áná, through*; and *σάρξ*, the flesh).—An effusion of serous fluid into the subcutaneous connective tissues, not limited to a particular locality, but becoming more or less diffused. See **DROPSY**.

**ANEURYSM** (*ἀνερίνω, I dilate*).—**DEFINITION.**—Aneurysm is a local dilatation of an artery, leading to the formation of a tumour which contains blood, and the walls of which are composed either of the tissues of the vessel, or those which form its sheath, or immediately surround it. Therefore every aneurysm, properly so called, consists of two parts—a *sac* and its *contents*.

**CLASSIFICATION.**—Aneurysms are usually divided, according to the varying composition of the sac, into the following varieties:—

1. *True* aneurysm, in which all the three coats of the artery form the sac or a portion of the sac. This variety is rare: at least it is so rarely possible to trace all the coats of the artery over any part of the sac beyond its orifice, that some pathologists deny the existence of this so-called 'true' form of aneurysm, and most admit its existence in the aorta only.

2. *False* aneurysm, in which the sac is formed by one only of the coats of the artery. This is almost always the external coat; but a sub-variety has been proved to exist as a consequence of wound of the outer part of the vessel, and is believed by some to take place spontaneously, in which the inner coat, or the inner and part of the middle coat, is dilated, pushed through the outer coat, and forms the sac. This is called *hernial false aneurysm*.

3. *Diffused* or *consecutive* aneurysm. Here the sac is formed of the sheath, cellular tissue, or other structures around the artery, which are matted together into the form of a membrane. The name 'diffused' is applied to this form of aneurysm to express the fact that the blood is at first diffused amongst the tissues, in consequence of the rupture or division, whether from injury or disease, of all the coats of the vessel, either in a part or the whole of its circumference; but it is not a good term, since, as soon as the aneurysmal sac is formed, the blood is diffused no longer, but, on the contrary, is encysted in the newly formed sac. So that the other term, 'consecutive,' seems a better one; expressing, as it does, the important fact that the formation of such aneurysms is always consecutive on a rupture, partial or entire, of the artery.

4. *Dissecting* aneurysm is seen only within the trunk of the body, and always involving the aorta—although it may spread from the main artery down to its branches. In this form the internal and middle coats have given way, or cracked; and the blood has forced its way, usually into the substance of the middle coat, sometimes perhaps between the middle and outer coats, distending the external portion of the vessel into a kind of aneurysm.

This is the nomenclature still in common use; but as the first and second varieties are practically indistinguishable during life, and the first, though called the 'true' form of

aneurysm, is very rare, it would be better to include both under the common name 'true' aneurysm, and apply the term 'false' to the third or 'consecutive' form.

5. Besides these, which are all forms of pure arterial aneurysm, there are aneurysms in which the vein and artery are simultaneously involved, and which are therefore called *arterio-venous*, which will be afterwards spoken of; and tumours having a certain analogy to aneurysm, which are formed of dilated and tortuous arteries—*cirsoid* and *anastomotic* aneurysms.

Other classifications of great importance are, according to the cause of the disease, into *spontaneous* and *traumatic*; or, according to the shape of the tumour, into *fusiform* and *sacculated*. In *fusiform* aneurysm there is a dilated tract of artery, often of considerable length, from either end of which springs the vessel of its natural calibre. *Sacculated* aneurysm springs like a bud from one side of the vessel, and the artery is often buried for some distance in the wall of the aneurysm; but there are many sacculated aneurysms which approach in shape to the fusiform, the vessel being dilated for some part of its extent, so that its two openings lie at different parts, and sometimes on different aspects of the sac.

**ÆTIOLGY AND PATHOLOGY.**—The proximate cause of spontaneous aneurysm appears to be usually a loss of the elasticity of the wall of the artery, whereby it loses its power of resilience after having been dilated by the force of the circulation. The loss of elasticity is commonly caused by atheroma, or else by partial calcification of the wall of the artery. In the latter case the blood often forces its way through the entire arterial wall, and an aneurysm of the consecutive variety forms,<sup>1</sup> or the external part of the artery is dissected off, and a dissecting aneurysm results. Inflammatory softening of the artery, without the presence of any definite atheromatous deposit, is looked upon by many writers of credit, such as Wilks and Moxon, as a common cause of aneurysm. Such low inflammation may have its origin possibly in rheumatism—and, as a matter of fact, aneurysm is often preceded by acute rheumatism; more certainly in violent strain, or in mechanical violence. Anything else which weakens the arterial wall, such as the exposure of the vessel in an abscess, is looked on as a cause of aneurysm. The yielding of a weakened arterial wall is doubtless accelerated by irregularities of the circulation. The influence of syphilis and of intemperance in causing aneurysm is widely believed, though perhaps as yet neither fact is absolutely established: the latter, at any rate, is rendered very probable from the consideration

that chronic alcoholism tends to impair the nutrition of all the tissues, including the arteries, and is accompanied by a constantly irritable condition of the circulation. That syphilis may cause a fibroid degeneration of the vessels must also be allowed to be at least possible, and that it does so is the opinion of many eminent pathologists. If so, the transition to aneurysm is natural, if not inevitable. Another proved cause of aneurysm is embolism, or the obstruction of a diseased artery by a fibrinous plug, which has been known to be followed by the dilatation of the artery immediately above the plug, just as in very rare cases the ligature of a healthy vessel has given rise to the formation of aneurysm above the tied part.<sup>1</sup> Violence is a very frequent cause of aneurysm, even in cases which are not technically denominated 'traumatic.' The latter term is generally restricted to cases in which the vessel is wounded by a cut, or is known to be ruptured, and the aneurysm makes its appearance at once; and in these cases the aneurysm is of the 'diffused' or 'consecutive' variety. But there are, no doubt, many cases in which the artery is partially torn, and the walls, being thus weakened, afterwards slowly yield at the injured spot. This fact is illustrated by the well-known experiment of Richerand, designed to explain the frequency of popliteal aneurysm. The experiment consists in hyper-extension of the knee in the dead subject. If this be carried on forcibly till the ligaments are heard to crack, it will usually be found that the two inner coats of the popliteal artery are torn.

All these causes of aneurysm act much more powerfully in later life than in childhood, and many are unknown in early years. Aneurysm, therefore, is very rare in children. In cases where the arterial system is extensively affected with atheroma, a great number of aneurysms may be found in the same person, or another may form after the cure of the first. To such cases the term 'aneurysmal diathesis' has been applied. This fact shows the great importance, in all cases of spontaneous aneurysm, of examining the whole body, to detect disease of the heart or any second aneurysm which may exist.

Almost all aneurysms contain more or less *clot*, and much of this clot is usually of the laminated variety, consisting almost entirely of fibrin mixed with more or less of the blood-corpuscles. These laminated coagula adhere very firmly to the interior of the sac; they are arranged concentrically like the coats of an onion; and usually lose their colour in proportion to their remoteness from the blood which still circulates through the sac. Their deposition depends in a great measure on the presence of rough projections from the wall or mouth of the sac, and on the

<sup>1</sup> Sometimes, however, the bleeding will go on without the formation of any aneurysmal sac, and lead to the loss of life or limb.

<sup>1</sup> For cases of this nature see *System of Surgery*, 3rd edit. vol. iii. p. 29.

shape of the aneurysm. When the latter is purely cylindrical, much less coagulum, possibly none, will be found in it. When the tumour stands well away from the artery, so that the force of the circulation is much broken, the formation of coagula is greatly favoured. The deposition of such firm coagula must be looked on as the commencement of spontaneous cure, and at any rate defends the patient from the risks of rupture, or of renewed growth of the tumour at the parts which are so lined.

**SYMPTOMS.**—The symptoms of arterial aneurysm are as follows:—There is a pulsating tumour, which is situated in the course of one of the arteries, and which cannot be drawn away from the vessel. The pulsation is equable and expansile, that is, it not only causes an up-and-down movement of the tumour, for such a movement may be communicated to any tumour by a large vessel lying in contact with it, but also expands the tumour laterally and in all other directions. The pulsation is in most cases accompanied by a *bruit* or blowing sound, heard on auscultation, which can be tolerably well imitated by the lips, and which is synchronous with the pulsation. Pressure on the artery above suspends both the pulsation and the bruit. Sometimes it may be noticed that the pulse below is retarded, that is, that it reaches the finger later than in the corresponding vessel on the other side. Besides these, which are the main signs of aneurysm, there are others, which are of less constant occurrence or of subordinate importance. Thus, on compression of the artery above, the tumour will empty itself more or less completely, and the greater or less change of size under these conditions is a useful test of the proportion of fluid and solid in the sac. Sometimes pressure on the artery beyond the tumour may cause an increase in its size. The pulse below the tumour is often found to differ strikingly from that on the sound side. There are many and various symptoms due to the pressure of the aneurysm on neighbouring veins, nerves, bones, and viscera—symptoms which are of subordinate importance in a diagnostic point of view in the case of external aneurysm, but are often of the greatest value in thoracic and abdominal aneurysms. Thus dyspnoea and ringing cough from pressure on the trachea, spasm or paralysis of the vocal cords from pressure on the recurrent laryngeal nerve, pain in the back from pressure on the vertebrae, or neuralgic pains from pressure on the nerves at the root of the neck, are well-known symptoms of aortic aneurysm; and, similarly, pain in the leg from pressure on the popliteal nerve, and œdema from compression of the vein, are frequent symptoms of popliteal aneurysm.

**DIAGNOSIS.**—The affections which are usually confounded with aneurysm are tumours

of various kinds lying upon arteries, abscesses, and cancerous tumours which have large vascular spaces in their interior, and therefore pulsate. The tumours which receive pulsation from arteries against which they lie are of various kinds; cysts and enlarged glands in the popliteal space, and enlargements of the thyroid body pressing on the carotid or innominate artery, are the most familiar examples. The diagnosis is usually easy. They have commonly little or no bruit, though in some cases a dull thud is produced by their pressure on the artery; they have not the expansile pulsation of aneurysm; they present no change in size or form when the circulation is stopped; and they can usually be drawn away from the artery sufficiently far to lose their pulsation. An abscess has often been mistaken for aneurysm, but the mistake has generally proceeded from a neglect of auscultation.<sup>1</sup> There are a very few cases in which aneurysms have lost their pulsation in consequence of the rupture of the sac, and in which no bruit may be audible,<sup>2</sup> and such tumours can hardly be diagnosed from abscess except by an exploratory puncture, which under these circumstances is justifiable: these cases are, however, extremely rare. The disease most commonly mistaken for aneurysm is pulsating cancer, and the resemblance has been sometimes so striking as to deceive the best surgeons, even after the fullest possible investigation of the case. These pulsating cancers almost always grow from the bones;<sup>3</sup> and the neighbouring bone can generally be felt to be enlarged, which is rare in aneurysm. They have not usually the well-marked bruit of an aneurysm, nor is the bruit usually audible over the whole tumour; the pulsation also is more indistinct, and not so expansile as in aneurysm; and the growth of the tumour is more rapid.

**COURSE AND TERMINATIONS.**—Aneurysm is generally a fatal disease if left to itself. The sac enlarges; parts of it give way, either by a process of inflammatory softening or by rupture; or it produces fatal pressure on the surrounding parts; or the whole tumour suppurates, and the patient dies of fever, of pyæmia, or of hæmorrhage. But to this general statement, independent altogether of what the effects of any special treatment may be, there are numerous exceptions. In some cases, and especially in the fusiform kind of aneurysm, the tumour, after having attained a certain size, remains stationary, and this stationary condition is sometimes produced by a deposit of coagulum

<sup>1</sup> See a paper by the author in *St. George's Hospital Reports*, vol. vii. pp. 175 *et seq.*

<sup>2</sup> See a case under the care of the writer, reported in the same paper, p. 190.

<sup>3</sup> In one case under the care of the writer the disease was unconnected with the bones, and affected the kidney only.—*Pathological Transactions*, vol. xxiv. p. 149.

lining the sac, and leaving a canal through which the blood-stream passes, as through the normal artery.<sup>1</sup> In these cases, however, the symptoms persist, but there are others in which a complete spontaneous cure is obtained; and this may happen in various ways.

*Spontaneous Cure.*—The first, and probably the most usual, method of spontaneous cure is by the gradual diminution of the circulation through the tumour, and the gradual filling of the sac by successive layers of fibrinous coagulum. The second is by impaction of clot in the mouth of the aneurysm, whereby in some cases possibly the sac of the aneurysm is cut off from the blood-stream, and its contents brought to coagulate. In other cases, where more than one artery opens out of the sac, the impaction of clot in one of the distal arteries leads to consolidation of all that part of the tumour through which the circulation used to pass into the obstructed vessel, and thus a practical cure is sometimes effected,<sup>2</sup> *i.e.* the symptoms are cured and the disease arrested, though the whole sac is not consolidated. The third method of spontaneous cure is by inflammation of the tumour. This is usually accompanied by suppuration of the sac and evacuation of all the contents of the aneurysm, the accompanying inflammation closing the mouths of the arteries which open out of it. If the arteries are not so closed, death from hæmorrhage will occur. It seems possible that inflammation of the sac and the cellular membrane around it may sometimes produce coagulation within the aneurysm without any suppuration. A fourth way in which coagulation of an aneurysm has been known to be caused is by retardation of the circulation or impaction of clot, caused by another aneurysm above; and there is an old idea, which can hardly yet be said to be exploded, that an aneurysmal sac may by its growth compress the artery, and so lead to its own coagulation. This, however, if it ever happens, is purely exceptional.

*Rupture.*—The rupture of an aneurysm may take place either through the skin, in which case the hæmorrhage is usually, but not always, fatal at once;<sup>3</sup> or into one of the cavities of the body, when death generally occurs immediately, if the rupture is into a serous cavity, and after one or two attacks of hæmorrhage if a mucous membrane has been involved; or lastly into the cellular tissue of a part. This event is marked by the cessation of the pulsation; by sudden swelling,

accompanied with ecchymosis if the blood is effused subcutaneously; and the abrupt fall of temperature below the aneurysm. A sensation of pain, or of 'something giving way,' is often experienced. Stethoscopic examination will probably detect a bruit.

*TREATMENT.*—*a. MEDICAL.*—The methods of treatment of aneurysm are very numerous, and it would be impossible in a summary of this kind to discuss fully all the indications for each. In the first place, those aneurysms which are inaccessible to any local treatment, or in which local treatment would involve great danger, are treated *medically*, that is by regimen, diet, and medicine, by which it is hoped that gradual coagulation will be promoted in the contents of the tumour, and thus a complete or a partial cure will be brought about, as in the natural process above spoken of. The method of Valsalva, of which the main features were starving and excessive bleeding, and which therefore produced considerable and often dangerous irregularity of the heart's action, is now given up in favour of another plan introduced by Bellingham and modified by the late Mr. Jolliffe Tufnell,<sup>1</sup> in which, by complete rest and restricted but nutritious diet, the absolute regularity of the heart's action is secured, and at a rate below that of health, both as to rapidity and force. Mr. Tufnell has given some interesting and conclusive examples of the complete cure of abdominal aneurysms thus accomplished, verified by dissection; and one, at least, in which an aneurysm of the arch of the aorta was in all probability entirely consolidated, though this fact was not verified by dissection. At any rate the patient was permanently restored to health. In this method of treatment drugs are only employed when necessary (as narcotics, laxatives, and tonics often are) to ensure the regularity of the functions, to control irritability, or to support the general health. The drugs which have been recommended as producing a direct effect on aneurysm by promoting the coagulation of blood in the sac, such as acetate of lead and iodide of potassium, do not, in the writer's opinion, produce any such effect, nor in fact any specific effect on the disease whatever. He has often seen a certain amount of improvement under the use of these drugs, but not, he thinks, more than the regimen and diet used at the same time would account for. Other drugs, as aconite and digitalis, are recommended in order to steady and reduce the heart's action, and the latter especially is sometimes a useful adjuvant, if employed with caution, to the treatment by restricted diet and rest. The rest is *total*, the patient never leaving his bed, nor ever rising from it or changing his position more than by occasionally turning on his side; the bowels

<sup>1</sup> See the representation of a case of this kind in *System of Surgery*, 3rd ed., vol. iii. p. 25.

<sup>2</sup> See a case of innominate aneurysm with remarks in *The Lancet*, June 15, 1872, p. 818.

<sup>3</sup> Instances of successful ligation of the artery above bleeding from ruptured aneurysm are on record. See a case in *The Lancet*, 1851, vol. ii. p. 30, in which the femoral artery was successfully tied after the bursting of a femoral aneurysm through the skin.

<sup>1</sup> *The Successful Treatment of Internal Aneurysm*, 2nd edit. 1875.

are so regulated as to avoid both constipation and looseness; and the diet is restricted to about 10 oz. of solid food, of which one half is meat or fish, and 8 oz. of fluid (comprising 2 or 3 oz. of light wine if necessary), per diem. The period may be extended indefinitely, so long as improvement continues; but in all cases the patient and his friends should be prepared for a confinement of not less than three months. *See* ABDOMINAL ANEURYSM; and AORTA, Diseases of: *Aneurysm*.

*b. SURGICAL.*—Most aneurysms which occupy an external position, and are therefore amenable to surgical treatment, are curable, when the degeneration of the vascular system is not too extensive, by mechanical means. Of these the chief and by far the most successful are either the *ligature of the artery*, whether in the sac, above it, or in some special cases below; or *compression*, applied either to the artery above the aneurysm, or to the tumour itself, or simultaneously in both situations, and either by the pressure of an instrument, of the fingers, or of Esmarch's bandage. But as these methods of treatment belong exclusively to the province of surgery, it is thought better in a work of this kind merely to name them, and to refer the reader to the standard works on surgery for their description.

The other methods of surgical treatment are far less successful than the above, and have the great drawback of being addressed exclusively to the contents of the sac; while in the treatment by the ligature and by compression the resilient power of the sac, and its consequent reaction on the blood which it contains, no doubt play a great part in the cure. The methods now to be mentioned, on the contrary, as far as they act on the sac at all, rather tend to confuse or to inflame it.

*Galvanopuncture.*—The first is galvanopuncture, in which a current of electricity of low tension, long continued, is passed through the blood in the sac, decomposing it, and causing its coagulation. Needles are plunged into the sac, and are then connected with the battery; and the action is continued until the reduction in the pulsation and the flattening of the tumour show that the blood has been partly coagulated. Authorities differ as to the details of the method. Some apply first the positive and then the negative pole to each needle; others the negative pole only, the positive being brought in contact with the neighbouring skin; while some, on the contrary, use the positive pole only. It will be found on experiment that a certain amount of coagulation takes place around both poles, the clot round the positive pole being smaller but firmer than that round the negative. The object of the operation is to fill the sac as much as possible with coagulum, which shall gradually harden, and shall attract to itself

fresh coagula. The dangers of the proceeding are those of inflammation of the sac, or of the cellular tissue around it; of suppuration within the tumour, or of sloughing of the punctures and hæmorrhage; and it must be allowed that the effects of galvanopuncture are very uncertain, both as to the amount and firmness of the coagulum produced. Still there is satisfactory evidence of benefit in many cases, and of a cure in a few. The danger of inflaming or cauterising the sac or the tissues around may be in some measure obviated by coating the needles with vulcanite, as recommended by Dr. John Duncan of Edinburgh. For a very clear exposition of the details of this method, as well as for statements regarding the success which has attended electrolysis hitherto, the reader is referred to the article on electrolysis by this authority, in Heath's *Dictionary of Practical Surgery*. The writer thinks himself justified in adding that electrolysis should be restricted to cases of thoracic, subclavian, or abdominal aneurysm, which cannot be cured by medical means, and in which rupture seems to be imminent, while the situation of the tumour forbids the application of ligature or pressure.

*Coagulating injections.*—Another method of producing coagulation of the blood in the sac is by the use of coagulating injections. Other fluids have been employed, but the only one in general use now is the perchloride of iron. The circulation is to be suspended by pressure on the artery above, and also, if possible, below the tumour, before the injection is made and for some time afterwards. The method is a very dangerous one for large aneurysms, on account of the risk of embolism, sloughing, and inflammation, but it may be used with success in small cirroid and anastomotic aneurysms, and also in varicose aneurysm.

*Introduction of foreign bodies.*—Aneurysms have also been treated by the introduction of foreign bodies into the sac, with the view of producing coagulation of the blood upon the foreign substance, such as fine wire, carbolised catgut, and horsehair; but no case of complete cure has hitherto been reported, though in a celebrated case of aneurysm of the abdominal aorta, operated on by Signor Loretta, of Bologna, the sac was completely consolidated. This was, however, followed by the rupture of the artery just above the tumour.

*Manipulation.*—Finally, aneurysms may be treated by manipulation. The object of this treatment is either to detach a portion of coagulum from the wall of the aneurysm, which may be carried into the mouth of the sac or the distal artery, and so effect a cure as in our second mode of spontaneous cure, or at any rate so to disturb and break up the clot, that its detached laminae may form nuclei for further coagulation. With this

view the aneurysmal tumour is grasped between the two hands to squeeze all the fluid blood out of it, and one wall rubbed against the other till a 'friction of surfaces is felt within the flattened mass.'<sup>1</sup> The proceeding is obviously a very dangerous and uncertain one, but some indubitable cures have been thus effected.

**Arteriovenous Aneurysms.**—A few words must be added with respect to the rarer forms of aneurysm. Arteriovenous aneurysms are generally, but not always, traumatic, and are divided into two chief forms:—1. *Varicose aneurysm*, in which there is a small aneurysmal tumour communicating both with the artery and with a vein which is always varicose; and 2. *Aneurysmal varix*, in which the opening between the two vessels is direct without any tumour interposed; the vein pulsates as well as being varicose, and the temperature of the limb and nutrition of the skin and hair are increased. In all forms of arteriovenous aneurysm the artery after a time becomes thin and much dilated. The signs of arteriovenous differ from those of arterial aneurysm mainly in this—that besides the intermittent blowing murmur caused by the arterial current, there is a continuous purring or rasping bruit due to the venous current; and that besides the intermittent pulsation there is a continuous thrill. Varicose aneurysm may be cured by digital pressure applied directly to the venous orifice, and indirectly to the artery above at the same time; or the old operation may be performed, the clots being turned out of the sac and the artery tied above and below, the vein being of course laid open and secured either by ligature or pressure; or the artery may be tied above and below without opening the sac. Electropuncture and coagulating injections have also been used with success. Aneurysmal varix does not usually require or admit of surgical treatment. If it does, the ligature of both parts of the artery is the only measure that can be adopted, on the failure of compression.

**Cirsoid and Anastomotic Aneurysms.**—*Cirsoid aneurysm*, or *arterial varix*, is a tumour formed by the coils of a single dilated and elongated artery; <sup>2</sup> while *aneurysm by anastomosis* is a tumour formed by the coils of numerous dilated and elongated arteries, with the dilated capillaries and veins which communicate with those arteries. It is often difficult to distinguish these two forms of arterial disease from each other. Aneurysm by anastomosis frequently originates congenitally as one of the forms of *nævus*. The usual situation of these tumours is on the scalp. They have often a peculiar continuous buzzing or rushing murmur, which is propagated over the whole head,

and much disturbs the patient's rest; while they are liable to ulcerate and to become the source of serious, and even fatal, hæmorrhage. Some cases of spontaneous cure are on record. Very numerous methods of treatment have been employed, of which the writer can only mention those which are most generally useful. When feasible, the total removal of the tumour with the knife is certain to effect a radical cure, but this operation is often too dangerous to be attempted. The entire removal by ligature is still more rarely practicable. The galvanic cautery is often successful: incandescent wire being drawn through the mass in various directions divides it into portions, and obliterates the vessels by producing cicatrices at the parts cauterised. Setons have also been used with success, when combined with the ligature of the trunk-artery; and the ligature of the artery alone has been said to be followed by success, but certainly is generally unsuccessful. Finally, coagulating injections and galvanopuncture have both effected a certain number of cures.

T. HOLMES.

**ANGEIECTASIA** (*ἀγγείον*, a vessel; and *ἔκτασις*, extension).—Extension or hypertrophy of the capillaries and minute vessels of the surfaces of the body, especially the skin; hence *angeiectasia capillaris*, a term applicable to several forms of vascular *nævus*. See TUMOURS: *Angioma*.

**ANGEIOLEUCITIS** (*ἀγγείον*, a vessel, and *λευκός*, white).—Inflammation of lymphatic vessels. See LYMPHATIC SYSTEM, Diseases of.

**ANGINA** (*ἄγχω*, I seize by the throat, strangle, or choke).—SYNON.: Fr. *Angine*; Ger. *die Bräune*.

The term *angina* was originally applied by Latin writers on Physic, and is still much used on the Continent, to indicate a condition in which difficulty of breathing and of swallowing exist either together or separately, caused by disease situated between the mouth and the lungs, or between the mouth and the stomach. By a special affix to the original term, significative of the seat or the nature of the disease, several morbid processes are known and described, for example:—*angina parotidea*, or mumps; *angina tonsillarlis*, or quinsy; *angina laryngea*, or laryngitis; *angina pectoris*, or breast-pang; *angina maligna*, or malignant sore-throat; *angina membranosa*, or croup.

These and numerous other diseases, differing essentially in their nature and pathological relations, and having nothing in common but certain difficulties in breathing or swallowing, are thus classed under the word *angina*. Such a classification is open to several objections, and has nothing to recommend it. With the exception, therefore, of *angina pectoris*, which has a special and familiar signification, the various dis-

<sup>1</sup> Sir W. Fergusson, *Médec. Chir. Trans.* xl. 8.

<sup>2</sup> See the figure on p. 93, vol. iii., of the *System of Surgery*, 3rd edition.

eases occasionally recognised by the term angina will be found described under the names by which they are generally known in this country. See also CYNANCHE.

RICHARD QUAIN.

**ANGINA PECTORIS.**—SYNON.: *Syncope Anginosa*; *Angor Pectoris*; Suffocative Breast-pang; Fr. *Angine de poitrine*; Ger. *Brustbräune*.

**DEFINITION.**—A paroxysmal affection of the chest, characterised by severe pain, faintness, and anxiety; coming on more or less suddenly; essentially connected with disorders of the pneumogastric, the sympathetic, and spinal nerves, and their branches; and frequently associated with structural disease of the heart.

**ÆTIOLGY.**—In searching for the *predisposing causes* of angina pectoris, we frequently notice: (1) A diseased state of the heart. (2) Morbid changes in the nerve-tissues. (3) The existence of a peculiar condition of the nervous system, which may be described as an undue susceptibility to impressions. It would seem to be often hereditary, and to be found in those temperaments in which there is a high development of the nervous element. Thus it is that this disease has caused the deaths of many who, by their intellectual development, have left their mark on the world's history. It will suffice to mention, as instances in this country, the names of Lord Clarendon, John Hunter, and Dr. Arnold. (4) The influence of *age* in relation to angina is conspicuous; the disease is rare before puberty; and, in the writer's experience, quite eighty per cent. of the cases occur after forty years of age.<sup>1</sup> (5) *Sex* also displays a marked influence amongst the predisposing causes of this disease. True angina pectoris is comparatively rare amongst women, a statement by the late Sir John Forbes showing that out of 49 fatal cases, only 2 occurred in females, and 4 out of 15 non-fatal cases—facts which entirely correspond with the writer's experience. In the hysterical temperament a form of spurious or false angina is not uncommon. (6) It is associated in many instances with certain habits of life, such as sedentary employments and indulgences at the table, and occasionally with those states of the system termed hysterical. (7) The peculiar *diathesis* which gives rise to neuralgia in various parts of the body, that in which lithic acid is in excess in the system, would seem to be in many cases an efficient cause of the symptoms of angina.

The *exciting causes* of the symptoms which

constitute angina pectoris are: (1) Mental emotion, especially anger or nervous shock. (2) Irritation propagated to the vaso-motor centre, causing a sudden rise of arterial tension, which reacts upon the heart. Such irritation may proceed from the periphery, through the cerebral nerves, as the branches of the fifth nerve in operations on the teeth; through the brachial plexus; and through the sympathetic and pneumogastric nerves distributed to the abdominal viscera. (3) Cold applied to the surface, especially by cold winds. (4) Physical exertion, or any other agency by which the heart's action is excited and its regularity disturbed. (5) Certain toxic agents, more especially excessive tobacco-smoking, malaria, and the like. (6) Indigestion, acidity, and flatulent distension.

**PATHOLGY.**—For a better comprehension of the symptoms comprised under the term angina pectoris, a description of the innervation of the heart, with the relations of its nerves to those of other organs and regions, will be found practically useful.

1. *Innervation of the Heart and its nervous relations.*—There are embedded in the heart-substance, especially towards the base of the ventricles, in the auriculo-ventricular groove, and in the auricular wall near the entrance of the great veins, minute ganglia, from which grey filaments are distributed to the muscle-tissue, whilst other fibres form a fine network beneath the endocardium. Connected with these so-called *terminal ganglia* is the cardiac plexus, and situated behind and in the concavity of the arch of the aorta, formed by the ultimate ramifications of branches from (a) the *pneumogastric*, and from (b) the *sympathetic*.

(a) The fibres of the *pneumogastric* come from the trunk, as well as from the superior and recurrent laryngeal branches of that nerve. Those fibres from the trunk and from the recurrent branch are of the 'small medullated or visceral' variety, retaining their white sheath as far as the terminal ganglia in the heart. They leave the medulla oblongata and spinal cord as part of the upper roots of the spinal accessory nerve, viz., those which arise with the vagus and with the first, second, and probably third cervical nerves, thus forming what Dr. Gaskell has designated the cervico-cranial outflow of visceral nerves (*Journ. of Physiology*, vol. vii.).

(b) The fibres of the *sympathetic* which enter the cardiac plexus are non-medullated. They are traceable thereto, with some difference in distribution on the two sides, from the superior, middle, and inferior cervical ganglia, as well as from the first thoracic ganglion of the sympathetic, and from the annulus of Vieussens, a ring of fibres which encircles the subclavian artery, and connects the two last-mentioned ganglia. These ganglia (*lateral*) are connected by communicating branches with the anterior primary branches

<sup>1</sup> Dr. J. W. Moore has directed attention (*Dublin Journal of Medicine*, February, 1890) to symptoms of angina pectoris in the acute infective diseases of children, which appear, he says, to arise from deficient innervation of the heart, granular or fatty degeneration of that organ, or from sudden increase of tension in the peripheral arteries.

of the corresponding spinal nerves, but those which join these ganglia with the eighth cervical and the first dorsal nerves are formed of grey fibres only. The central source of the cardiac sympathetic fibres is probably situated in the upper part of the spinal cord, from whence they pass out as fine medullated or visceral fibres in the *rami communicantes* from the second, third, fourth, and fifth thoracic spinal nerves. Thence passing upwards to the first thoracic ganglion, in the annulus of Vieussens, to the inferior and superior cervical ganglia, they lose their medulla, and issue forth as grey fibres, to form the superior, middle, and inferior sympathetic cardiac nerves, to the cardiac plexus above mentioned. Numerous communications take place between the various branches of the vagus and sympathetic in their course to the cardiac plexus.

With reference to the *functions* of the several nerve-structures just described, it is probable that the intrinsic cardiac ganglia are mainly concerned in distributing the impressions which reach them by the vagus and sympathetic nerves, and in regulating the nutrition of the muscular fibres of the heart's walls, which, according to recent researches, seem to possess from the cerebro-spinal centres independent automatic motor power.

The *efferent* impulses from the cerebro-spinal centres are of two kinds. The one accelerating (katabolic), which reaches the heart by way of the sympathetic fibres from the cervical and first thoracic ganglia; the other inhibitory (anabolic), transmitted by those branches of the vagus derived from the spinal accessory which pass to the cardiac plexus from the trunk and recurrent laryngeal. It should be remembered that these accelerating impulses reach the heart by channels which correspond to those taken by the vaso-constrictor nerves of the body, that is from the spinal cord by the *rami viscerales* of the upper thoracic spinal nerves. The inhibitory stimuli to the heart, on the other hand, are carried by the like branches of the cervical region through the spinal accessory.

The various *afferent* impressions, which probably originate at the endocardial surface, travel to the nerve-centres mainly, if not entirely, by the branches of the pneumogastric. Those special centripetal impulses, which by inhibiting the vaso-motor centre in the medulla oblongata, cause paralysis of the vessels of the splanchnic area, are known as 'depressor,' and reach the centre by those branches which join the cardiac plexus from the superior laryngeal and cervical cardiac nerves. There is reason to believe that painful impressions also take this course.

Lastly, the connexions of this nervous apparatus with other organs and regions must be briefly mentioned, as the paths by which cardiac disturbance may be induced. These connexions are numerous and

extensive. Directly or indirectly, most of the cranial nerves communicate with the vagus and sympathetic, such as the trifacial, and, more remotely, the *motores oculi*; the facial, both with the sympathetic and posterior auricular branch of the vagus; the glosso-pharyngeal, with filaments from the superior cervical ganglion, as well as with the ganglion of the root and posterior auricular branch of the vagus; the spinal accessory, with a communicating strand from the ganglion of the root of the vagus, and by incorporation in the pneumogastric of the entire internal branch; the hypoglossal, with branches from the superior cervical ganglion and ganglion of the root of the vagus; and lastly the cervical and brachial plexuses. In this way the greater part of the head and neck, the arms, diaphragm, and chest-walls, are brought into indirect nerve-relationship with the heart. The intimate association of the pneumogastric and sympathetic in their distribution to the pharynx, larynx, and lungs, to the gullet, stomach, and intestines, as well as the liver and other abdominal viscera, accounts for irritation in these organs reflexly affecting the heart's action. That certain emotional states influence the heart is well known, though the paths taken cannot be clearly defined.

Seeing thus how widespread are the relations existing in these various systems of nerves, and seeing how complicated are the causes which may, directly or indirectly, disturb their functions, and with them the functions of the heart itself—we can recognise the difficulty of identifying and establishing the pathology of the class of cases known as 'angina pectoris.'

2. *Anatomical Characters*.—Vascular and inflammatory changes in the nerves, such as occur in the gouty diathesis, new growths affecting the nerve-tissues, and atheromatous patches on the cardiac vessels, involving the fibres and ganglia of the cardiac plexus, and the vagus and sympathetic, have been observed and described in cases of angina pectoris.

With reference to the condition of the heart, angina may exist and lead to a fatal result without any discoverable disease in this organ or in its appendages. On the other hand, in the great majority of cases various forms of structural disease of the heart and aorta have been observed—such as atheromatous or calcareous degeneration at the orifices of or in the coronary arteries, in the valves, or in the aorta; dilatation of the cavities of the heart, or of the aorta; fibroid degeneration of the heart's walls, and accumulation of fat in their texture; and lastly, and probably the most important change of all, fatty degeneration of the muscular tissue, the nature of which was first described by the present writer. This lesion is constantly associated with the calcareous and atheromatous states just men-

tioned, and which, being more prominent, alone attracted the notice of former observers. Nay more, this lesion of the walls of the heart is in itself a frequent and sufficient cause of one of the most prominent symptoms of angina pectoris—fainting. This condition has been elsewhere described by the writer (*Medical and Chirurgical Society's Transactions*, vol. xxxiii.), under the name of *Syncope lethalis*, or fatal-faintness—a designation analogous to that given by Parry to angina pectoris, which he called *Syncope anginosa*.

The cavities of the heart have been found dilated and containing blood; or contracted and empty.

Intimately associated with the state of the heart just described are some interesting observations by Professor MacWilliam of Aberdeen, as to that condition of the cardiac muscle which is known as 'delirium cordis or fibrillar contraction.' This consists in a rhythmic, inco-ordinate, and rapidly repeated contraction of the various muscular bundles, whereby, the usual beat being abolished, the ventricles are thrown into a tumultuous state of quick, irregular, twitching action, with at the same time a great fall in blood-pressure. Such action is quite incompetent to empty the ventricle, which becomes distended with blood, and remains in a condition of diastole. This state appears to be induced by very trifling causes, frequently not recognisable. Such mechanical disturbance as the presence of a flatulent stomach or over-distended colon would certainly appear a sufficient cause, and this is a common association of an attack of angina or fatal syncope. It is probable that recovery from this condition, possible in experiments on lower animals, is impossible in man. The susceptibility of the muscle to assume this state is doubtless due to some malnutrition of a degenerative character.

3. *Summary*.—To summarise the preceding statements, it may be said that whilst there is often in angina pectoris an absence of any tangible or evident structural disease, the morbid state producing the anginous symptoms may be situated in the medulla oblongata; in the course of the nerves, or in their branches; or in the cardiac ganglia themselves. It may be the result of congestion or inflammation of the nerves, such as occurs in the lithic acid or gouty diathesis; or of other textural changes, such as connective-tissue growth or other growths involving the nerve-fibres and ganglia. An attack may be produced by emotions acting centrifugally; or by irritation acting centripetally, reflected, as just said, through the vaso-motor centre, from impressions made on the peripheral extremities of nerves. Thus acidity of the stomach, the result of indigestion, often gives rise to symptoms which very closely resemble, if they do not constitute, an attack of angina. It is by a

similar mechanism that anginal symptoms have been produced by irritation reflected from the fifth nerve, as in pivoting teeth; by such irritation of the surface of the skin as results from severe herpes; by cold, or by exposure to wind. But the most frequent source of the symptoms of true angina is to be found in those structural affections of the heart already described.

**SYMPTOMS**.—An attack of angina pectoris commences suddenly with pain in the region of the heart, generally on a level with the lower end of the sternum; occasionally it may be traced from a remote point, following the course of the nerves, even from the ends of the fingers. The pain is severe, and of an oppressive, grasping, crushing, or stabbing character. It extends sometimes across the chest, but more frequently backwards to the scapula, and upwards to the left shoulder and arm. The pain is accompanied by a distressing sense of sinking, of faintness, which causes the patient to seek support, or of impending death. The action of the heart is generally irregular. The pulse at the wrist corresponds; but in many well-marked cases of angina it is tense and resisting, yielding a sphygmographic tracing indicative of extremely high tension, more especially in the early stage of the attack. A fear of aggravating the pain prevents the patient from breathing deeply, though the respiratory function may not be really interfered with. The expression is anxious, the face is pallid, the lips are more or less livid. The whole surface of the body is pale, cold, and covered with a clammy sweat. Flatulence is often present; urine in some cases is passed at short intervals, and generally in abundance.

The attack, more or less severe, having lasted a variable time—from a few minutes to one or two hours—comes to an end, either suddenly or by degrees. The pallor and coldness of the surface are replaced by a uniform glow—the face may even flush; the pulse becomes soft and full; and there is a general feeling of relief. A sense of numbness or tingling occasionally remains along the course of the nerves derived from the brachial and cervical plexuses of the affected side.

**CLINICAL VARIETIES**.—All the phenomena of an anginal seizure as above described may be more or less modified. The attack may come on when the patient is at rest, and occasionally it sets in during sleep; but it is more commonly induced by emotion or by physical exertion, especially by walking up an ascent or by exposure to cold air or wind. The pain may be comparatively slight, and as such may recur with interruptions, it may be, during months or even years, representing anginoid symptoms rather than true angina pectoris. On the other hand it may be so severe as to mark a first, a single, and a fatal attack.

The pain may be almost limited to the region of the heart, or the lower part of the sternum; it may extend all over the chest to both arms, or spread to the sides of the head and neck and down one or both legs; and it may, in some cases, involve the diaphragm. The action of the heart may be slow, weak, and fluttering; or excited and bounding—causing a distressing sense of palpitation; it may be regular or irregular. Obstruction to the circulation from spasm of the arterioles is amongst the most constant phenomena of the seizure. The breathing sometimes assumes an asthmatic character, with comparatively little pain. There may be laryngeal spasm. The mental functions are generally undisturbed; but there is sometimes slight wandering as the attack passes off, and unconsciousness is said to be occasionally observed. The sense of danger of impending death is a characteristic symptom of angina, and one not often absent; whilst a sensation of gasping or choking, with difficulty in swallowing, is occasionally present. The position of the patient varies. Sometimes he stands, resting his arms on any convenient object; sometimes he stoops or leans forward, unwilling to be disturbed, even to speak. As a rule the attack passes off abruptly, as it commenced, leaving the sufferer free from discomfort; in other cases its disappearance is slow. The varieties in the symptoms of angina pectoris, thus seen to be remarkably numerous, characterise a form of disease which may be comparatively mild and of long duration, or one of intense suffering, hastening to a fatal termination.<sup>1</sup>

**COMPLICATIONS.**—Amongst the diseased conditions with which angina pectoris may be said to be associated, rather than complicated, are disorders of the liver and of the digestive organs, gout, renal disease, diabetes, and certain affections of the nervous system. Indeed, so marked is the latter connexion, that Trouseau dwelt on the relation which he believed to exist between epilepsy and angina—a re-

<sup>1</sup> There may be mentioned here a case of a gentleman accustomed to pass lithic acid, who, for several years liable to pains over the right side of the chest as low as the hypochondrium, was seized at night with a severe aggravation of these pains, with coldness of the surface, irregular action of the heart, depression, and other symptoms, which, had the attack commenced on the left side of the chest, would have been recognised as typical angina pectoris. Similar attacks recurred at intervals for some weeks; they were easily brought on even by walking on a level surface for a few hundred yards. The most careful examination failed to elicit any evidence of organic disease in the organs of circulation or respiration. The patient is now alive after several years and in fair health. Similar cases have since fallen under the writer's notice. An interesting case has been recorded by Dr. Alexander Morison (*Edin. Med. Journal*, February 1879), in which disease of the right side of the heart was accompanied by symptoms of angina affecting the corresponding side of the chest and arm.

lation which seems to depend on that susceptibility to nervous maladies already noticed as presented by some individuals, rather than on any special pathological connexion between these two diseases. More than one striking instance of this apparent connexion has fallen under the writer's notice. A marked example might be mentioned in which this susceptibility was such, that an oppressive meal of indigestible food brought on a first and distressing anginal seizure, which was followed by other attacks. Brain disease with epileptic symptoms was subsequently developed.

**PROGRESS, DURATION, AND TERMINATIONS.**—The progress and duration of angina pectoris will depend wholly upon the nature of its cause. Many cases in which a first attack proved fatal have come under the writer's observation, a post-mortem examination revealed the fact that there was in each slight partial hæmorrhage into the walls of the heart, which had been the seat of fatty degeneration, connected with calcification of the coronary arteries. The symptoms in these cases were those of the most severe form of angina pectoris. On the other hand, cases present themselves in which symptoms may occur at intervals for years. Thus in many instances individuals present all the symptoms of marked angina, accompanied by most of its distressing phenomena, and by the anxieties and fears that they beget; yet these cases, having more a neurotic or gouty origin, are controlled by treatment, the sufferers being restored to health, or continuing for years to enjoy comparative comfort. On the other hand, in the cases in which angina is connected with structural disease of the heart or of the nerves controlling cardiac action, the attacks progress in frequency and severity, and tend, with more or less certainty, to a fatal termination—it may be within a few days or weeks.

**DIAGNOSIS.**—A typical case of angina pectoris, such as has been already described, can hardly be mistaken. But when the several symptoms constituting an attack are variously modified, some being lessened in severity and others exaggerated; or when these symptoms depend on, so to speak, remote and removable causes; it is often difficult to say how far the disease is what may be regarded as a remediable neuralgia, or an attack of what is commonly recognised as true angina pectoris. So also it may be difficult to say, in cases of true angina, whether the seizure is dependent on structural lesions which admit of no improvement, or on some condition that may be amenable to treatment. But it is with this affection, as with so many others, more difficult to determine the nature of the cause on which the symptoms depend, than merely to recognise the presence of the disease itself. With reference to the diagnosis of the structural diseases of the heart above referred to,

it is unnecessary to describe here that which will be found fully treated of under special heads. It remains but to say that in every case the closest scrutiny must be made into the condition of the heart and great blood-vessels, with a view to determine the presence or absence of disease. The investigation must further extend to other viscera, such as the liver and stomach, and to the digestive organs generally, as well as to the several other sources from which symptoms of angina may be excited by the reflected irritation already described. Certain symptoms resulting from the presence of other diseases should not be confounded with angina—such, for example, as the pain and dyspnoea caused by pressure of aneurysms or of tumours within the chest; by rheumatic or gouty neuralgia of the chest-walls; by pleurodynia, or acute pleurisy; or by indigestion, acidity, and flatulence. Each and all of these several conditions must be considered by way of exclusion in determining the nature and origin of the disease.

**PROGNOSIS.**—In anticipating the future of an attack of angina pectoris, we must be guided chiefly by a knowledge of its cause; in some respects also by its severity; and by the previous history of the case. Thus, if we can ascertain that the attack has been brought on by some clearly established and removable cause, such as fatigue, a chill, or indigestion, a favourable prognosis may be fairly entertained. On the other hand, if the history of the case tells that there have been several previous attacks, each increasing in severity and connected with heart-disease, we can scarcely avoid being led to the conclusion that the complaint will tend, with more or less rapidity, to a fatal termination. Between these two classes of cases there exist a large majority of instances in which the symptoms of angina, of greater or less severity, depend on neurosis, on gouty diathesis, or on other sources of nerve-disorder, amenable to treatment; and in which, therefore, a favourable prognosis may to some extent be given. But in all cases great caution should be exercised; for many instances occur in which, from slight and obscure beginnings, severe and even fatal examples of the disease have been developed.

**TREATMENT.**—The treatment of angina pectoris must, first, have reference to relief of the attack itself; and, secondly, during the interval, to the removal, if practicable, of the causes on which the attacks may depend.

*During the attack,* it is necessary first, if possible, to lessen the patient's anxiety and fear. He should be allowed to retain the position in which he feels most comfort, and an attempt must immediately be made to relieve the suffering. If the exciting cause be one that can be removed, this should be accomplished. For example, if the stomach be full of undigested food, an emetic of mustard

might be given with advantage; or if flatulence be present, peppermint, ether, and other antispasmodics will be useful. If cold have produced the seizure, the feet and hands should be immersed in hot water, hot bottles applied to the surface of the body, and poultices of linseed or mustard, or embrocations of chloroform or laudanum, should be applied to the chest. For the more immediate relief of pain some of the agents now known to be effectual for the purpose should be administered. The nitrite of amyl, originally introduced by Dr. Lauder Brunton, has been found a more efficient remedy than any other hitherto recommended for the direct relief of the distressing symptoms of the disease. Five or six minims of this drug, which is usually dispensed in capsules, should be carefully inhaled from a handkerchief or piece of lint, and, if necessary, the inhalation may be repeated.

Nitroglycerine, suggested by Dr. Murrell, seems also to act efficiently in doses of  $\frac{1}{100}$  grain, in the form of the official tabellæ or 1 per cent. alcoholic solution. The dose may be repeated three or four times at short intervals; but these drugs must be used with caution, as in many cases they produce headache and superficial congestion.

The action of these agents depends on the power they possess of relaxing the arterioles, thus diminishing the peripheral resistance, freeing the heart's action, and readjusting the circulation. If the pain be of a more persistent character, hypodermic injection of morphia may be used with advantage.

In those cases in which debility and exhaustion exist, the ordinary stimulants will be required, and various antispasmodics, such as ether, ammonia, &c., may be given with more or less benefit. In cases of sudden cardiac failure Professor MacWilliam recommends faradic excitation of the heart, in the form of a series of periodic single induction shocks, at the normal rate approximately of cardiac action. These should be sent through the heart so as to affect the auricles as well as the ventricles, one electrode being applied over the seat of the cardiac impulse, the other over the fourth dorsal vertebra. Large sponge-electrodes should be used, they and the skin being well moistened with salt solution. The shocks employed should be strong—sufficient to excite powerful contraction in the voluntary muscles.

*During the intervals.*—It is of course desirable to avoid all causes likely to bring on an attack of angina, such as mental excitement, bodily exertion, exposure to cold, and indulgence in indigestible food or heavy meals. The leading principle in treatment should, however, be to endeavour to determine and to remove, when possible, the cause of the attacks. Whether they depend on organic disease of the heart, whether on simple neuralgia, whether on gout or dyspepsia,

whether on debility, or on fulness of habit—to each of such conditions must appropriate treatment, as described in other parts of this work, be persistently directed. A variety of specific remedies has been recommended, such as iodide of potassium or sodium in diseased states of the arteries, arsenic, phosphorus, iron, zinc, and the different sedatives and antispasmodics. Galvanism, in the form of the continuous current from thirty cells, has proved of use in some uncomplicated cases, the positive pole being placed on the sternum, and the negative on the lower cervical vertebrae. Huchard, in his exhaustive work on angina pectoris, discusses the action of antipyrin and phenacetin, which he regards as useful in false angina. He describes as useless, or even dangerous, the employment of chloral, sulphonal, atropine, and certain other drugs. Excellent, however, as many of the remedies above named may be under special and suitable circumstances, the result of treatment must entirely depend on the cause of the disease, and how far it is within reach of well-directed agencies. Some cases of apparently severe angina will be found to yield to treatment; whilst, as might be expected from the nature of the complaint, others unhappily proceed to a fatal termination in spite of every effort directed to their relief.

RICHARD QUAIN.

**ANHIDROSIS** (*ἀ, priv.*; and *ἰδρῶς*, sweat).—Absence or want of perspiration. *See PERSPIRATION, Disorders of.*

**ANHIDROTICS** (*ἀ, priv.*; and *ἰδρῶς*, sweat).—Agents which check perspiration. *See PERSPIRATION, Disorders of.*

**ANILINE POISON.**—The aniline dyes, which are a modern discovery, present the most brilliant hues of yellow, blue, and red; as such they have been used for dyeing stockings, gloves, &c. These articles when worn are apt to produce an intense form of inflammation and vesication of the skin, which is rebellious against treatment, and liable to relapse for many months after the original attack has subsided. *See DERMATITIS.*

**ANIMAL POISONS.**—*See POISONS.*

**ANKYLOSIS** (*ἀγκύλος*, crooked).—Marked stiffness or absolute fixation of a joint. Ankylosis may be fibrous, bony, or vital. Ankylosis is said to be 'vital' when it is due to the impairment of motility caused by rigidity of the muscular apparatus surrounding the joint. *See JOINTS, Diseases of.*

**ANKYLOSTOMA** (*ἀγκύλος*, crooked; and *στόμα*, a mouth).—A genus of nematoid worms. *See ENTOMOZOA.*

**ANODYNES** (*ἀ, priv.*; and *ἰδρῶς*, pain).—**DEFINITION.**—Medicines which relieve pain by lessening the excitability of nerves or of nerve-centres.

**ENUMERATION.**—Anodyne medicines include Opium and its alkaloids—Morphine and Codeine; Antipyrin, Antifebrin, Phenacetin, and Exalgin; Bromide of Potassium; Cannabis Indica; Belladonna and its alkaloid—Atropine; Hyoseyamus and Hyoseyamine, Stramonium; Cocaine; Aconite and Aconitine; Veratrum and Veratrine; Conium and Conine; Lupulus and Lupulin; Gelsemium: Chloroform, Ether, and their allies; Chloral Hydrate; Butyl-chloral Hydrate; and Camphor.

**ACTION.**—Pain is due to a violent stimulation of a sensory nerve being conveyed to some of the encephalic nerve-centres (probably the cerebral hemispheres), and perceived there. The impression produced on all sensory nerves, except the cephalic nerves, is conveyed, for a part of its course, to the head along the spinal cord. The primary impression which is felt as pain, is usually made upon the peripheral ends of the sensory nerves; but it may also be made upon their trunks, upon the spinal cord, or possibly upon the encephalic centres directly, without any affection of the nerves themselves, as, for example, in hysteria. Pain may therefore be relieved, while the source of irritation still remains, by lessening the excitability of the ends of the sensory nerves which receive the painful impression; of their trunks; of the spinal cord along which the impression travels; or of the encephalic centre in which it is perceived. Opium acts by lessening the excitability of the sensory nerves, the spinal cord, and the encephalic ganglia. Bromide of potassium is also believed to act on all three, although to a much less degree than opium. Antipyrin and its allies probably affect the conduction of painful stimuli through the spinal cord or sensory tracts in the brain. Belladonna and atropine affect the sensory nerves, as probably does hyoseyamus. Stramonium, aconite and aconitine, veratrine, chloral and butyl-chloral, lupulus and lupulin, and gelsemium probably act on the encephalic centres.

**USES.**—As opium and morphine act upon all the nervous structures concerned in the production of pain, they may be used to relieve pain whatever its cause. Cannabis indica and bromide of potassium may be employed under the same circumstances as opium, but they have very much less power. Antipyrin, antifebrin, phenacetin, and exalgin are powerful analgesics, and relieve pain without disturbing the brain. They are very useful in neuralgic pains, in headaches, and in pain depending on some affections of the spinal cord—for instance, to relieve the lightning pains in locomotor ataxy. Chloral seems to relieve pain only by inducing sleep, and does not produce an anæsthetic effect unless it is given in dangerous doses. Butyl-chloral also induces sleep, but

seems to have a special sedative action on the fifth nerve; as likewise has gelsemium—and hence both these agents are used in the treatment of facial neuralgia. As the action of belladonna is exerted chiefly on the peripheral ends of the sensory nerves, this remedy is usually applied directly to the painful part in the form of plaster, liniment, or ointment. Aconite, veratrine, cocaine, and opium are also used as local applications in several forms, for the relief of pain. The various anodynes may be administered not only by the mouth, but by other channels, such as by inhalation, by enema or suppository, by hypodermic injection, or by endermic application.

Several therapeutic measures are employed as local anodynes, such as the application of dry or moist heat; cold; electricity; various forms of counter-irritation; acupuncture; and the abstraction of blood.

T. LAUDER BRUNTON.

**ANOREXIA** (*ἀ*, priv.; and *ἀρεξία*, appetite).—Want or deficiency of appetite, not accompanied with disgust for food. *See* APPETITE, Morbid Conditions of.

**ANOSMIA** (*ἀ*, priv.; and *ὄσμη*, smell).—Loss of the sense of smell. *See* SMELL, Disorders of.

**ANTACIDS**.—DEFINITION.—Medicines used to counteract acidity of the secretions.

ENUMERATION.—The antacids include Potash, Soda, Lithia, Ammonia, Lime, Magnesia, and their Carbonates; as well as the salts which the alkalis form with vegetable acids, such as Acetates, Citrates, and Tartrates.

ACTION.—Antacids are divided into: (1) those which act *directly*, lessening acidity in the stomach; and (2) those which act *remotely*, diminishing acidity of the urine. The alkalis and alkaline earths and their carbonates, with the exception of ammonia, have both a direct and remote influence; for when swallowed they act on the stomach, and being absorbed from the intestinal canal, they are excreted by the kidneys, thus lessening the acidity of the urine. Ammonia and its carbonate are direct but not remote antacids; for, although they neutralise acidity in the stomach, they are partly excreted in the form of urea, and do not diminish the acidity of the urine. The acetates, citrates, and tartrates of the alkalis and alkaline earths, on the other hand, are remote but not direct antacids. They have no antacid effect in the stomach, but undergo combustion in the blood, being converted into carbonates, in which form they are excreted in the urine, and diminish its acidity.

USES.—Excessive acidity of the contents of the stomach gives rise to acid eructations and heartburn. It may sometimes depend on the secretion of a too acid juice by the stomach, but is generally caused by the formation of acid from the decomposition of

food when the process of digestion is slow and imperfect. Antacids are given after meals to lessen acidity in the stomach, and afford immediate relief to its attendant symptoms. They may prove even more efficacious by preventing acidity when given before meals (*see* ALKALIS). If the action of the bowels be regular, soda is preferable; but lime should be used if they are relaxed, and magnesia if there is a tendency to constipation. Remote antacids are given to lessen the acidity and irritating qualities of the urine in cystitis and gonorrhœa; and to prevent the deposition of uric acid gravel or calculus in gouty persons. For this purpose potash and lithia are preferable, as their urates are more soluble than those of the other bases.

T. LAUDER BRUNTON.

**ANTAGONISM**.—This term is employed to express the fact that the physiological action of certain substances may be affected, even to the extent of neutralisation, by the presence in the body, at the same time, of other substances having an action of an opposite character. It is important to distinguish between *antidotal action* and *physiological antagonism*. By an *antidote* is meant a substance which so affects the chemical or physical characters of a poison, as to prevent its having any injurious action on living animal tissues. Thus acids and alkalis neutralise each other, so as to form innocuous salts; tannin may render tartar-emetic and many vegetable alkaloids insoluble; and the hydrated sesquioxide of iron may be used to precipitate arsenious acid. In these cases, the action is limited chiefly to the alimentary canal; and the object of administering the antidote is to form insoluble salts, or compounds which will be physiologically inert. But the *physiological antagonism* of certain substances is presumed to take place in the blood or in the tissues. When such a substance as strychnine, for example, is introduced into the alimentary canal, it is quickly absorbed, and carried by the blood throughout the body. It does not, so far as observation has discovered, influence all the tissues; but it so affects the spinal cord, and possibly the brain, as to give rise to severe tetanic convulsions, chiefly of a reflex character. This effect is, no doubt, due either to some interference in the nutritional changes between the blood and the tissues composing the nerve-centres, or to some specific action of the poison on the nerve-centres themselves (*see* AFFINITY). These changes, which are termed physiological, and on which the normal action of the nerve-centres depends, are probably of a molecular or chemical nature; and it is possible to conceive that they may be modified in different ways by different substances. Thus has arisen the idea of physiological antagonism; and ex-

periment has shown that, within certain limits, which will no doubt vary in each case, such an antagonism is possible. Antagonism may be either local, affecting one organ, as is seen in the opposite effects upon the pupil of opium or morphine upon the one hand, and stramonium, hyoscyamus, or belladonna upon the other; or it may extend apparently to more important organs or groups of organs, as in the case of the antagonism between strychnine and the hydrate of chloral. The most important investigations upon the subject of physiological antagonism are the following:—

(1) *Physostigmine* and *Atropine*, by Professor Fraser—an inquiry which showed that the fatal effect of three and a half times the minimum fatal dose of physostigmine may be prevented by atropine. (2) *Atropine* and *Prussic Acid*, a research by Preyer of Jena—of a more doubtful character as regards the point to be proved, but still sufficient to show that, within certain limits not yet indicated, it is possible to prevent the fatal action of prussic acid by atropine. (3) *Atropine* and *Muscarine* (the active principle of *Agaricus muscarius*)—which were found by Schmiedeberg and Koppe to have entirely antagonistic actions on the ganglia of the heart—muscarine exciting the intracardiac inhibitory centres, and stopping the heart in diastole, while atropine has the contrary effect. (4) *Chloral* and *Strychnine*—an antagonism first pointed out by Oscar Liebreich, who showed that minute doses of strychnine might so rouse an animal from the effects of an overdose of chloral as even to save its life. And (5) *Strychnine* and *Chloral*—with respect to which Hughes Bennett demonstrated the converse of the last-mentioned observation, namely, that in the rabbit a fatal dose of strychnine might be so antagonised by a dose of chloral as to save life. Other examples of antagonism are: chloroform and amyl-nitrite, cocaine and morphine, atropine and jaborandi, strychnine and hydrocyanic acid, thebaine and chloral, and digitalin and aconitine.

**CONCLUSIONS.**—It has unfortunately to be admitted that the practical results of the preceding researches have not been very encouraging. In all of these investigations it was quite apparent that the limits of physiological antagonism were very narrow. Three elements affect the chances of success in the way of saving life:—(1) the age and strength of the animal; (2) the amount of the doses of the two active substances—so that if either the one or the other active substance be given slightly in excess, death will probably take place; and (3) the time between the administration of the two active substances. If the stronger be introduced first, and be allowed to manifest distinctly its physiological action, it is almost impossible to counteract this by that of another substance; but if the two

substances be introduced simultaneously, or if the supposed antagonist to the more active substance be introduced first, the chances of success are much greater. It is apparent, therefore, that the facts relating to physiological antagonism at present known in science do not hold out much hope of good results from their application in practice; but still the physiological facts are so definite as to indicate a precise mode of treatment. For example, no one acquainted with the investigations mentioned above would hesitate in attempting to relieve the tetanic spasms of a case of poisoning by strychnine by repeated doses of hydrate of chloral, or by the administration of chloroform. A practical result of such researches is that the principle of physiological antagonism may serve as a guide to the application of remedies in disease. Thus excessive secretion, say from mucous membranes or from salivary glands, may be modified or arrested by the use of sulphate of atropine, a striking experimental demonstration of which may be seen in the antagonism between bromal hydrate and sulphate of atropine in the rabbit.

JOHN G. MCKENDRICK.

**ANTEFLEXION.**—A bending forwards of any organ. The term is specially used in relation to the uterus, when this organ is bent forwards at the line of junction of its body and cervix. See WOMB, Diseases of.

**ANTEVERSION.**—A displacement forwards of any organ. The term is particularly applied to a change of position of the uterus, in which this organ is bodily displaced in the pelvic cavity, so that the fundus is directed against the bladder, and the cervix towards the sacrum. See WOMB, Diseases of.

**ANTHELMINTICS** (*ἀντι*, against; and *ἔλμυς*, a worm).—**DEFINITION.**—Medicines which kill or expel intestinal worms.

**ENUMERATION.**—The principal anthelmintics are:—Oil of Male Fern; Kamala; Koussou; Oil of Turpentine; Pomegranate Root; Santonica (Worm-seed, so-called), and its active principle, Santonin; Areca; Mucuna; Rue; and drastic purgatives. As purgatives only expel the worms, they are termed *vermifuges*; while the other anthelmintics which kill the worms are called *vermicides*.

**ACTION.**—The oil of male fern, kamala, koussou, oil of turpentine, and bark of pomegranate root, act as poisons to tape-worms. Santonica and santonin remove round-worms, and also thread-worms. They were formerly supposed to kill the worms, but it would appear that they only weaken or intoxicate them, so that they are more readily expelled. Castor oil, jalap, scammony, and other purgatives do not kill the worms, but dislodge and expel them, by the increased peristaltic action which they occasion.

USES.—Drastic purgatives may be used for worms of any sort; areca for both tape- and round-worms; and the other agents for the worms on which they severally act as poisons. Vermicides are generally given after the patient has fasted for several hours, in order that, the intestines being empty, the drugs may act more readily on the worms. A purgative is usually given some hours afterwards, in order to expel the dead worms. As thread-worms chiefly inhabit the rectum, they are most effectually killed by enemata, which may consist of a strong infusion of quassia; salt and water; vinegar and water; solution of the sulphate or perchloride of iron; oil of turpentine; castor oil; decoction of aloes; or infusion of senna. As abundance of mucus in the intestines forms a convenient nidus for the growth of worms, anything that diminishes this tends to prevent their occurrence; and for this purpose preparations of iron and bitter tonics are useful.

T. LAUDER BRUNTON.

**ANTHRACOSIS** (*ἀνθραξ*, a coal).—A deposit of black material in the tissues or organs. See LUNGS, Inflammation of.

**ANTHRAX** (*ἀνθραξ*, a coal).—A synonym for carbuncle, and for malignant pustule. See CARBUNCLE; and PUSTULE, MALIGNANT.

**ANTIDOTE** (*ἀντι*, against, and *δίδωμι*, I give).—DEFINITION.—An antidote is any remedy which, by its physical or its chemical effect upon a poison, or in both ways, is capable of preventing or counteracting the physiological effects of that substance (see ANTAGONISM). Sometimes, however, the term is used in a more comprehensive sense, so as to include the general treatment of a person affected by a particular poison. Thus, in poisoning by opium, the use of the stomach-pump, enforced exertion, chafing the limbs, and artificial respiration may be included in the general antidotal treatment.

MODES OF ACTION, AND APPLICATION.—Most antidotal substances form with the poison insoluble or innocuous compounds. Without attempting to give a complete list, the following are examples of the more common poisons and their respective antidotes:—(1) *arsenious acid*: solution of dialysed iron administered freely, or light magnesia; (2) *prussic acid*: newly precipitated oxide of iron with an alkaline carbonate; hypodermic injection of 2 to 4 ℥ of liquor atropinæ sulphatis every half-hour; (3) *oxalic acid*: chalk, common whiting, or magnesia suspended in water; (4) *tartar emetic*: tannin, catechu, or other vegetable astringents; (5) *acetate of lead*: sulphate of magnesium, or the phosphates of sodium and magnesium; (6) *caustic potash*: dilute acetic acid, fixed oils, lemon juice; (7) *corrosive sublimate*: albumin, white of

egg, flour, or milk; (8) *mineral acids*: chalk, common whiting, plaster from the walls or ceiling, or carbonate of magnesium; (9) *chloride of zinc*: albumin, milk, or carbonate of sodium.

Vegetable poisons cannot thus be counteracted. If they have been taken in the form of seeds, leaves, or roots, the proper course is to remove them from the stomach or bowels as soon as possible by emetics and purgatives, and at the same time to sustain the flagging strength of the patient by the administration of stimulants. On the other hand, if the alkaloid has been taken, it is so soon absorbed that emetics and purgatives are of little avail, or may even be injurious. In these circumstances we must rely on the administration of the physiological antagonist of the poison (such as chloral hydrate in the case of strychnine-poisoning), and on supporting the strength of the patient. The following are the best antidotes to the vegetable poisons most frequently met with:—(1) *aconite root*: emetic of sulphate of zinc and stimulants; (2) *belladonna leaves, berries, or root*: emetic of sulphate of zinc, ammonia, stimulants, and after some time an active purgative; (3) *digitalis*: emetics, stimulants, and the maintenance of the recumbent position; (4) *hyoscyamus leaves*: emetics and stimulants; (5) *hydrochlorate or meconate of morphine, or any of the preparations of opium*: hypodermic injection of apomorphine; external stimulation by warmth, turpentine or camphor liniments; enforced exertion; artificial respiration, and small repeated doses of sulphate of atropine; (6) *chloral hydrate*: the same as for opium; (7) *strychnine or nuxvomica*: animal charcoal suspended in water, repeated large doses of chloral hydrate, or chloroform; (8) *atropine*: stimulants, subcutaneous injection of caffeine, morphine, or pilocarpine, or of very minute doses of physostigmine; (9) *calabar bean*: atropine, or stimulants, artificial respiration; (10) *mushrooms*: subcutaneous injection of 2 to 4 ℥ of liquor atropinæ sulphatis, stimulants, castor oil; (11) *tobacco or nicotine*: stimulants, subcutaneous injection of minute doses of strychnine. See POISONS.

JOHN G. M'KENDRICK.

**ANTIMONY**, Poisoning by.—See TARTAR EMETIC, Poisoning by.

**ANTIPERIODICS**.—DEFINITION.—Medicines which prevent or relieve the paroxysms of certain diseases which exhibit a periodic character.

ENUMERATION.—The chief antiperiodics are:—Cinchona-bark and its alkaloids—Quinine, Cinchonine, Quinidine, and Cinchonidine; Bebeeru Bark and its active principle, Beberine; Salicin, Salicylic Acid and its salts; Eucalyptus globulus; and Arsenic.

ACTION.—The mode of action of antiperiodics is at present unknown.

**USES.**—Cinchona, and still more quinine, is almost a specific in the treatment of intermittent fevers, periodic headaches, neuralgias, and other affections caused by malaria. Though less certain in its action than in intermittent fevers, quinine is also the best remedy in the remittent fevers of the tropics, in which, however, it must be given in very large doses. Its action in these fevers appears to be assisted by antipyrin or other antipyretics, given so as to reduce the temperature to the normal during the remission. The other alkaloids of cinchona have a similar action to that of quinine, but they are not so powerful. Beberine is only about one-third as powerful, and is by no means so certain; and the same remark applies to the other remedies enumerated. In some cases of ague and other intermittent affections arsenic proves successful when quinine fails. Emetics and purgatives are useful auxiliaries to quinine in the treatment of ague, and are employed alone for the cure of this disease in some parts of the world where quinine is not available.

T. LAUDER BRUNTON.

**ANTIPHLOGISTIC** (*ἀντι*, against; and *φλέγω*, I burn).—A term for any method of treatment that is intended to counteract inflammation and its accompanying constitutional disturbance.

**ANTIPYRETICS** (*ἀντι*, against; and *πυρετός*, a fever).—**DEFINITION.**—Therapeutic agents which reduce the temperature in fever.

**ENUMERATION.**—The principal agents used as antipyretics are—Cold Baths, Cold Applications, Ice; Diaphoretics; Alcohol; Chloral; Quinine; the Salicyl compounds; Antipyrin, Antifebrin, Phenacetin, and allied bodies; Eucalyptol; Essential Oils; Aconite; Digitalis; Green Hellebore; Purgatives; and Venesection.

**ACTION.**—The temperature of the body may be reduced, either by increasing the abstraction of heat, or by lessening its production. The direct application of cold, by means of baths, affusion, or sponging, or by enveloping the body in sheets wrung out of cold water, is the most powerful and rapid means of abstracting heat. But the loss of heat which constantly occurs, even in health, by evaporation of the sweat, and the radiation and conduction of heat from the skin, may be increased by the use of diaphoretics, such as salts of potassium, preparations of antimony, or acetate of ammonium; or by such medicines as dilate the cutaneous vessels, so as to allow the heated blood to circulate freely through them, and to become cooled by the external media surrounding the skin. Alcohol, in the form either of wine or spirits, and chloral have an action of this sort, though alcohol also influences the production of heat. Alcohol, quinine, the salicyl compounds, antipyrin, antifebrin, phenacetin, pyrocin, and their allies, eucalyptol, and

essential oils lessen the production of heat within the body, probably by diminishing oxidation of the tissues (*see* ALCOHOL). Aconite, digitalis, and green hellebore reduce the temperature, but their mode of action is not precisely ascertained.

**USES.**—Antipyretics act much more powerfully in reducing the temperature of the body in fever than they do in health. They may be used when the temperature has risen either from exposure to a high external temperature, as in thermic fever; in consequence of inflammation, as in pneumonia, pericarditis, or phthisis; or in specific fevers, as acute rheumatism, typhus, and scarlatina. The most rapid and powerful antipyretic remedies are cold baths; next probably come antifebrin, antipyrin, phenacetin, large doses of salicylic acid or salicylate of sodium, and quinine. In acute rheumatism the salicylates are the most useful antipyretics. Quinine seems to act very efficiently in thermic fever when injected subcutaneously.

T. LAUDER BRUNTON.

**ANTISEPTICS** (*ἀντι*, against; and *σηπτικός*, from *σήπω*, I make putrid).—**SYNON.**: Antiputrescents.

**DEFINITION.**—An antiseptic is a substance which prevents or retards putrefaction—that is, the decomposition of animal or vegetable bodies, accompanied by the evolution of offensive gases. The putrefactive change occurs only in dead matter, and requires the presence of moisture, heat, and a ferment. That there is no putrefaction in the absence of moisture is obvious, for bodies, such as albumin and blood, which in the moist state are highly susceptible of putrefaction, may be kept for an indefinite time without change if they be perfectly dry. Heat also has an important influence on putrefaction. At very low temperatures the putrefactive change ceases, while elevated temperatures, such as prevail in tropical climates, are favourable to it. An additional element besides heat and moisture is, however, required, and the opinion generally accepted is that this consists of minute vital organisms, which excite putrefactive decomposition.

**MODE OF ACTION.**—The substances used as antiseptics act either directly on the bodies in which putrefaction is occurring or might occur, forming with them combinations that are not susceptible of the decomposing action of a ferment; or they act indirectly, by destroying the vitality or otherwise preventing the development and propagation of the organisms of which the ferment is composed. In this respect *antiseptics* may be distinguished from *disinfectants*, the action of the latter being directed only against the exciting causes, and offensive or deleterious products, of a class of changes which are themselves more comprehensive than those implied by the term putrefactive.

ENUMERATION. — There are numerous chemical agents possessing antiseptic properties, among the chief of which are:—Chlorine, Sulphur Dioxide (Sulphurous Acid), Nitric Oxide, and Peroxide of Nitrogen, as gases; Carbolic Acid, Asepsol, Creasote, Benzol, Sulphites, Hyposulphites, and Hypochlorites, which emit vapours at common temperatures; and Mercuric Chloride (Corrosive Sublimate), Mercurio-Zinc Cyanide, Chloride of Zinc, Chromic, Boric, Tannic and Salicylic Acids, Permanganate of Potassium, Naphthol, Resorcin, and Charcoal, from which no vapour is emitted.

USES.—1. In therapeutic practice antiseptics are chiefly employed in the *treatment of surgical operations and of open wounds*, to prevent the occurrence of putrefactive decomposition. See ANTISEPTIC TREATMENT.

Those antiseptics are best suited for this purpose which, acting efficiently on the ferment, have no injurious effect on the parts in which the healing process is going on. Gases, except in solution, cannot be locally used, as it would be necessary to enclose the substance to be preserved in an air-tight vessel containing the gas. The volatile antiseptics, which slowly emit a vapour, have been preferred to those which emit no vapour, although among the latter salicylic and boric acids, when properly diluted, being devoid of any irritating properties if applied to inflamed surfaces, present on this account a marked advantage. Latterly a cyanide of mercury and zinc, which is more powerfully antiseptic than those last named, and equally devoid of irritating properties, has been successfully used and recommended by Sir Joseph Lister. Corrosive sublimate, which appears to be one of the most powerful of antiseptics, is also now frequently used in the form of a weak solution, one part in 2,000 parts of water being efficient.

Carbolic acid, which was the first active disinfectant used in surgical operations, still retains its position as one of the most valuable of this class of chemical agents. It is usually employed in solution in water, one part of the pure crystallised acid being dissolved in from 20 to 100 parts of water, and applied as a lotion, and sometimes in the form of spray, diffused through the atmosphere during a surgical operation or the dressing of a wound. The solution may also be used on lint or cotton-wool, for covering the affected part. Antiseptic gauze for a similar purpose may be made by adding one part of crystallised carbolic acid to five parts of common resin and seven parts of paraffin, melted together and applied to coarse muslin, so as to form a thin coating, which, when it has hardened, is used for covering the parts to be protected.

Mercuric preparations, and especially the cyanide of mercury and zinc, have to some extent taken, and will probably still further

take, the place of carbolic acid for the purpose under notice. The cyanide, however, although having the advantage of its non-irritating properties, has the defect of insolubility.

Boric acid is prepared as a dressing for rodent ulcers. This is composed of boric acid and white wax, each one part, paraffin and almond oil, each two parts. The boric acid and oil are added to the melted wax and paraffin, and the whole is stirred in a mortar until it thickens, and then set aside to cool and harden, after which it is to be rubbed in the mortar until it acquires the consistence of an ointment. This is thinly spread on fine rag and applied to the wound. The oil separates and is absorbed by lint or rag placed over the dressing, while a firm plaster, which is easily removed when necessary, remains attached to the skin.

Salicylic acid is sometimes substituted for boric acid in the dressing last described; or the finely powdered acid may be merely sprinkled over the part affected. Its very slight solubility in water presents an obstacle to its use in solution, unless something be added to render it more soluble; borax, which is itself a good antiseptic, may be used for this purpose. One drachm of salicylic acid, two drachms of borax, and half an ounce of glycerine, with three ounces of water, form, if aided with a little heat, a clear solution which may be used as an antiseptic lotion.

2. In *medical practice* antiseptics are also employed, either as local applications or as internal remedies. Those which are chiefly available include creasote, carbolic acid, the sulphocarbolates, sulphurous acid, the sulphites or hyposulphites, chlorine water, permanganate of potassium, borax or boric acid, chlorate of potassium, charcoal, salicylic acid, menthol, and thymol. They are principally used for the prevention and treatment of infectious fevers, and in low forms of ulceration of the throat.

3. In using antiseptics for the *preservation of anatomical specimens*, a wider range of chemical agents may be taken, and a selection made of substances that would be inapplicable in the treatment of the living subject. Arsenious acid, corrosive sublimate, in other than the weak solutions previously noticed, and chloride of zinc in solution, are of service for this purpose; and chromic acid, even when diluted with from five hundred to one thousand parts of water, possesses the property of preserving animal matter from decomposition, as also does a solution of one part of borax in forty parts of water. Alcohol and glycerine are also employed for the preservation of animal and vegetable substances as specimens. For the like purpose, drying, or drying and smoking are used, as in the preparation of bacon, pemican, fish, &c.

4. Antiseptics of a milder nature, such as

common salt, nitre, and sugar, are adopted for *preserving articles of food*.

T. REDWOOD.

### ANTISEPTIC TREATMENT. —

**PRINCIPLES:**—The 'Antiseptic System' of treatment, introduced by Sir Joseph Lister about 1867, which has in great measure revolutionised modern surgery, depends upon certain postulates, the appreciation and acceptance of which are essential to its successful application. They are:—

I. Septic processes in a wound, and the constitutional changes resulting from them, are caused by the development in it of minute living organisms.

II. These living organisms are (with some few exceptions) always introduced from outside the body, either by the hands or instruments of the operator, by floating particles of dust, by lotions or dressings, or in some similar way.

III. There are means at our disposal, such as the employment of certain germicidal agents, superheating, &c., by which the vitality of these organisms may be destroyed, and their entrance into a wound in an active state effectually prevented, or their influence combated if they be present.

**APPLICATIONS.**—In order to carry out an antiseptic operation it is necessary:—

1. To purify the skin of the region to be operated upon, for a considerable distance beyond the part which will be occupied by the wound.

2. To purify the hands of the operators, the instruments, sponges, and everything coming in contact with the wound; and to ensure against their defilement until the operation is completed.

3. To prevent any living septic organisms falling upon the wound during an operation, or to destroy their vitality before the dressing is put on.

4. To apply a dressing containing a store of some antiseptic material; and to change it before this material has been so much washed away by the discharges that a track is left along which septic organisms may be able to spread from the edge or surface of the dressing to the wound.

**MATERIALS EMPLOYED.**—In the present work it is necessary to deal only with generalities, because the details of the treatment are numerous, somewhat complex, and ever changing. Still, it is deemed advisable to add a few words on the concrete side of the question, particularly with respect to the antiseptic substances at present commonly in use, and their chief characteristics.

**1. Carbolic Acid.**—This is occasionally used pure (liquefied by adding 6 to 10 per cent. of water to the crystals) to purify some already septic wounds; or a 1 in 20 watery solution is used for purifying the skin of the part to be operated upon, the hands and

instruments of the operator, or in the spray, if that be ever employed. A 1 in 40 watery solution may be used for the sponges, or for bathing the wound during the operation and at the changing of the dressing; but it is less trustworthy and less convenient than a weak mercurial solution. Carbolic acid gauze is not so much used as formerly, but in some cases is still the best dressing at our disposal. A solution of 1 in 10 in oil is useful for lubricating catheters.

Carbolic acid has the advantages of being very diffusible, and of having an attraction for oily and greasy substances, and hence a superiority over mercurial salts for application to the skin; it does not blacken steel or silver instruments. It has the disadvantage of being irritating, and hence causing more effusion of serum than do the mercurial salts. Dressings containing carbolic acid must be kept in airtight boxes.

**2. Mercurial Salts.**—(a) *Bichloride of mercury* is employed in solutions of 1 in 500, 1 in 2000, or still weaker. The 1 in 500 solution is used after the carbolic acid lotion, for purifying the skin; for purifying an already septic wound; or as a final application to an aseptic wound at the end of an operation. The 1 in 2000 solution is used for the sponges, and for application to a wound during an operation and at the changing of the dressings. Mercuric chloride forms an insoluble albuminate when mixed with blood; hence the solutions are practically much weakened immediately they come into contact with a bleeding surface. Its advantage is its high germicidal quality; its disadvantages are that a 1 in 500 solution applied for some time to the skin causes vesication, and that if mixed with tap water a large proportion of the salt is thrown down as insoluble chlorides. The latter difficulty is overcome by adding a small quantity of chloride of sodium; or by substituting for the simple corrosive sublimate, the very soluble but decidedly irritating *sal alambroth*, a double chloride of ammonium and mercury, which for a while was very largely employed, but is now almost superseded. It is thus obvious that mercuric chloride alone is not a very suitable substance for the active principle of a permanent dressing. Weaker solutions—1 in 10,000 and 1 in 40,000—are excellent for irrigating abscess cavities, or wounds during the process of suturing.

(b) *Biniiodide of mercury* has many of the characteristics of the bichloride; but it does not form so copious a deposit of insoluble albuminates when added to blood. It is more irritating, and not at present much used in the pure state.

(c) *Cyanide of mercury*, in the form of a kind of double salt with *cyanide of zinc*, is the essential constituent of the latest antiseptic dressing introduced by Lister. This is very insoluble and very unirritating, but

at the same time powerfully antiseptic, and at present promises to take the place of all other materials for a permanent dressing.

3. **Boric Acid** is a mild and not very trustworthy antiseptic. It is, however, useful either in the form of boric lint, boric ointment, or boric lotion (a saturated watery solution) for application to superficial granulating surfaces. It is very unirritating.

4. **Salicylic Acid** is not a very powerful antiseptic. It is used in the form of salicylic wool, of various strengths, as an external dressing outside one containing some more potent material.

5. **Iodoform** is a powerful deodorant, but its antiseptic qualities are not great. It is used with great benefit in the form of crystal or powder to stinking wounds.

6. **Chloride of Zinc** in solution—gr. xl to ʒj—is invaluable as an application to surfaces which it is necessary to leave exposed to the air, such as those of the mouth or rectum, or for purifying septic cavities. It usually prevents septic changes for two or three days.

This list might be multiplied almost indefinitely, and would have to include various coal-tar products, such as creoline, which is good for application to ulcers, naphthaline, &c., oil of eucalyptus, thymol, iodol, iodine, and countless others, but it is impossible to mention in detail more than those most commonly in use.

USE.—(1) The steps of a simple operation, where the skin is unbroken, such as the removal of a tumour or the opening of an abscess, will now be described as an illustration.

The skin of the part to be operated upon, and the hands of the operator, are washed first with soap and water, then with 1 in 20 carbolic acid solution, and then with 1 in 500 sublimate solution. The instruments should have been placed some time previously in a flat tray filled with carbolic acid solution 1 in 20; and a large supply of sublimate solution, 1 in 2000, should be prepared, in which the sponges are to be washed out, and the hands of the surgeon and his assistants dipped from time to time. It is convenient to surround the part to be operated upon with towels wrung out of one of the antiseptic solutions, so that instruments may not be thoughtlessly laid down upon an unpurified surface. During the operation it is advisable to irrigate the wound occasionally with some of the 1 in 2000 sublimate solution by means of a sponge, and it is an additional safeguard, at the end of the operation, to sponge it over with some of the 1 in 500 sublimate solution. Finally during the application of the sutures some of the 1 in 2000 solution should be dropped over the wound from a wet sponge or an irrigator; and this is to be kept up until the incision is covered by the deep part of the dressing.

Before changing an antiseptic dressing, scissors and forceps, and a piece of rag or wool should be placed in a bowl of the antiseptic solution preferred, and the fingers of the surgeon should be purified. As soon as the deep part of the dressing is removed, some of the solution should be dropped upon the wound by means of the rag or wool, and the process should be frequently repeated until the deep part of the new dressing is in position.

By most surgeons the spray has now been completely abandoned; some still employ it throughout their practice. The present writer reserves its use for cases like those of empyema, where large volumes of air, possibly bearing particles of dust, are sucked into an inaccessible cavity.

(2) *If the skin have been previously injured*, as in a compound fracture, or in abscesses which have burst, the strongest antiseptic solutions must be applied with great vigour to the exposed surface, their influence being often advantageously supplemented by the use of the sharp spoon or the nail-brush.

Besides these matters, thus briefly hinted at, the acceptance of the antiseptic principle has effected marvels in the improvement of the hygiene of hospital wards, inasmuch as septic wounds are now dressed at frequent intervals with the most suitable antiseptic and deodorising dressings, the discharges not being allowed to collect and fester as in former times beneath putrid poultices and water-dressings. And it should effect still more when it is thoroughly carried out in all the details of the sick-room, by proper ventilation, by the methodical purification of bed-pans, catheters, urinals and other utensils, and in a host of other ways too numerous to mention.

RICKMAN J. GODLEE.

**ANTISPASMODICS** (*ἀντι*, against, and *σπᾶσμα*, a spasm).—DEFINITION.—Medicines which prevent or allay spasm.

ENUMERATION.—Antispasmodics may be arranged in groups as follows:—Valerian, Valerian Acid and its salts; Musk, Castor, Asafœtida, Sumbul and Galbanum; Camphor, Brominated Camphor, Oil of Amber; Ammonia and its Carbonate; Alcohol, Ether, Acetic Ether, Chloroform, Nitrite of Amyl; Bromide of Potassium, Bromide of Ammonium; Conium, Lobelia, Opium, Gelsemium, Indian Hemp, Belladonna, Stramonium; and the Essential Oils. As adjuvants may be mentioned—Cold Baths, moderate Exercise, Friction, Heat, and Moisture; and also Quinine, Arsenic, Zinc, and Silver.

ACTION.—Certain nerves and nerve-centres, when excited, produce contraction of voluntary or involuntary muscular fibres; other nerves and centres arrest movements; and by the combined action of these two systems the motions of the various contractile struc-

tures in the body are regulated, and subordinated to the requirements of the organism as a whole. Excessive contraction or spasm of one part of the body may therefore arise either from excessive action of the motor, or deficient action of the inhibitory centres. Spasm may affect the involuntary muscular fibres of the intestines—as in colic; of the vessels—as in some forms of headache, and in vaso-motor neuroses of the uterus and bladder; of the bronchi—as in spasmodic asthma; of single voluntary muscles, or groups of muscles—as in various forms of cramp; or it may involve the muscular system generally—as in tetanus, epilepsy, and hysteria. Antispasmodics may act by lessening the irritability of motor centres, as, for example, bromide of potassium and conium; or by stimulating those portions of the nervous system which restrain and co-ordinate movements, as alcohol probably does. There are no direct experiments to show the action of antispasmodics on the inhibitory centres; but it seems probable that they have such an action, although it may not be confined to these parts alone. Thus small doses of alcohol and ether, which stimulate the nervous system generally, and usually increase motor activity, will restrain and co-ordinate excessive muscular action, as in colic, nervous agitation, trembling, and hysteria. It is at present impossible to localise the part of the nervous system affected by valerian, asafetida, and other drugs of this class. As spasms occur when the nervous system is deficient in power, nervine and general tonics, such as quinine, zinc, and iron, are often found to be useful adjuvants.

**USES.**—In such convulsive diseases as epilepsy, laryngismus stridulus, and infantile convulsions, bromide of potassium is the most powerful antispasmodic; in hysteria—valerian, asafetida, and the bromides; in chorea—arsenic, conium, copper, and zinc; in spasmodic asthma—lobelia and stramonium; in spasm of the blood-vessels—nitrite of amyl. In all spasmodic affections, cold baths or sponging, exposure to sunlight, moderate exercise, and a plain but nutritious diet should be employed; and late hours, a close atmosphere, exhausting emotions, or excessive bodily or mental work should be avoided.

T. LAUDER BRUNTON.

**ANTRUM, Diseases of.**—See NOSE, Diseases of.

**ANURIA.**—Absence of urination, whether from suppression or retention of urine. See MICTURITION, Disorders of; and URINE, Suppression of.

**ANUS, Diseases of.**—The principal affections of this part are:—1. Congenital Abnormalities; 2. Epithelioma; 3. Irritable Sphincter Ani; 4. Irritable Ulcer; 5. Prolapsus; 6. Prurigo; and 7. Tumours and Ex-

creescences. For Fistula in Ano see RECTUM, Diseases of.

1. **Congenital Abnormalities** (*atresia*) may be classed as follows:—1. Imperforate anus without deficiency of the rectum. 2. Imperforate anus, the rectum being partially or wholly deficient. 3. Anus opening into a *cul-de-sac*, the rectum being partially deficient. 4. Imperforate anus in the male, the rectum being partially deficient, and communicating with the urethra or neck of the bladder. 5. Imperforate anus in the female, the rectum being partially deficient, and communicating with the vagina or uterus. 6. Imperforate anus, the rectum being partially deficient and opening externally in an abnormal situation by a narrow outlet. 7. Narrowness of the anus. These imperfections can be remedied, if at all, only by operation.

2. **Epithelioma.**—The anus, like other parts where a junction takes place between the skin and mucous membrane, is liable to epithelioma. It is easily recognised by the ordinary characters of the sore. Warty growths and flaps of skin at this part are subject to this form of degeneration. The treatment applicable to this disease is to remove the growth by excision.

3. **Irritable Sphincter Ani.**—In this complaint the anus is strongly contracted and drawn in by the action of the sphincter. Any attempt to examine the part produces spasm, and the finger passed through it is tightly grasped as if girt by a cord. In cases of old standing the muscle becomes hypertrophied, and forms a mass encircling the finger like a thick unyielding ring. This state is the source of serious trouble in defecation, owing to the expulsive power of the bowel being insufficient to overcome the impediment caused by the muscle to the passage of the feces. Irritability of the sphincter, independent of fissure or ulcer, occurs generally in hysterical females, and is relieved by mild laxatives, the local application of cocaine or an opiate ointment, and the occasional passage of a bougie coated with a sedative ointment.

4. **Irritable Ulcer.**—This is a small superficial sore, situated just within the circle of the sphincter, usually at the back part, commonly known as *fissure*, from its appearance in the contracted state of the part. The feces passing over the sore excite spasm of the muscle, and cause a sharp burning pain which lasts for two or three hours. The distress often does not come on till an interval of ten minutes or more has elapsed after defecation. The pain is sometimes so acute that patients resist an action of the bowels, and allow them to become costive. The irritable ulcer occurs usually in middle life, and is more frequent in women than in men. It seldom gets well under the influence of local applications, but an incision through

the centre of the sore sets the muscle at rest, and allows the part to heal. Forceful dilatation of the sphincter with the fingers under an anæsthetic is a very effective mode of treatment. When the suffering is moderate, a cure may be attempted by giving a laxative to ensure soft evacuations, by enjoining rest in the recumbent position, and by the application of an ointment containing morphine, belladonna, or cocaine.

5. **Prolapsus.**—See RECTUM, Diseases of.

6. **Prurigo.**—Itching, though a common symptom in disorders of the lower bowel, may occur as a distinct affection, a neurosis liable to paroxysms. It is caused by worms in the rectum, and by congestion of the hæmorrhoidal veins. Patients suffer more after taking stimulating drinks and when heated in bed. The itching is extremely teasing and annoying, especially at night, keeping the sufferer awake for hours. Friction aggravates the mischief, excoriates the skin at the margin of the anus, and causes it to become dry, harsh, and leathery. As regards treatment, stimulants and condiments are to be avoided. The bowels should be regulated, and the part should be washed with soap and water after each evacuation. Every effort should be made to avoid friction. Cotton-wool should be used after defecation in the place of paper. A piece of cotton-wool soaked in oxide of zinc lotion may be kept applied to the anus, or the part may be smeared with some mercurial ointment, such as one of calomel (ʒj) and vaseline (ʒj). Lotions of cocaine and glycerine, of borax and morphine, or of carbolic acid, are often efficacious. In weak persons quinine and arsenic help the cure.

7. **Tumours and Excrescences.**—Besides the flaps and folds of integument consequent on external piles, tumours of a fibrous texture sometimes form in the subcutaneous areolar tissue, which as they increase become pedunculated. They are usually small in size, lobulated, and have a firm feel. These growths may be easily and safely removed by excision. *Papillomata* are liable to be developed around the anus, and sometimes grow so abundantly as to constitute a large cauliflower-looking excrescence. They then form projecting processes of various sizes, densely grouped together, with their summits isolated, expanded, and elevated on narrow peduncles. They give rise to a thin, offensive discharge. They probably originate in want of cleanliness. In some persons there is so strong a disposition to the formation of warts, that it is difficult to prevent their growth. They require to be removed by excision, the quickest and most effectual mode of treatment. Flattened growths from the skin, commonly called *mucous tubercles*, a secondary result of syphilis, are liable to occur around the anus. They yield readily to the

local application of mercury and general specific treatment. See MUCOUS TUBERCLES.

T. B. CURLING. FREDERICK TREVES.

**ANXIETY.**—Anxiety or distress, whether subjectively felt, or expressed in the features, attitude, or general behaviour. The term is also especially associated with a peculiar sensation experienced in the region of the heart. See PRÆCORDIAL ANXIETY.

**AORTA, Diseases of.**—The diseases to which the aorta is liable may be thus considered:—1. Aortitis, Acute and Chronic; 2. Atheroma; 3. Primary Fatty Degeneration; 4. Primary Calcification; 5. Coarctation; 6. Simple Dilatation; and 7. Aneurysm.

1. **Aortitis.**—*Acute Aortitis (Hyperplastic Arteritis)* is exceedingly rare. It may result from the direct irritation of an atheromatous aorta by a thrombus or an embolus, in persons of gouty diathesis; but has never been observed as an extension of acute endocarditis. The *morbid changes* consist in hyperæmia, with thickening and softening of the coats of the vessel, and deposit of fibrin upon its internal surface. The ascending portion of the arch is the part most frequently affected. The *symptoms* are acute substernal pain, with oppression, palpitation, quick and feeble pulse, and elevated temperature. With these symptoms may be associated a harsh systolic murmur, originating at the seat of inflammation, and transmitted to a distant part of the aorta.

*Sub-acute and Chronic Aortitis (Endarteritis).*—These are the usual forms of inflammation of the aorta. The disease may be general, arising from a blood-dyscrasia such as gout or syphilis, from pyæmia, or from various septic agents; but it is usually limited to a definite portion of the vascular surface, being the result of local irritation.

**ÆTIOLOGY.**—Excessive and continued strain of the vascular walls is, according to its degree, the most frequent cause of sub-acute and chronic aortitis. Hence, the portion of the arterial system most directly affected by the impulse of the left ventricle, namely, the arch of the aorta, is that in which inflammatory irritation is first, and often exclusively, exhibited. Labour of any kind requiring great and repeated muscular effort whilst the breath is held, must necessarily subject the aorta to extreme tension, partly through the obstruction arising from the pressure of the contracted muscles upon the subjacent arteries, and partly from the back-pressure in the distended veins. Hence, sledgers, rammers, ship-porters, &c., are those who most frequently suffer from the effects of aortitis. As a necessary result of such efforts the left ventricle soon becomes hypertrophied, and the evils arising from vascular tension are thereby proportionately increased. Furthermore, the free use of alcoholic stimulants, in which such labourers

habitually indulge, contributes to the same result. The British soldier has been especially liable to the evils above sketched, owing to a vicious system of forced drill with a breathing-capacity diminished by faulty construction of his dress and accoutrements.<sup>1</sup>

**ANATOMICAL CHARACTERS.**—Sub-acute aortitis occurs in disseminated patches, and involves all the coats of the vessel. These are infiltrated with leucocytes at an early period; become soft and tumid, assuming a bluish-white tint; and, owing to loss of normal elasticity, project outwards, thus causing unevenness, or pitting of the internal surface. In the aorta the inflammation is usually primary; but exceptionally it may be produced by the mechanical irritation of an embolus derived from an inflamed focus. Inflammatory softening is a frequent cause of aneurysm at all periods of life; and in the young it is the ordinary precursor of that disease.

In chronic aortitis, which is the most common form of the disease, the internal coat is alone involved. The outer portion of the intima exhibits the result of irritation in the abundant production of new cells. These cells occupy the fusiform spaces between its lamellæ, and, gradually distending them, ultimately project the internal and unaffected portion of the tunic into the lumen of the vessel. The prominence so caused is comparatively solid, presents a faint bluish tint, and constitutes the condition described as *sclerosis*. The thickened intima is prone to undergo granular and fatty disintegration, and the consecutive change is called *atheroma*. Dr. Mott has suggested that this process may be partly accounted for by the fact that the deeper layers of the inner coat of the larger arteries are probably nourished by the *vasa vasorum*, through the fenestræ of the elastic lamina. Where these nutrient vessels are diseased, as in Bright's disease and syphilis, where the walls are often greatly thickened—possibly almost obliterated, the parts indicated are imperfectly supplied with blood and undergo degenerative changes.

**2. Atheroma.**—This morbid condition is most common in the first portion of the aorta.

**ANATOMICAL CHARACTERS.**—Atheroma commences with inflammatory overgrowth by multiplication of the cells of the outer portion of the intima, as described in a preceding paragraph. The neoplasts, from their situation, readily undergo fatty change and caseation; the septa of unaltered tissue intervening between them soon lose their vitality and are absorbed; and the disease thus spreads, while it advances by the same process towards the internal surface of the

vessel. Examined microscopically, atheromatous matter is found to consist of fat-granules, crystals of cholesterin, and tissue-débris. At an early stage the disintegrating process makes its way towards the lumen of the vessel, and a rupture of the innermost layers of the intima takes place, forming what is termed an *atheromatous ulcer*. From this may result a false aneurysm.

In the most advanced stage of atheromatous change many patches undergo calcification by deposit of lime-salts in the altered cells. The calcareous lamellæ so formed, being concentric with the vessel, and contracting by loss of their liquid constituents, may erode the intima by their sharp edges. From the injury thus inflicted aneurysm may arise, or interstitial thrombosis, by which the vessel may be entirely blocked and gangrene of the extremities produced. In consequence of the foregoing changes the vessel loses its elasticity and becomes dilated; its internal surface is mottled with yellow or fawn-coloured plaques and patches of various sizes, being also rough, spiculated, and fissured; and thus the condition described by Virchow under the name of *endarteritis chronica deformans* is established.

The uric acid and oxalic acid diatheses favour these changes, not only by stimulating the minute arteries to contract, and so raising the blood-pressure in the larger vessels, but likewise by furnishing material for cretification. The subjects of constitutional syphilis are liable to 'ulcerated steatomatous' (atheromatous) changes of the intima.

**3. Primary Fatty Degeneration.**—Virchow has described, under the name of *fatty erosion*, a form of fatty degeneration of the cells of the internal coat, unpreceded by inflammation, commencing on the free surface, and gradually extending outwards. The internal surface of the vessel is marbled with minute yellow dots, which are groups of fatty cells; these undergo liquefaction; and disintegration of the internal coat, followed by aneurysm, is the usual result.

**4. Primary Calcification.**—Exceptionally, in the distant portions of the aorta the muscular fibre-cells of the middle coat are liable to calcification, as a remote result of endarteritis. Owing to the transverse arrangement of the calcified cells, fissuring of the middle coat under the pressure of the blood-current, and dissecting aneurysm, are ordinary results of this change. Finally, the entire middle coat, and even all three coats of the artery, may be infiltrated with lime-salts as a primary change. This is most probably due to precipitation of these salts from the congested *vasa vasorum*, in consequence of the escape of their ordinary solvent, carbonic acid.

**5. Coarctation or Stenosis.**—This condition may be either *congenital* or *acquired*.

<sup>1</sup> Also by the constrained and fixed position in which the walls of the chest are placed when the shoulders are forced backwards, with the view of producing the appearance of an expanded chest.  
—E.

*Congenital* stenosis of the aorta is most frequently located at the point of junction of the *ductus arteriosus*, and is of very limited extent; in many cases presenting the appearance of a linear constriction, or of a perforated diaphragm. In a few examples, the vessel, at the seat of contraction, has been entirely closed and converted into a ligamentous cord. On the cardiac side of the constriction the aorta is dilated, and often thickened and atheromatous, whilst on the distal side it is reduced in calibre as far as the junction of the collateral vessels. Congenital stenosis of the aorta is compatible with life of moderate duration. In 24 out of 38 cases analysed by Dr. Peacock the age attained varied from twenty-one to fifty years. The diagnosis of the condition rests mainly on disproportionate pulsation of the arteries arising from the aorta on the cardiac, as contrasted with those on the peripheral side of the obstruction; and on the enlargement of the collateral vessels, namely, the transverse cervical, internal mammary, and intercostals. The ordinary consequences are exhibited in dilated hypertrophy of the left ventricle, and relative inadequacy of the aortic valves. Death usually occurs from progressive debility and failure of the left ventricle; from pulmonary congestion; from rupture of the aorta or one of its dilated branches; or from acute inflammatory change in the heart or aorta. Congenital stenosis of the entire arch may result from imperfection of the inter-ventricular septum or patency of the foramen ovale allowing the blood to take an exceptional course.

Congenital narrowing of the aorta *throughout its course* was described by Rokitansky and Virchow. The latter pointed out its existence in women who have through life presented the signs of persistent chlorosis (see *CHLOROSIS*). In many of these cases valvular disease of the heart, especially stenosis of the mitral orifice, has been observed.

In the *acquired* form, stenosis of the aorta at any portion of its course may result from inflammatory thickening or calcareous change of the coats of the vessel, followed by thrombosis; or it may follow the natural cure of an aneurysm. The aorta may be much reduced in calibre without being disproportionately narrowed. Such will be its condition in connexion with mitral inadequacy in early childhood, should the patient survive a few years. In such cases the left ventricle will have become dilated and hypertrophied, and a marked disproportion will be observed between the force of cardiac and that of radial pulsation. This circumstance, taken in conjunction with the age of the patient, the existence of disease at the mitral orifice, and hypertrophy of the left ventricle, would warrant the positive diagnosis of narrowing of the aorta.

6. **Simple Dilatation.**—This disease

consists in a uniform enlargement of the vessel or a portion of it, from impairment or loss of its normal elasticity; and depends primarily upon arterial obstruction or resistance beyond its seat, and directly upon consecutive hypertrophy of the left ventricle. The continued tension, to which the walls of the aorta are subjected between these two opposing forces, necessarily leads to progressive impairment of nutrition and loss of elasticity in its middle coat. The immediate consequence of this change is exhibited in further hypertrophy of the left ventricle; and its remote effects in still further impairment of nutrition and deterioration of tissue in the vascular tunics, through the increased tension to which they are now exposed. No elementary change of structure is, however, discoverable. Simple dilatation of the aorta commences in the ascending portion of the arch, and to this it is usually limited; but it occasionally extends into the transverse portion. The other portions of the vessel are never dilated, except in association with atheromatous change. The condition under notice is manifestly in close relationship with inflammatory irritation of the vessel. It has, however, a distinct pathological existence, analogous to that of the early stage of vesicular emphysema of the lung.

No morbid results, with a single exception, are directly traceable to simple dilatation of the aorta. But, should the dilatation extend into the transverse portion of the arch, and engage especially its superior wall, the primary branches may become tortuous, and exhibit abnormal pulsation in the neck, simulating aneurysm. In a noteworthy example observed by the late Dr. Hayden the existence of this pulsation on *both* sides of the neck, and the facility with which it was arrested by forcibly extending the neck and shoulders, and so unbending the vessels, sufficed to establish the diagnosis. Tortuosity of the cervical arteries, dependent upon a local dilatation of the aorta, may be confined to one side of the neck.

Simple dilatation of the aorta most frequently occurs in connexion with the contracted or granular form of chronic renal disease. It may, however, likewise arise from simple functional hypertrophy of the left ventricle dependent upon habitual vascular excitement; or from dilated hypertrophy consecutive to inadequacy of the aortic valves.

7. **Aneurysm.**—*ÆTIOLOGY AND PATHOLOGY.*—Aneurysm of the aorta is essentially a disease of the middle period of life. Of 92 cases observed or analysed by the late Dr. Hayden, 60 occurred between the ages of thirty and fifty years; 12 over fifty; and 5 under thirty years. Thus, whilst deterioration of the arterial coats, as typified in atheroma, is most common after the age of sixty, one of its ordinary consequences, aneurysm, belongs to an earlier period of life. The apparent discrepancy

may be explained by the more frequent employment of men under fifty in severe labour, and their greater capacity for extreme muscular effort earlier in life, the condition of the arterial wall which favours aneurysm having been already established. Aortic aneurysm is more common amongst males than females in the proportion of about 8 : 1—a difference no doubt due to the more active and laborious habits of the male sex. Soldiers, mechanics, and porters suffer from it in larger proportion than persons of other callings; and in most instances the first symptoms of aneurysm of the aorta may be traced to a great muscular effort, involving vascular strain, or to a severe shock or blow, causing a direct contusion.

Aneurysm of the aorta is always consecutive to disease of its coats. Inflammatory softening, atheroma, and calcification are the usual antecedent conditions, and in exceptional instances primary fatty or calcific transformation of the internal and middle coats; whilst a definite overstrain or a direct contusion of the vessel is frequently the immediate cause of the disease.

**ANATOMICAL CHARACTERS.**—Aortic aneurysm may be presented under the following forms, viz., (a) *true*; (b) *false (circumscribed, and diffused or consecutive)*; (c) *dissecting*; and (d) *varicose*.

a. *True aneurysm* of the aorta is rare; it may be either fusiform or sacular. It is essentially transitional, leading to the false variety of the disease; and differs from simple dilatation of the aorta only by its sharp limitation, and by the existence of inflammatory products in its walls. True aneurysm never contains clots, save by incidental thrombosis; and rarely, as such, attains dimensions capable of producing extrinsic symptoms or signs. It may, however, unlike simple dilatation, be the cause of valvular inadequacy, and so give rise to a murmur of reflux at the orifice of the aorta.

b. *False aneurysm* is either *circumscribed* or *diffused*. *Circumscribed false aneurysm* (or, as it is also termed, *false aneurysm*) is the most common form of the disease in connexion with the aorta. It is necessarily confined to a portion of the circumference of the vessel, the yielding of which relieves the remainder from extra tension. Hence, it is usually sacular in general outline; but, owing to unequal resistance at different points of its surface, it may, and commonly does, present one or more secondary prominences. The internal and middle coats are usually broken;—the adventitia supplemented by the surrounding structures, more or less condensed, forming the sac.

Disintegration of the inner coat, already in a state of atheromatous change by mechanical strain or vascular tension, is ordinarily the immediate cause of false aneurysm. Ulceration of the intima from fatty erosion may also cause it, as well as progressive arterio-

sclerosis. Rupture of the coats of the vessel by mechanical strain is usually indicated by definite symptoms of the utmost significance, namely, a feeling of something having given way within the chest or abdomen; followed by faintness often amounting to syncope, dyspnœa, palpitation, and occasionally hæmoptysis. These symptoms of shock usually subside within a period of one to two hours, but the patient is thenceforward incapable of his accustomed exertion, being easily put out of breath, and distressed by excitement or rapid movement, especially that of ascent; there is likewise a fixed pain at some point of the chest, back, or abdomen. A fusiform false aneurysm may become 'invaginating' by abruptly expanding and ensheathing the artery at its proximal or distal side, or in both these situations.

*Diffused false aneurysm* (or, as it is otherwise called, *diffused aneurysm*, or *consecutive aneurysm*) is produced by escape of blood from the artery, and its diffusion to a greater or less extent amongst the surrounding structures, according to their previous condition or anatomical arrangement. It may be the result of mechanical violence by strain or shock to the artery in a previously diseased condition; or of progressive disintegration of the sac of a circumscribed aneurysm. In the latter case the diffusion of the extravasated blood is usually limited by antecedent adhesive inflammation of the surrounding parts, where, as in the transverse portion of the arch of the aorta, the position of the aneurysm is favourable to that process. Under ordinary circumstances diffused false aneurysm of the aorta cannot occur within the pericardium, owing to the isolation of that portion of the vessel, and the fragile structure of its serous investment. Hence, a yielding of the sac proper in this situation is, in most instances, followed by instant death from hæmorrhage into the pericardium and paralysis of the heart. In a few recorded cases, owing to previous adhesion of the pericardium, the patients survived rupture of the sac in this situation for several days.

Diffused false aneurysm of the abdominal aorta is frequently formed by irruption of blood into the retro-peritoneal tissue, between the layers of the transverse meso-colon or the mesentery, or into the fibrous envelope of the psoas muscle. When the aneurysm grows backwards the sac is quickly eroded by pressure against the vertebræ, the naked and carious surface of which then forms its posterior boundary. Diffusion in such cases rarely occurs until the vertebræ are entirely absorbed; the blood may then escape into the spinal canal, causing general paralysis and immediate death. Diffusion may also occur amongst the muscles and areolar tissue of the loins, or behind the diaphragm into either pleural cavity, usually the left. Any

portion of the aorta outside the pericardium may be the seat of diffused false aneurysm, but the transverse portion of the arch and the abdominal aorta are the parts most frequently affected.

*Consecutive false aneurysm*, consisting in a primary bulging of all the coats of the artery, the internal and middle coats having subsequently given way, constitutes the ordinary form in which false aneurysm originates: it therefore demands no further notice here.

*c. Dissecting aneurysm* consists in a breach of the internal and middle coats, and a subsequent detachment of these from the external tunic, by the force of the blood-current, to a variable extent over the length and circumference of the vessel; or in a splitting of the middle coat by the same agency. This form of aneurysm is of two kinds—that with a single aperture through which the blood enters the abnormal channel and returns to the artery; and that which exhibits two openings, one by which the blood escapes from, and another through which it re-enters the vessel. The former is the more usual variety of dissecting aneurysm; and it is likewise the more grave, because liable at any moment to terminate fatally by rupture of the external coat.

Any portion of the aorta may be the seat of dissecting aneurysm; the ascending part of the arch is most frequently affected, and next in order of frequency comes the abdominal aorta. The primary lesion consists in a transverse rent of the internal and middle coats; when this is close to the heart, the outer or the anterior wall of the vessel is its usual site, and detachment of the tunics rarely extends beyond the ascending portion of the arch, and seldom engages more than a limited area of its circumference. In this situation, too, an aperture of re-entrance is rarely formed, the disease usually terminating by rupture of the external coat within the pericardium. When, on the contrary, the second curve of the arch, or any portion of the aorta beyond this point, is the seat of primary lesion, separation of the coats is usually found to extend along the remainder of the length of the vessel and over the whole or greater part of its circumference, whilst the blood has re-entered through an opening in one or both common iliac arteries.

The establishment of a second aperture of communication with the artery is an attempt at 'natural cure;' and when this happens the patient may survive for many years. Amongst the eccentricities of dissecting aneurysm may be mentioned detachment of the laminated clot from the walls of the sac proper, and subsequent escape of blood by rupture of the latter; and separation of the mucous from the muscular coat of the œsophagus, with irruption of blood into the stomach. Dissecting and ordinary false aneurysm may

coexist, the former being usually a consecutive lesion, and the immediate cause of death.

*d. Varicose or anastomosing aneurysm* consists in a direct communication between an aneurysm of the aorta and (a) one of the chambers of the heart; (b) the pulmonary artery or one of its branches; or (c) one of the venæ cavæ or innominate veins. This form of the disease is necessarily consecutive, and usually late as to the period of its development. In the great majority of recorded examples the primary aneurysm was connected with the ascending portion of the arch, and in a large number it arose from one of the sinuses of Valsalva. The communication, with few exceptions, is formed with one of the chambers of the heart, the pulmonary artery, or the descending vena cava. Of the cavities of the heart, the right ventricle is most often implicated; next in order is the right auricle; then the left ventricle; and lastly the left auricle. The pulmonary artery and the descending cava have been frequently involved, as might have been inferred from their close relationship to the ascending aorta; the innominate veins in fewer instances, and only when the aneurysm engaged the upper portion of the arch. An aneurysm of the abdominal aorta has communicated with the inferior vena cava in a few cases. In the process of formation of a varicose aneurysm of the aorta, the apposed surfaces are agglutinated by adhesive inflammation; and the composite septum is subsequently eroded by progressive absorption, or suddenly rent by the force of the arterial current. The immediate effects of communication are engorgement and increased tension of the receiving chamber or vessel; diminished blood-current and vascular tension in the aorta and its branches; and admixture of arterial with venous blood when the right side of the heart or one of the great veins is the seat of discharge. The special symptoms and signs by which the disease is characterised have direct reference to these results. Death has followed most rapidly in those cases in which the aneurysm had established a communication with the pulmonary artery or the left auricle.

*Effects upon the left ventricle.*—Hypertrophy of the left ventricle cannot be regarded as a consequence of aneurysm of the aorta. The association, when it exists, is accidental; hypertrophy depending upon antecedent or consecutive disease or inadequacy of the aortic or the mitral valves, chronic atheroma of the aorta, granular degeneration of the kidneys, or excessive functional activity.

**SYMPTOMS.**—The symptoms of aortic aneurysm may be discussed under the three heads of: (a) Pain; (b) Excentric Pressure; and (c) Tumour.

*Pain.*—The pain of aneurysm is of two kinds, *intrinsic* and *extrinsic*. The former is due to subacute inflammation and tension of

the sac, and varies with intra-vascular pressure. It is dull, aching, and localised, and promptly alleviated by measures which depress the circulation or reduce local tension. Extrinsic pain usually arises from pressure upon adjacent nerves, and may be direct or reflex. This kind of pain has the characters of a diffused and aggravated neuralgia, being paroxysmal, and wandering to a greater or less extent over the back, chest, shoulders, arms, abdomen, and thighs. In the abdomen, when due to pressure upon the splanchnic nerves or tension of the solar plexus, it is of the most excruciating character. The extrinsic pain of aneurysm may be fixed and boring. When of this character, it is usually located in the back, and arises from progressive absorption of the vertebræ.

*Excentric Pressure.*—The parts affected by the pressure of an aneurysm, and the symptoms thereby developed, vary according to its situation and the direction of its growth. Parts which are exposed to counter-pressure, or are otherwise fixed, suffer most; whilst those which are flexible or movable are less injuriously affected. The symptoms have reference to the respective functions of the organs or structures pressed upon; whilst their severity is in direct proportion to the importance of those functions, and the degree of pressure exercised. Structures subjected to the remittent pressure of an aneurysm are slowly removed by absorption, but between the sac and the resisting surface union has been previously established by adhesive inflammation. Hence the sac itself is at the same time absorbed, and escape of blood will inevitably occur where further resistance is not presented. When serous cavities are laid open by this process, entrance is effected by a rent; and if the cavity be large, *e.g.* the pleura or the peritoneum, death by hæmorrhage, almost instantaneous, is the result; in case of irruption into the pericardium or the spinal canal, death occurs with equal rapidity from compression and paralysis of the contained organ. Communication with a mucous canal or with the cutaneous surface is effected by a process of sloughing, and bleeding occurs by 'leakage,' in variable quantity and at uncertain intervals, till the slough is finally detached, when death by copious hæmorrhage immediately ensues. The irruption of an aneurysm into a gland-duct, such as the ureter or one of the biliary passages, may be fatal by obstruction and suspended secretion, the duct and its tributaries having been blocked by coagulum. Pressure upon the thoracic duct proves slowly fatal by inanition. When the aneurysm opens into a vein, a varicose aneurysm, characterised by special symptoms and signs, and of greater or less gravity according to its situation, will be the result.

The symptoms of nerve-pressure vary according to the nerves affected. Thus, pressure upon the roots or branches of sentient

nerves is attended with neuralgic twinges or paroxysms referred to the seat of their peripheral distribution, and, when the pressure is extreme, with numbness in the same situation. Irritation of motor nerves is indicated by spasm or paralysis, according to the degree of pressure, of the muscles supplied by them. Irritation of the cervical sympathetic, or of its cilio-motor roots, is revealed by dilatation of the pupil on the corresponding side; or, if the pressure be such as to cause paresis, by contraction of the pupil with ptosis, hyperæmia, hyperæsthesia, and elevation of temperature in the eye and corresponding side of the face. The effects of pressure upon the pulmonary and cardiac plexuses have been less precisely determined, owing to the difficulty of distinguishing the symptoms due to this cause from those which arise from direct pressure upon the trachea or bronchi, the great vessels, or the heart, or from structural disease of the heart or the coronary arteries; but that the paroxysms of bronchial spasm and of angina, so often witnessed in connexion with aneurysm of the arch of the aorta, are in some degree dependent upon pressure on the pulmonary and cardiac nerves, and occasionally are due to it exclusively, there can be no doubt. The symptoms arising from the pressure of an aneurysm upon the pneumogastric or recurrent nerve of either side have reference to the larynx, and are eminently characteristic. They are of two kinds, according to the degree of pressure; namely, those due to spasm, and those to paralysis, of the laryngeal muscles on one or both sides. They are presented under the several forms of dysphonia, aphonia, stridor, metallic cough, and paroxysmal dyspnoea. The latter is frequently of the most urgent character, and sometimes is the immediate cause of death.

Adjacent organs, such as the heart, lungs, liver, and kidneys, are occasionally displaced by an aneurysm, the direction being determined by that of the pressure, and in part also by the direction in which the organ is movable. Obstruction or occlusion of adjacent arteries, as indicated by diminished or suppressed pulsation, may likewise result from the pressure of an aneurysm. Pressure on a vein is evidenced by venous stasis distal to the seat of obstruction; upon the pulmonary artery, by engorgement of the right chambers of the heart and general venous congestion. Obstruction or occlusion of either bronchus or of one of its primary branches is evidenced by distress in breathing or shortness of breath; by bronchial breathing over the seat of compression; and by diminution or suppression of respiratory sound in the corresponding portion of the lung. Inasmuch as the symptoms of excentric pressure may be produced by a tumour of any kind, they possess, in regard to aneurysm, a diagnostic value only correlative to other and more positive evidence of that disease.

*Tumour.*—The tumour formed by an aneurysm of the aorta is fixed, smooth, and compressible. It is alternately tense and soft, in unison with cardiac pulsation; and is especially characterised by a movement of general and equal expansion, synchronous with the impulse of the heart. It is further distinguished by a remarkable liability to vary in the rate and direction of its growth, retrocession at one point coinciding with enlargement at another; a new set of symptoms being at the same time developed by its encroachment upon fresh territory. In the progress of cure the tumour may become solid, incompressible, and uneven, by deposition of laminated fibrin within the sac.

*PHYSICAL SIGNS.*—The physical signs of aneurysm of the aorta are those which may be elicited by *inspection, palpation, percussion, and auscultation.* They supply the most valuable, and, indeed, the only positive evidence of the disease.

*Inspection.*—Abnormal pulsation, and sometimes the existence of tumour, may be detected by inspection of the chest or abdomen; the signs observed being confirmed by those afforded by palpation.

*Tactile signs.*—The tactile signs of aneurysm, *impulse, fremitus, and remittent tension,* are contingent on the presence of a tumour. The impulse may be single or double. It is most frequently single, and is then always systolic in rhythm, coinciding approximately with the impulse of the heart. In character the systolic impulse is heaving and expansile; and it is diffused, in most instances equally, over the entire tumour. It is due to sudden expansion of the sac from active influx of blood during ventricular systole: hence, in those portions of the aorta which are close to the heart, it is synchronous with the apex-pulsation; but in the descending thoracic and abdominal aorta perceptibly post-systolic in time. The force of systolic impulse and the expansion of the sac will be directly as the contractile power of the left ventricle, and inversely as the deposit of coagulum within the sac. A second and more feeble impulse of diastolic rhythm is occasionally, but much less frequently, exhibited by an aortic aneurysm. This is the 'back-stroke,' or 'impulse of arrest' of authors. It coincides with the first period of ventricular diastole and the second sound of the heart, and is due to asynchronism of reaction between the aorta and the sac—that of the latter being notably later, owing to its defective elasticity. The consequence is a sudden arrest to the recoil of the sac by influx from the aorta. Diastolic impulse is therefore eminently characteristic of aneurysm.

Fremitus or thrill is of more frequent occurrence than diastolic impulse, and, when not communicated from the heart, no less distinctive of aneurysm. It accompanies the systolic impulse, and is due to the vibration

of a thin sac from an eddy in the current of influx, produced either by a spiculated condition of the orifice, or by a pendent flake of fibrin.

*Percussion-sound.*—The intrinsic percussion-sound of aortic aneurysm is absolutely dull to the extent of the tumour. Posteriorly the dullness is not sufficiently distinguishable from that of the vertebral column and muscles to be of positive diagnostic value; whilst laterally and in front, when the tumour is not in actual contact with the walls of the thorax or abdomen, it is modified or masked by the intervention of the lung or the intestinal canal. Under any circumstances, dullness *per se* cannot afford positive evidence of aneurysm, inasmuch as it may be due to a tumour of any kind, solid or liquid, in the same situation; but, the presence of a tumour having been determined, the shifting of percussion-dullness from one point to another, or its cessation where it had been previously detected, would be in the highest degree suggestive of aneurysm.

*Acoustic signs.*—These are *tone or sound, and murmur.* *Sound* without murmur is of frequent occurrence in aneurysms of the arch, but comparatively rare in those of the descending thoracic and abdominal aorta. It is usually double, corresponding in time to the sounds of the heart, but exaggerated, the second aneurysmal sound being especially intensified. The first sound is occasionally 'splashing,' and both are not infrequently of a 'booming' quality,—characters no doubt due to the density, rigidity, and great capacity of the sac.

The *murmur* of aneurysm is most frequently single, systolic, and blowing; it is occasionally double (systolic and diastolic); and still more rarely single and diastolic. As to quality, the systolic murmur may be accompanied by a musical note, 'cooing,' or shrill, and audible over the whole or only a portion of the tumour. Lastly, it may be, and usually is, of a 'buzzing' character in cases of varicose aneurysm. The essential cause of the murmur of aneurysm consists in friction of the blood against the orifice, and the production of an eddy or a 'fluid vein' within the sac. A certain force of ventricular contraction is, however, likewise necessary. Hence the not infrequent coincidence of cessation of murmur with failure of the left ventricle for some time before death. A strongly contracting ventricle, a relatively small orifice, a capacious sac, and a liquid state of its contents, supply the most favourable conditions for the production of murmur. The orifice of entrance need not be absolutely narrow; a large fusiform aneurysm, even of the true kind, with rough walls, and containing liquid blood, may yield a loud systolic murmur, as the writers have frequently observed. A murmur may be absent in an aneurysm lined by thick laminae of fibrin

through which there is a smooth channel, or in a lateral aneurysm communicating by a small orifice with the vessel.

**DIAGNOSIS.**—The positive diagnosis of aneurysm of the aorta may be made from the existence of a tumour, forming a second centre of pulsation and of sound; the pulsation being systolic, expansile, and equally diffused over the tumour, accompanied by thrill, and succeeded by a minor pulsation of diastolic rhythm; whilst the sound, single or double, and accompanied or not by murmur, is always sharp and ringing, and occasionally of a 'booming' quality. The foregoing signs are rarely all associated in the same case. Various other groupings of rational symptoms and signs would be scarcely less conclusive as to the existence of aneurysm of the aorta. General systolic expansion, thrill, diastolic impulse, and exaggerated sound, at a point more or less distant from the heart, constitute the most positive signs of the disease.

The existence of aneurysm of the aorta may be inferred with greater or less confidence from certain symptoms and signs, according to their individual or correlative value. Even negative signs, if associated with others in themselves of minor significance, may be scarcely less conclusive than the most positive evidence would be. Thus, for example, suppressed respiration with undiminished percussion-resonance on the left side of the chest, dyspnoea, hæmoptysis, fixed pain in the back, and left intercostal neuralgia—the entrance of a foreign body into the left bronchus and the existence of cancer of the posterior mediastinum having been excluded—would be all but conclusive as to the existence of aneurysm. A foreign body in the bronchus might be diagnosed from the history of a misadventure in swallowing, followed immediately by dyspnoea, hæmoptysis, and the special signs of bronchial obstruction, which, in nine cases out of ten, would be on the right side; whilst the diagnosis of mediastinal cancer would rest upon evidence eminently suggestive, namely, the presence of cancerous enlargements in the neck and axilla, and of extreme dulness over the root of the lung, without corresponding pulsation or sound. Finally, aneurysm of the aorta may be, though it very rarely is, strictly latent in regard to both symptoms and signs.

**PROGNOSIS, DURATION, AND TERMINATIONS.** The prognosis of aortic aneurysm is in the highest degree unfavourable. Recovery is, however, under favourable circumstances and appropriate treatment, quite within the range of medicine. Numerous examples of the cure of aortic aneurysm, both thoracic and abdominal, have been lately recorded.

The duration of life, in connexion with aneurysm of the aorta, varies from ten days to eleven years (Hayden), but it may be much longer. The situation and relations of the aneurysm; its complications; the con-

stitution of the sac, and the state of its contents; the previous health and present habits of the patient; and the advantages enjoyed in regard to rest and treatment—will all materially influence the prognosis, whether as to duration of life or prospect of recovery.

Death in aneurysm of the aorta may result from—(a) rupture of the sac; (b) exhaustion from pain, loss of sleep, or leakage of blood; (c) asphyxia; (d) syncope; (e) inanition; or (f) intercurrent disease. The foregoing represents the order of relative frequency of the several causes mentioned. Rupture of the sac is not, of necessity, immediately fatal. Hæmorrhage may be stayed, and life thus protracted for several days, by obstruction from the extravasated blood (itself arrested and coagulated in the surrounding tissues), or by its pressure upon the aorta on the proximal side of the sac. Rupture into one of the chambers of the heart, the pulmonary artery, either vena cava, or the innominate vein, the portal vein, or the biliary passages, is usually fatal within a very brief period; whilst rupture into one of the serous cavities in the absence of previous adhesion, into the trachea or bronchi, or into the alimentary or the spinal canal, is instantaneously fatal.

**TREATMENT.**—The treatment of aneurysm of the aorta is *palliative* and *curative*. Pain from nerve-pressure is most effectually relieved by hypodermic injections of morphine, one quarter to half a grain in solution, repeated and increased in quantity according to necessity; cocaine administered hypodermically may also be employed. The pain and oppression due to congestion of the sac and the surrounding structures is best treated by local or general abstraction of blood, or the use of an ice-bag over the tumour, combined with the use of cardiac and vascular depressants, especially iodide of potassium (20 to 30 grains every fourth hour), chloral hydrate (20 grains), veratrum viride, or aconite (5 to 10 minims of the tincture every third hour). Antipyrin (10 to 20 grains every third hour) has been found to give relief. Mechanical support by means of a well-constructed shield is likewise useful when the tumour projects externally. Fixed pain in the back, due to erosion of the vertebræ, is most effectually relieved by leeching, or by the application of a seton or issue in the vicinity of its seat.

The *curative* treatment of aneurysm of the aorta may be classified under the heads of—(a) compression of the artery, proximal or distal; (b) distal ligature; (c) absolute rest and regulated diet; (d) the use of medicinal agents promotive of coagulation within the sac; and (e) local measures—such as (1) the application of the galvanic current, the positive pole, carefully insulated, being introduced into the sac, the negative pole being placed on an indifferent part in its neighbourhood;

(2) the passage of fine iron wire, horsehair, or catgut into the sac, so as to cause coagulation. For the details of these several modes of treatment the reader is referred to the articles in this volume respectively entitled ANEURYSM; ABDOMINAL ANEURYSM; and THORACIC ANEURYSM.

THOMAS HAYDEN. C. J. NIXON.

### AORTIC VALVES, Diseases of.—

See HEART, Valvular Diseases of.

**APEPSIA** (*ἀ*, priv.; and *πέπρω*, I digest). Indigestion. See DIGESTION, Disorders of.

**APERIENTS** (*aperio*, I open).—Medicines which produce a gentle action of the bowels. See PURGATIVES.

**APHAGIA** (*ἀ*, priv.; and *φάγω*, I eat).—Inability to swallow. See DEGLUTITION, Disorders of.

**APHASIA** (*ἀ*, priv.; and *φημι* or *φάω*, I speak).—SYNON.: Aphemia; Alalia; Fr. *Aphasie*; Ger. *Sprachlosigkeit*.

DESCRIPTION.—Aphasia is the name given to a defect of speech from cerebral disease. The patient is found to be unable to utter any proposition, though his occasional distinct pronunciation of some one or two words shows that his speechless condition is not due to a mere difficulty in the more mechanical act of articulation. Moreover, the patient's intelligent manner and gestures may plainly show that he understands what is said, and is capable of thinking, even though he is quite unable to give expression to his thoughts. This kind of powerlessness as regards speech is most frequently encountered in persons suffering from right hemiplegia, though it is occasionally met with in those who are paralysed on the left side, and at other times in persons who are not hemiplegic at all.

The aphasic condition is not always, as it ought to be, clearly distinguished from that of another group of cases which may be encountered in association with some amount of hemiplegia or independently, and to which the name *amnesia* is given. The essence of this latter defect lies in the fact that the patient's speech is defective because of his inability to recall the proper words for the expression of his thoughts or wishes; and he very frequently substitutes wrong words or names in the place of those he wishes to employ, as when speaking of his 'hat,' he calls it a 'brush;' or when seeking a 'pen,' he asks for a 'knife.' In a bad case of this kind the patient may be quite unable to arrange words into a sentence capable of conveying a definite meaning, so that his speech is rendered unintelligible. Where this species of defect exists there seems to be either a defective or an inco-ordinate action of one or other of those higher cerebral centres whose function it is to translate thought into the corresponding motor acts

of speech, so that we get hesitation in the utterance of right words, or the substitution occasionally of entirely wrong words or even of a meaningless set of sounds. These amnesic or inco-ordinate defects were not at first recognised as being distinct in nature from those of an aphasic type, in which there is rather a loss than a misdirection of power in one or other of the higher centres whence the incitation to the motor acts of speech proceed. The two kinds of defects, indeed, not unfrequently coexist to some extent in the same individual.

When occurring in association with hemiplegia, aphasia varies much in intensity according to the degree of general mental impairment with which it may be combined. During the first week or ten days after the onset of such an attack the special defect may be scarcely recognisable, owing to the masking influence of the general mental impairment. But after a time such a patient may regain a considerable amount of general mental power. He may be left more or less hemiplegic, and may also present the aphasic defect to a marked degree. He readily comprehends everything that is said to him, and can often understand what he reads.

But at other times, though able fully to understand when read to, he does not seem to understand when he himself attempts to read—a disability now often spoken of as 'word-blindness.' He can, perhaps, play draughts or chess well, and by means of gestures and pantomime can make his wants and most of his wishes fairly well understood by those accustomed to interpret them. Yet he may only be able to articulate some one or two words, or else combinations of mere unmeaning sounds, such as 'poi, boi, bah,' 'sapon.' On rare occasions, under the influence of strong emotion, the patient may blurt out some simple expletive or short phrase, such as 'oh dear!' He cannot be made to repeat a word, or even the simplest vowel-sound, which he has just heard uttered. In a few cases the patient has seemed unable to understand what is said, as though from some difficulty in realising the meaning of words. Words may have to be uttered very slowly and repeated several times to such a patient, and even then they may fail to convey their meaning—a defect known as 'word-deafness.' Yet the language of gesture, appealing as it does to the sense of sight, may be at once understood.

The patient's power of writing is necessarily interfered with when aphasia, as is so often the case, co-exists with right hemiplegia. Many such patients, however, learn to write with the left hand to a variable extent. The variations as regards the power of writing are, in fact, almost as marked as the variations in power of speaking, though these two classes of defects by no means run parallel with one another in the

same individual. The writer has known patients who were quite unable to express themselves in spoken words, or even utter a single articulate sound, write a good letter with no, or very few, mistakes (*aphemia*); on the other hand, the performance of an aphasic patient, without a copy before him, may be, and most commonly is, limited to writing his own name. At other times the patient shows an amnesic defect in writing, and writes much as an amnesic patient speaks. Still more rarely it is found that an aphasic patient is, though not from want of manual power, unable to write even a single letter (*agraphia*)—in attempting to do so he makes mere unmeaning strokes.

The disability spoken of above as 'aphemia' may be complete, and then the patient is absolutely speechless. But incomplete aphemia or aphemic defects exist in many degrees of completeness. These terms are now often applied to defects in the power of articulation—the mere mechanical part of speech—in which difficulties exist such as go with diseases in the bulb or the pons, or the slighter defects in utterance, constituting mere 'thickness of speech.' This kind of defect differs from (and may be easily distinguished from) aphasia, seeing that the subjects of it will always attempt at once to utter any word or simple sound, when they are bidden to do so. The term 'anarthria' has been used by some writers for what would be called bad aphemic defects—such, for instance, as are met with in bulbar diseases, where the articulatory motor centres are themselves the seat of lesion.

Aphasia occasionally supervenes, independently of paralysis or convulsions, in individuals who have been subjected to great excitement or prolonged overwork, when it may be due, perhaps, to mere functional derangements. In other cases it presents itself as a temporary condition, lasting only for a few hours or a few days, in a patient who has just had an attack of right-sided unilateral convulsions; or, lastly, as has already been indicated, it occurs in conjunction with a right-sided hemiplegia produced either by brain-softening or by cerebral hæmorrhage. Cases belonging to the latter category vary very much amongst themselves as regards the degree of co-existing hemiplegia. If the third left convolution alone is damaged by softening, the hemiplegic condition may be absent, or transient and incomplete—never, perhaps, affecting the leg appreciably. This condition is often induced by a small hæmorrhage, or by a patch of softening produced by an embolism of that branch of the middle cerebral artery which supplies the third frontal convolution; but where the hæmorrhage is larger, or where the main trunk of the middle cerebral artery is obliterated, either by an embolus or a thrombus, the aphasia, or aphasia with

amnesia, is combined with much graver and more persistent hemiplegic symptoms.

**PATHOLOGY.**—The modern concentration of attention upon these defects of speech was started by the enunciation of Broca's views as to the dependence of the aphasic defects upon lesions in or about the third left frontal convolution. And all pathologists are now agreed as to the fact that such lesions are much more prone to give rise to aphasic symptoms than are corresponding lesions on the right side of the brain. It is commonly believed, moreover, that *amnesic* defects of speech are most commonly produced by superficial lesions of the left hemisphere, though by lesions situated farther back—that is, in the neighbourhood of the posterior extremity of the Sylvian fissure.

The third left frontal convolution is not now supposed, as Broca put it, to be the seat of any 'faculty of language,' though the anatomical investigations of Meynert and of Broadbent have shown that its relations with other convolutions are exceptionally complex. Whether certain higher 'motor' centres for speech are situated in this part of the brain, as is commonly believed, or whether such centres are rather, as the writer believes, kinæsthetic centres, it must at least be conceded that this convolution is intimately concerned with the physical expression given to thought in articulate speech.

We know that the left hemisphere is the one from which the volitional incitations proceed in the case of written language, and it is presumed that the same half of the brain also takes the lead in the production of articulate speech. It is, therefore, a point of much interest when we find that, in some of the exceptional cases in which aphasia has occurred in association with lesions on the right side of the brain and left hemiplegia, the individuals had been left-handed during life. Some of the other exceptional cases, however, have not admitted of this interpretation.

*Different kinds of Word-memories.*—For the proper understanding of these different defects of speech and writing many details are essential. There are four different kinds of word-memory—three essentially different types, but one of them existing in two forms, so as to make four varieties in all. These varieties of verbal memory are as follows: (1) Auditory memory—the memory of the *sounds* of words, that is, of the auditory impressions representative of different words; (2) Visual memory—the memory of the *visual appearances* (printed or written) of words, that is of the visual impressions corresponding with different words; (3) Kinæsthetic memory—(a) the memory of the different groups of sensory impressions resulting from the movements of the vocal organs during the utterance of words (impressions from muscles, mucous

membranes, and skin)—these the writer has proposed to speak of as ‘*glosso-kinæsthetic*’ impressions; (b) the memory of the different groups of sensory impressions emanating from the muscles, joints, and skin during the act of writing individual letters and words—these the writer has proposed to speak of as ‘*cheiro-kinæsthetic*’ impressions.

The organic seat of each of these different kinds of word-memory is in relation with its own set of afferent fibres; and the several centres are also connected with one another by commissural fibres, so that the recollection of a word in one or other of these modes doubtless involves some amount of simultaneously revived activity in one or two of the other word-centres. The relative intensity (in the process of recollection of words for ordinary speech) of the memorial revival in each of these centres is probably subject to more or less marked variation in different individuals. In the majority of persons the revival of words in the auditory centre is the most potential process, and that of which such persons are most conscious. In a smaller percentage of persons the revival of words in the visual centre seems to be the most essential process. While in a very much smaller percentage it seems possible that revival in the kinæsthetic speech-centre may be the most conscious process occurring during the recollection of words.

Owing to the fact of the existence of this multiple memory of words, it happens that loss of speech by no means always, or often, implies the loss of the memory of words. Many a patient who cannot speak can yet revive his auditory or his visual memory of words, so as more or less perfectly to understand what he hears, or what he sees (in print or writing), or perhaps what he both hears and sees.

It is also of importance to be borne in mind that for ordinary persons (that is, those who are neither congenitally blind nor congenitally deaf) the four memories of words are mainly called into play in definite couples, namely, the auditory and the glosso-kinæsthetic revivals taking place in ordinary speech; and the visual and the cheiro-kinæsthetic revivals taking place during ordinary writing. So that in expressing oneself in spoken words the memories of such words are first principally revived in the auditory centre, and then the nerve-units thus called into activity rouse in immediate succession the corresponding glosso-kinæsthetic elements before the pronunciation of the word can be effected through the aid of the motor centres in the medulla. Similarly, when expressing our thoughts by writing, though the memories of words are probably first revived in the auditory word-centre, like memories are almost simultaneously revived (through the intervention

of the audito-visual commissure) in related parts of the visual word-centre; and from this region stimuli must pass through corresponding cheiro-kinæsthetic elements, before the actual writing of the word can be effected through the instrumentality of motor centres in the cervical region of the cord.

There can be no doubt that the functional association existing between the auditory and the glosso-kinæsthetic centres is of the closest kind. But the bond of association between the auditory and the visual centres is no less intimate. These latter centres are often necessarily called into activity in immediately successive units of time. This happens, for instance, in two such common processes as reading aloud and writing from dictation.

In reading aloud the primarily excited visual word-centre must arouse (through the visuo-auditory word-commissure) related parts of the auditory word-centre, since this is the part which ordinarily calls the glosso-kinæsthetic centre into activity, and from it properly co-ordinated incitations issue to call into play the motor centres in the medulla.

Again, in writing from dictation, the sounds of words reach the auditory word-centre, and the activity thus aroused becomes transmitted (through the audito-visual commissure) to related parts of the visual word-centre, this being the part which usually arouses the cheiro-kinæsthetic centre into activity for the production of writing movements.

In deaf-mutes, in the congenitally blind, and also in certain cases of disease, as it would seem, the relations between the centres are rather those indicated by the dotted lines (*f e*) in fig. 5.

*Concerning the Localisation of the different Word-centres.*—Looking to the extremely important part that ‘words,’ either spoken, written, or printed, play in our intellectual life, and to the manner in which they are interwoven with all our thought-processes, it becomes highly probable that most important sections of the auditory and visual perceptive centres are devoted to the reception (and consequently to the revival in thought) of impressions of words; so that, for convenience of reference, we may speak of these sections as auditory and visual ‘word-centres’ respectively. Similarly, there must be kinæsthetic word-centres of two kinds—the one in relation with speech movements, and the other in relation with writing movements. It is possible that the parts of the general auditory and visual centres which are in relation with word-impressions may be as distinctly defined as are the analogous parts of the general kinæsthetic centres that are in relation with speech movements. Certain it is that there are some varieties of amnesia in which the part of the visual centre in relation with words seems to be specially at fault (causing ‘word-blindness’); just as

there are other cases in which the part of the auditory centre in relation with words is either wholly or partially inactive (causing 'word-deafness'), in each case without evident defect in other parts of the general auditory or visual word-centres.

In regard to the visual centre as a whole, it seems to be now established that it is more or less diffused through the occipital lobe. As to the localisation of the general auditory centre, there is some clinico-pathological evidence to show that the auditory word-centre may be in, or in the immediate neighbourhood of, the upper temporal convolution.

The situation of the two kinæsthetic word-centres can, however, be much more precisely localised. The writer holds the opinion that the so-called cortical 'motor centres' of Ferrier and others are really sensory centres of kinæsthetic type, by means of which movements are guided (*see* KINÆSTHESIS). That being so, Broca's region, or Ferrier's centre for the movements of the mouth and tongue (*viz.* the posterior part of the third frontal and the inferior part of the anterior ascending frontal convolution) is, in reality, the part of the brain which we have been alluding to as the glosso-kinæsthetic word-centre. The situation of the cheiro-kinæsthetic word-centre cannot be localised as yet with as much confidence, though the tendency is to follow Exner, who believes it to be situated in the posterior part of the second frontal convolution.

It must be supposed, therefore, that the auditory and the visual word-centres are situated, the one not far away from, and the other actually within, some part of the cortex of the occipital lobe, and that they are connected together by a double set of commissural fibres (*fig. 5, a, b*). We must also

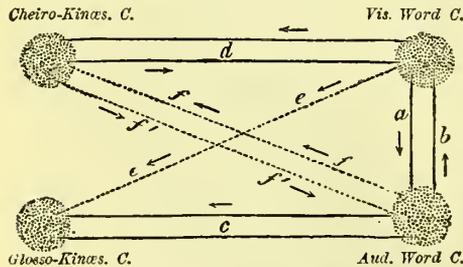


FIG. 5. Diagram showing the commissural connections between the different word-centres.

- a. The visuo-auditory commissure.
- b. The audito-visual commissure.
- c. The audito-kinæsthetic commissure.
- d. The visuo-kinæsthetic commissure.

suppose that two other sets of commissural fibres exist which are of much importance in regard to speech and writing, *viz.*, one set (*c*) through which the auditory word-centre acts upon the glosso-kinæsthetic centre, for the production of articulate speech; and the

other (*d*) by means of which the visual word-centre acts upon the cheiro-kinæsthetic centre, for the production of writing.

Thus, for speech we have the combined auditory and glosso-kinæsthetic word-centres, acting from the cortex through internuncial fibres, upon motor centres in the medulla; and for writing we have combined visual and cheiro-kinæsthetic word-centres, acting from the cortex through internuncial fibres, upon motor centres in the cervical region of the cord. In the study of speech-defects generally it is, therefore, needful to consider the effects of lesions: (1) in the different kinds of word-centres; (2) in the different commissures by means of which the centres are connected; (3) in certain internuncial fibres; and (4) in certain motor centres.

*Defects of Speech from Lesions in Word-Centres.*—According to the writer's views it is not at all needful, as many authorities have thought, to postulate the existence of a separate 'centre for conceptions or ideas.' The supposed theoretical necessity for assuming the existence of some such centre may in most cases be obviated by a fuller recognition of the different degrees of functional excitability that may exist in the auditory and the visual word-centres respectively. It should, in fact, be constantly borne in mind by those who study these defects of speech that each perceptive centre is capable of being called into activity in three modes: (*a*) by 'voluntary' recall of past impressions, as in an act of recollection; (*b*) by 'association,' that is by impulses communicated from another centre during some act of perception or during some thought-process; (*c*) by means of external impressions. Now the excitability of these centres is liable to vary much—as a result of advanced age, or of different general or local morbid states. The molecular mobility may be so much lowered that they are only capable of responding to powerful stimuli; in such cases either 'volitional' recall alone, or 'associational' recall as well, may be impossible or more or less difficult, responses occurring only to external impressions—that is, the patients are only able to repeat words pronounced, or to copy words written before them.

The effects of lesions of the different word-centres are as follows: (1) A lesion of the glosso-kinæsthetic centre produces simple aphasia; whilst (2) one of the cheiro-kinæsthetic centre produces simple agraphia. In neither condition is the power of thought very appreciably interfered with. These are commonly supposed to be motor defects, but this the writer believes to be an erroneous interpretation—since they are defects due to the loss of certain sensorial aptitudes (of the kinæsthetic order) just as much as are the other forms of aphasia now about to be mentioned, the existence of which were first pointed out and explained by the writer in

1869, though they were subsequently more prominently explained in 1874 by Wernicke under the name of 'sensory aphasia.' One of these has been known as 'word-deafness' and the other as 'word-blindness.' But defects in the activity of the auditory and of the visual word-centres respectively give rise to varied and complicated results, which can only be very inadequately summarised by these phrases. Thus under the head of (3) lesions of the auditory word-centre, we must refer to totally different defects according to the degree of functional degradation of the centre: (a) slight auditory amnesia from functional defect of the centre, leading to impaired spontaneous speech, owing to forgetfulness of names or nouns; (b) profound auditory amnesia, in which, from similar causes, both voluntary and associational speech are greatly impaired, though imitative speech is retained, and the almost speechless patient may be able to read aloud correctly and fluently; (c) word-deafness and aphasia owing to a destructive lesion of the auditory word-centre, in which the patient is not only unable to comprehend speech, but is himself speechless, and may also be unable to write spontaneously. Lesions of (4) the visual word-centre, whether functional or structural, reveal themselves in less varied modes; when severe they would result in the production of word-blindness together with agraphia.

*Defects of Speech from Lesions of the Commissures between Word-centres.*—It seems to the writer better to reserve the word 'commissure' as an appellation for the fibres that connect centres of the like kind, that is, either sensory centres or motor centres; and to name 'internuncial' the fibres which connect sensory with motor centres.

As already stated, double commissures exist between the auditory and the visual word-centres (fig. 5, a, b) which are habitually called into play in certain mental operations, so that their destruction leads to very definite defects. Thus, in naming objects at sight, or in reading aloud, stimuli have to pass from the visual to the auditory word-centre (by the visuo-auditory commissure) before the naming or the reading aloud can occur. Again, in writing from dictation, and probably also in writing any spontaneous effusion, stimuli require to pass between these two centres in an opposite direction, namely, from the auditory to the visual centre, and through a different set of fibres (the audito-visual commissure). In 1880 the writer published (*The Brain as an Organ of Mind*, p. 640) some details concerning a man (who has ever since been under observation from time to time) who suffers from a lesion destroying the commissures in question. This man understands perfectly all that is said to him and all that he reads, yet he cannot read aloud a single word or even name a single letter, though immediately

that he hears the word or letter pronounced he can repeat it at once. Again, he cannot write a single word, or even letter, from dictation, though he can at once, with his left hand, proceed to copy any such word or letter that may have been written for him on a sheet of paper. Cases have been recorded also in which the separate halves of the commissure between the auditory and the visual word-centres have been damaged; so that reading from dictation or naming at sight have been interfered with and not writing from dictation, or *vice versa*.

The other commissures whose lesions have to be considered are those (fig. 5, c, d) connecting the auditory with the glosso-kinæsthetic, and the visual with the cheiro-kinæsthetic word-centres. Defects in the course of these commissures are of interest, more especially from the point of view of a regional diagnosis. The writer some years ago pointed out, for instance, that a lesion of any part of the audito-kinæsthetic commissure (fig. 5, c) should produce an aphasia indis-

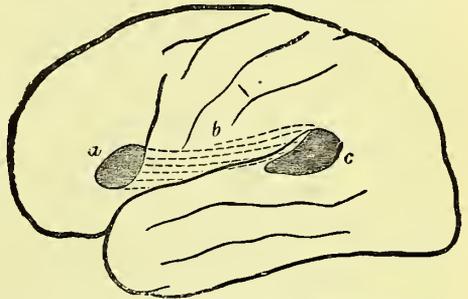


FIG. 6.—Diagram showing the possible location of an aphasia-producing lesion, either in the glosso-kinæsthetic centre (a), in the audito-kinæsthetic commissure (b), or, in association with word-deafness, in the auditory word-centre (c).

tinguishable from that which would be produced by damage to Broca's convolution, that is, to the glosso-kinæsthetic centre itself. This is, of course, a very important point, since it shows that it is an error to suppose that typical aphasia can only be caused by a lesion in Broca's region. A precisely similar result should also be produced by complete destruction of the audito-kinæsthetic commissure, in any part of its course between that region and the neighbourhood of the occipital lobe where the auditory word-centre is situated. If the latter centre itself be damaged, as before stated, we then get the aphasia complicated with word-deafness, and thus have the means of arriving at an accurate regional diagnosis. Once clear of the centre, however, a lesion involving any part of the audito-kinæsthetic centre should produce a typical aphasia absolutely indistinguishable clinically from that resulting from a lesion in Broca's region itself. This explains the occurrence of aphasia in associa-

tion with lesions in the island of Reil, and other sites nearer the posterior extremity of the Sylvian fissure (see *Brit. Med. Jour.*, Nov. 5, 1887, p. 986).

This point, like the question of the existence and interpretation of aphemia, will be found to be a touchstone for testing the truth of the doctrines here set forth concerning the pathogenesis of speech-defects, as compared with those of Stricker and of Hughlings Jackson. According to their views, the commissural fibres now referred to pass between the auditory and a true 'motor' centre for articulation. According to Stricker (*Le Langage et la Musique*, 1885, p. 73), a damage occurring in the course of these fibres would cause, not aphasia but word-deafness. His view being that words are realised mainly by memorial revival in motor cortical centres, in the case assumed there would be word-deafness because stimuli could not pass from the auditory to the assumed motor centres in which the essential part of the apperceptive process is carried on. It seems clear also that these doctrines necessitate the view that an isolated lesion in Broca's region only should also cause word-deafness as well as aphasia. This, however, is certainly not the case—a fact which is itself sufficient to negative the truth of the views of Hughlings Jackson and of Stricker.

Damage to any part of the visuo-kinæsthetic commissure (fig. 5, *d*) would also be capable of producing agraphia of just as complete and typical a kind as if the lesion had been in the cheiro-kinæsthetic centre itself.

*Defects of Speech from Lesions of Internuncial Fibres.*—We have here to do with a kind of defect known as aphemia. This is a speech-defect, and there is no analogous defect of writing, except that which is occasioned by actual paralysis of the right hand. In complete aphemia there is absolute loss of articulate speech without any mental impairment, and with the power of communicating the thoughts by writing preserved absolutely intact. An excellent example has been recorded by the writer in the *Brit. Med. Jour.*, Nov. 5, 1887.

Such a condition is produced when the glosso-kinæsthetic centre is itself uninjured, but where the whole of the internuncial fibres emanating from it and proceeding to the true motor articulatory centres in the bulb are damaged in some part of their course—whether just beneath the cortical centre, in the internal capsule, in the peduncle, or in the pons. Sometimes the defect may be of a functional rather than of a structural type, constituting Charcot's 'hysterical mutism in the male.'

Incomplete aphemias, however, exist of all degrees of severity, and they are about the commonest of all speech-defects occurring in association with hemiplegia. They are due to partial damage to the group of inter-

nuncial fibres above referred to, and are represented by defects of articulation, varying from almost complete unintelligibility to the mere blurred utterance or thickness of speech which is so common.

Aphemia is clearly not a sensory defect—it is not a form of amnesia—because the subjects of it can revive words in all possible modes, and are, therefore, able to think and express their thoughts with an unimpaired freedom by writing. If the aphemia be in any way incomplete, moreover, such a case can be easily discriminated from a case of aphasia by the fact that the aphemic patient will always at once make an attempt, when bidden, to pronounce some simple word or syllable (however poor the attempt may be), while the typical aphasic patient is unable to make any such attempt—he will not try even to repeat the simplest vowel-sound.

*Defects of Speech due to Lesions in the Bulbar Articulatory Centres.*—In these cases, as where the lesions occur in the internuncial fibres, all the centres in which the memory of words can be revived are intact. There is consequently nothing whatever to interfere with the flow of thought, and in incomplete cases nothing to prevent attempts at articulation being made. These two classes of cases are indeed less separable clinically than they are from a theoretical or scientific point of view.

It would be well to reserve the term 'anarthria,' as introduced by Kussmaul, for the defects of speech caused by morbid states of the bulbar articulatory nuclei—with the full understanding, however, that aphemia and anarthria will often be indistinguishable from one another by their own intrinsic characters, though they will generally be quite capable of being diagnosed from one another by taking into consideration the mode of onset and the particular grouping of other attendant signs.

**DIAGNOSIS.**—The means of distinguishing the different aphasic and amnesic speech-defects from one another has already been fully enough set forth in the preceding sections. The diagnosis of the exact nature of the defect is often exceedingly difficult, unless the patient be thoroughly examined in a systematic manner, and in accordance with some definite plan. Elsewhere (*Paralyses; Cerebral, Bulbar, and Spinal*, 1886, p. 125) the writer has given such a 'Schema for the Examination of Aphasic and Amnesic Persons,' which has been used with success for many years. The other side of the question, viz., the pathological diagnosis, must be made in accordance with the rules generally applicable to this part of the diagnostic problem in cases of brain-disease.

**TREATMENT.**—Where aphasia occurs after excitement or overwork, without paralysis, it is a warning of much importance, since it may be the precursor of much graver symp-

toms. Under such circumstances the patient requires an absolute cessation from work for a time, and most careful watching. Stimulants may need to be diminished; and bromide of potassium, with sumbul and other sedative remedies, should be administered. Where aphasia is a temporary condition in association with right-sided convulsions, or where it is lasting and co-exists with right-sided paralysis, the treatment of the aphasic condition becomes merged in that of the associated convulsive tendency or paralytic condition, since, as a rule, an amelioration takes place in the patient's power of speaking coincidentally with his improvement in other respects. This, however, is not always the case where aphasia has co-existed with a partial hemiplegic condition: the paralysis may be recovered from, whilst the aphasic defect may remain more or less as it was. Where this is the case, an attempt should be made to teach the patient to speak again, especially if the defect be of the aphemic type. Such efforts have occasionally been crowned with success (see *Trans. of Clin. Soc.*, vol. iii. p. 92, and *Brit. Med. Journ.*, Nov. 5, 1887, p. 988), but much judgment and untiring patience have to be called into play in order to obtain satisfactory results.

H. CHARLTON BASTIAN.

#### APHEMIA (*â*, neg.; and *φημι*, I speak).

A term differently applied by different writers, but commonly understood to be a designation for certain articulatory defects (see ANARTHRIA) dependent upon injury, direct or indirect, to the internuncial fibres connecting Broca's convolution with the motor centres of the bulb. Where the aphemia is incomplete the nature of the defect is generally obvious; where it is complete the patient is absolutely speechless, and is apt loosely to be described as aphasic, though the fact that he can express himself freely by writing shows that this is not a correct designation of his condition. See APHASIA.

#### APHONIA (*â*, priv.; and *φωνή*, the voice).

Absence of voice, that is, of intonated utterance. See VOICE, Disorders of.

#### APHRODISIACS (*Ἀφροδίτη*, Venus).—

DEFINITION.—Medicines which increase the sexual appetite and power.

ENUMERATION.—The direct aphrodisiacs include: Nux Vomica and Strychnine, Phosphorus, Cantharides; Urtication and Flagellation; Cannabis Indica, Damiana, Opium, and Alcohol in small doses. Iron and bitter tonics; meat diet; warm clothing, especially around the hips and loins; and abstinence from severe mental and bodily work, act as indirect aphrodisiacs.

ACTION.—Aphrodisiacs may act by increasing the excitability of the nerves passing to or from the genital organs, or of the genital

centre in the spinal cord (see ANAPHRODISIACS), as, for example, strychnine, nux vomica, and probably phosphorus; by causing irritation of the nerves of the genital or urinary organs or of adjoining parts, as cantharides and urtication; or by stimulating the brain, as Indian hemp or small doses of opium. Alcohol in large doses has a double action, increasing the sexual desire by stimulating the brain, while lessening the power of erection, probably by weakening the nervous mechanism through which erection is produced. As the sexual passion becomes diminished when the nervous system is weakened with the rest of the body, and increases with returning strength, iron with bitter tonics, and generous diet act indirectly as aphrodisiacs.

USES.—When the sexual functions are abnormally depressed, strychnine and phosphorus are the most generally useful of the direct aphrodisiacs. Cantharides, although sometimes valuable, must be employed with caution.

T. LAUDER BRUNTON.

APHTHÆ (*ἀπρω*, I inflame).—SYNON.: Aphthous Stomatitis; Fr. *Muguet*; Ger. *Fasch*.

DEFINITION.—A form of catarrh of the buccal mucosa, characterised by the appearance of one or more patches (*aphthæ*) possessing a peculiar structure.

ANATOMICAL CHARACTERS.—Aphthæ have at first the appearance of vesicles; but microscopical sections show that they are due to elevation of the epithelium by a solid, fibrinous exudation, over which the epithelium usually dies, soon separating with the fibrinous flake. By the time this is effected the mucosa is again, as a rule, thinly covered by epithelium, so that a true ulcer is never present; but slight suppuration may be established, with the destruction of the superficial layer of the mucosa.

SYMPTOMS, COURSE, AND DURATION.—Aphthæ occur chiefly in children during the first dentition, upon any part of the buccal mucosa—most frequently inside the lower lip and on the tip of the tongue. At first they look like small vesicles, but after a few hours they resemble ulcers with adherent grey or yellowish sloughs, each being surrounded by a ring of injection. The patches vary in size from that of a lentil downwards; they are single or multiple; grouped or scattered; two or three may coalesce, but the patches never reach a large size. Aphthæ cause slight pain, and are specially sensitive to substances like salt or sugar. Dyspeptic troubles are usually present, and the breath is often foul.

Each aphtha runs its course in three or four days, and when multiple the patches come out in a crop. This may be succeeded by other crops, so that all stages of the lesion may be present at one time. The trouble

may thus continue for weeks. In other cases patients are liable to crops of aphthæ at varying and sometimes long intervals.

**DIAGNOSIS.**—Aphthæ have chiefly been mistaken for patches of thrush. Absence of the red areola, and presence of the characteristic fungus, will distinguish thrush even when no history or statement of symptoms can be obtained.

**TREATMENT.**—The occasional application of a little powdered borax to the aphthæ causes their speedy healing. In prolonged cases, the health in general, and stomach and intestines in particular, must be attended to.

STANLEY BOYD.

**APHTHOUS.**—A term applied to diseases in which aphthæ are present.

**APLASTIC.**—(ἀ, priv.; and πλάσσω, I mould).—Incapable of being organised or of forming tissues; generally applied to inflammatory exudation.

**APNEUMATOSIS** (ἀ, priv., and πνεῦμα, respiration).—A synonym for atelectasis. *See* ATELECTASIS.

**APNŒA** (ἀ, priv.; and πνέω, I breathe).—Apnœa, literally signifying breathlessness, is used by some medical writers as synonymous with asphyxia, the condition which supervenes on suspension or obstruction of the respiratory function. *See* ASPHYXIA.

By physiologists, and with more correctness, the term is employed to signify the cessation of respiratory movements which is brought about by hyperoxygenation of the blood; as when an animal is made to inhale oxygen, or to breathe more rapidly than the needs of the economy require.

**APOLLINARIS, Waters of.**—Acidulous alkaline table-waters. *See* MINERAL WATERS.

**APOPLEXY.**—**DEFINITION.**—The word *apoplexy* means, by its etymology, a *striking from* (ἀπό, from, and πλῆξις, a striking), and was at first and is still chiefly used to signify sudden abolition of consciousness and power of motion, which, in common English, is also called a *stroke*. The idea fossilised in the term is that of an unseen actor, and brings to our lips unconsciously the thoughts of an age that is much less distant in the time that is measured by centuries, than in that which is estimated by the cycles of thought.

Cerebral hæmorrhage is the most frequent cause of apoplexy; thence 'hæmorrhage into the brain' and 'apoplexy' came to be used as synonymous expressions. Subsequently the effusion of blood itself was spoken of as *the apoplexy*, the word being used to designate the pathological condition causing the symptoms which it at first epitomised. Ultimately it was applied to such spontaneous hæmorrhage elsewhere; and thus extravasations into the substance of the lung, the spleen, or the retina were, and still are, termed pulmonary,

splenic, or retinal 'apoplexies.' It would be difficult to find a more striking instance of the looseness of the process of nomenclature, or of the strange way in which precision in words lagged behind the process of pathological discovery.

The term *cerebral apoplexy* is sometimes used to particularise hæmorrhage into the brain, but it is more commonly employed to denote an apoplectic condition depending on any cerebral lesion, and in that sense it will be here employed. W. R. GOWERS.

**APOPLEXY, CEREBRAL.**—**SYNON.**: a 'stroke'; Fr. *Apoplexie*; Ger. *Schlag*.

**DEFINITION.**—Loss of consciousness, of sensation, and of voluntary motion, coming on more or less suddenly, and due to a morbid state of the brain.

The condition of *coma* is termed 'apoplectic' when of sudden or rapid onset. Loss of consciousness may be due to other causes acting directly on the brain, such as defective or excessive supply, or altered condition of blood; but it is customary to include among the forms of *apoplexy* that only which is due to cerebral congestion, and to consider as *apoplectic states* those only which result from distinct toxæmia. Unconsciousness resulting from acute anæmia of the brain, or such as succeeds an epileptic fit, is not termed apoplectic. The term has, of necessity, become restricted, as increasing knowledge has explained the nature of many conditions previously confounded with apoplexy. It should be further observed that the term has always been restricted to such conditions as occur without conspicuous external cause. The same state when due to an injury, is not called by the name which is employed if there is no injury. The explanation of the inconsistency is to be found in the idea that is to be traced alike in the native and the foreign word. The 'stroke' determines the designation when the agent is unseen. It is to the conception of an unseen striker that the word is due.

The essential cause of apoplexy is a sudden cerebral lesion, occurring without external injury. The sudden abolition of function seldom occurs except at the onset of a positive and permanent damage, and a sudden lesion cannot occur except as a result of an alteration in the physical conditions of the circulation, no other element in the brain being capable of thus suddenly producing a distinct lesion. The great cause of apoplexy is the rupture of a vessel, and hence the 'stroke' became so confused with the mechanism that the term became synonymous with a spontaneous hæmorrhage in any organ. The same state may, however, be caused by obstruction of a vessel by thrombosis or embolism. Profound coma is rarely due to any other spontaneous cerebral lesion. A very small hæmorrhage may cause apoplexy.

Apoplectic symptoms may, however, occur without obvious lesion of the brain. The coma which is associated with congestion of the head, is sometimes termed 'congestive apoplexy,' and that which occurs in the absence of any recognisable cause, has been called 'simple apoplexy.'

**PATHOLOGY.**—In all these cases the apoplexy is in relation chiefly to the extent and suddenness of the lesion, and the intensity of the damage. Roughly speaking, its occurrence may be said to depend on the suddenness, together with the intensity, of the cerebral mischief; its degree on the extent of the same. But the occurrence of apoplexy depends sometimes on the size of the lesion; and the degree varies not only directly with the extent of the mischief, but with the extent of brain-tissue which is exposed indirectly to the irritative influence of the primary lesion. Hence position of lesion has an influence in determining the apoplectic symptoms. For these several reasons apoplexy is especially profound when the hæmorrhage affects both hemispheres, either by simultaneous extravasation on each side, or as the result of hæmorrhage into the lateral ventricles.

The precise condition on which the essential element in apoplexy—the arrest of all manifestation of brain function—is immediately dependent has been a matter of much dispute. It was formerly ascribed to the pressure exerted by the clot on the rest of the brain, either influencing directly the cerebral tissue, or pressing on and emptying its capillaries (Niemeyer). That such pressure is exerted by a large hæmorrhage is unquestionable. The convolutions on the side of the extravasation are flattened, and the falx is bulged to the opposite side. It cannot be doubted that the intensity of the apoplexy in these cases is due in part to this cause. But this will not explain the occurrence of the symptom in small hæmorrhages, by which no general pressure is exerted, or not more than is at once relieved by the displacement of the mobile fluid which surrounds the vessels. It will not explain symptoms in laceration of the brain, nor the instant loss of consciousness at the very onset of a severe hæmorrhage, in which, as Jaccoud insists, it should, if merely due to pressure, be a late rather than an early symptom. There can be little doubt from these considerations, and from the cases in which there is no recognisable brain-lesion, that what is called shock is an important element in the causation of apoplexy. Thus in cerebral hæmorrhage the apoplectic symptoms are due in part to the influence on the rest of the brain of the irritation of the nerve-elements by laceration. A hæmorrhage, for instance, stops for a few hours all reflex action by the irritative inhibition downwards, and it stops also the cerebral action by the same process

acting upwards. We can thus understand why vascular occlusion causes a slighter degree of apoplexy, since the immediate irritation of the local anæmia is less than that of laceration; and also why lesions of the pons produce, as they do, such deep and long-continued coma, since the irritated fibres are connected with a large part of the cerebrum, and thus influence it indirectly.

*Simple apoplexy* was a term given by Abercrombie to the cases, once thought to be frequent, in which apoplexy occurs without recognisable brain-mischief or blood-poisoning. Some of these cases were probably instances of uræmic poisoning, and others may have been due to undiscovered and extensive embolism, causing death before there was time for softening to occur. But cases are not infrequent to which neither of these explanations applies; in which death occurs in an apoplectiform attack, all the organs are found healthy, and the brain only exhibits, in common with the other organs, that passive congestion which results from an asphyxial mode of death. The nature of these cases is still mysterious, but they may be grouped with those in which fatal coma follows an epileptic attack, and is apparently due to the brain-shock produced.

*Serous apoplexy* is a term sometimes applied to cases of fatal apoplexy in which no lesion is discoverable except excess of serum on the surface of the brain. It is now understood that such serous effusion is associated merely with senile atrophy of the brain, and that the name, as a pathological designation, is no more than a refuge for a destitute diagnosis. There is no reason for associating its presence with the apoplectic symptoms. The cases described under this term were probably instances of uræmia, or of 'simple apoplexy' in old persons with atrophied brains.

**SYMPTOMS.**—Apoplexy is itself a symptom, and can only claim to be treated as a disease on account of the frequency with which it presents itself as the prominent element in a patient's state. The striking feature of apoplexy is loss of consciousness without obvious failure of the heart's action. The onset is often instantaneous, so that the sufferer falls to the ground. The face may be flushed or pale—it is rarely very pale. The heart and arteries vary in their state according to the cause; at first the pulse is often feeble from shock, even in cases in which it soon becomes full and tense. Respiration continues, but is laboured and stertorous. The limbs are motionless. In severe cases no reflex action can at first be excited. The pupils may be dilated, contracted, or unchanged: in profound coma they are usually dilated; and they often vary in size spontaneously, being sluggish in their action to light. The patient can usually swallow, although often with

difficulty. The sphincters permit the escape of urine and fæces, or the urine may be retained. In a case of moderate severity the reflex action soon returns, the conjunctivæ become sensitive, the patient can be roused to exhibit some sign of consciousness, and he shows returning power of motion. At first there is merely a half-purposive withdrawal of a limb when it is pinched or pricked. Afterwards more prolonged and voluntary movements are produced: he opens his eyes when spoken to, and tries, when told to do so, to protrude his tongue. On the other hand, the apoplexy may continue or may deepen in intensity; the patient dying at the end of a few hours or a few days. Death rarely occurs in a shorter time than two or three hours. In very rare instances an extensive hæmorrhage into the pons or medulla, or an effusion into the fourth ventricle, may stop the respiration and kill the patient in a few minutes.

It is not often, however, that there is this simple loss of cerebral function, uniformly distributed, and gradually deepening or passing away. Much more commonly the symptoms of a local cerebral lesion are added to those of apoplexy. Frequently such symptoms precede the loss of consciousness—unilateral weakness, deviation of the mouth, convulsion. These may be recognised during the attack. The limbs on the one side exhibit more complete muscular relaxation than those on the other; they fall more helplessly when raised; or there is unilateral rigidity or clonic spasm, unvaried in its seat. Or inequality of pupils may be observed, or rotation of the head and conjugate deviation of the eyes. As a patient recovers, these local symptoms become more and more distinct, the tongue deviates on protrusion, speech and swallowing are difficult, or the patient may have lost the use of language.

In *ingravescent* apoplexy the commencement of the cerebral mischief is marked by symptoms of general shock, without any, or with merely transient, loss of consciousness. There is commonly pain in the head, and there may be other localising symptoms. After some hours, during which the patient may continue his occupation, coma gradually comes on and deepens into death. This form of apoplexy, first described by Abercrombie, is usually due to a slowly increasing cerebral hæmorrhage.

The temperature in cerebral apoplexy is at first lowered in most cases, but usually the fall is small, and is succeeded, after twelve to twenty-four hours, by a rise. In some cases there is a considerable rise within the first six hours, and the temperature then commonly continues to rise, reaching 104° or 106° Fah., and even higher. Such a symptom is a precursor of death.

DIAGNOSIS.—The first diagnostic problem is whether the patient's state comes under the

general name of apoplexy; or is one of the morbid conditions in which there is a loss of consciousness, similar in general characters to apoplexy, but due to one of the causes excluded in the definition given above. From the unconsciousness due to cardiac *syncope*, apoplexy is easily distinguished. In the former the heart's action fails, the pulse is weak and imperceptible, the face is very pale, the respiration is sighing and irregular, reflex action is rarely abolished, and the sphincters are seldom relaxed.

From the several forms of *toxæmia* the diagnosis is often easy, sometimes extremely difficult. It is easy when, on the one hand, the symptoms of apoplexy are preceded or accompanied by those of a local cerebral lesion; or when, on the other hand, the direct or circumstantial evidence of poisoning is clear, or the symptoms of *toxæmia* unmistakable. Where there are no local symptoms, and where no guiding history is to be obtained, the diagnosis is difficult, but a correct opinion may commonly be formed by an attentive comparison of the symptoms present.

There may be, as just observed, indirect evidence of *toxæmia*: the breath may smell of opium or alcohol; the urine may contain albumin. But albuminuria or a smell of spirit may mislead. Cerebral hæmorrhage often occurs after drinking; spirit is constantly given to a person in a fit. A smell of spirit must therefore only be allowed weight in the absence of any evidence of cerebral mischief. So, too, albumin is always present in the urine in uræmia, but it is also very frequently present in cases of cerebral hæmorrhage. Alone, this evidence of Bright's disease is of little value, except there be general œdema and the patient be young; then uræmia is more probable than vascular degeneration and cerebral hæmorrhage. The urine should always be examined microscopically with reference to structural changes in the kidneys. But with other symptoms which indicate uræmic poisoning, albuminuria is conclusive.

The age of the patient should be considered. Late life is in favour of brain-disease. The history of a fall or blow on the head adds weight to other symptoms of cerebral mischief.

The character of the coma will sometimes guide us. In uræmia, and commonly in alcoholism, it is less profound than in cerebral mischief, so that the patient can readily be roused. In apoplexy, in opium-poisoning, and in the most intense form of alcoholic poisoning, the coma may be profound. On the other hand, in cerebral hæmorrhage the patient may sometimes be roused to answer questions. Violent struggling is strongly in favour of drink.

The mode of onset of the coma is important. In apoplexy it is sudden; in uræmia it is

slow. The uræmic patient becomes first drowsy, then comatose. But with convulsions uræmic coma may come on suddenly. The onset of the coma of opium- and alcohol-poisoning is also slow. Ingravescens apoplexy is of deliberate onset, but a profound degree of coma is quickly reached.

General convulsions at the onset exclude drunkenness, and usually opium-poisoning, while they favour the probability of uræmia, especially if they are followed by rigidity that changes its seat. When uræmic convulsions begin locally, the place at which they begin often varies. Cerebral mischief sometimes commences with a convulsion, but the convulsion is then commonly unilateral, at least in onset, and the preponderant affection of one side at a time is indicated by the deviation of the head. It is seldom that well-marked deviation of the head and eyes occurs, first to one side and then to the other, as the fit progresses, unless there is an organic lesion of the brain, and one-sided symptoms are almost always to be recognised afterwards. Rigidity of limbs or local muscular twitching during the coma is, if constant in seat, in favour of cerebral mischief; if variable in position, it is in favour of uræmia (Reynolds). In diabetic coma, the odour of the breath, and the result of an examination of the urine (never to be omitted) will prevent mistake, even in the absence of a history of diabetes. But the absence of local cerebral symptoms must be carefully noted, since the presence of diabetes does not exclude a cerebral lesion. Post-epileptic coma is of course preceded by a convulsion, and should be borne in mind.

The state of the pupils alone is of little importance. Great contraction occurs in and suggests opium-poisoning, but it is present in hæmorrhage into the pons Varolii. The pupils may be either normal or dilated in uræmia, in alcoholic or in belladonna-poisoning, and in apoplexy. Inequality of pupils, a unilateral symptom, points to brain-mischief. The retina should be examined, since the presence of albuminuric retinitis points, in the absence of the signs of a localised cerebral lesion, strongly to uræmia.

Lastly, the temperature should be noted. In uræmia there is persistent uniform depression; in cerebral lesions the initial depression is succeeded by a rise to a point above the normal.

The second problem in diagnosis is the precise cause of the apoplexy, the existence of which has been ascertained by the considerations just reviewed. This element, however, will be described more fully under the heads of the chief lesions that produce the state—cerebral congestion, hæmorrhage, and softening. In may be here pointed out that slight and transient apoplexy, without local symptoms, with flushed face, and coming on during effort, points to cerebral

congestion; slight and transient apoplexy with marked local symptoms points to softening; early and profound loss of consciousness to cerebral hæmorrhage. Post-epileptic coma may be distinguished by the history of epileptic attacks; or, if this be not forthcoming, it may be suspected if symptoms of a local cerebral lesion or indications of toxæmia are absent, if the patient be under 40, and exhibits indications of speedy recovery, and also if he has bitten his tongue and passed urine. 'Simple apoplexy' cannot be diagnosed during life, since freedom from the symptoms of a local lesion does not afford ground for inferring that there is no such lesion.

PROGNOSIS.—The prognosis in cerebral apoplexy depends in part upon the intensity of the attack. As long as unconsciousness is complete, and reflex action abolished, the patient is in danger of speedy death. The longer the apoplectic condition lasts without improvement, the less prospect is there of recovery. Persistent depression of temperature, or a rise of several degrees above the normal either with or without an initial fall, are both of grave significance; such cases rarely recover (Charcot, Bourneville).

The nature, extent, and position of the cerebral lesion, when they can be inferred, furnish other prognostic indications. In hæmorrhage the prognosis is more serious than in softening. A sudden occurrence or increase of apoplectic symptoms, a few hours or days after a slighter attack, is always grave, indicating a fresh extravasation. If such apoplectic symptoms become profound and uniform, the prognosis is fatal, rupture into the ventricles or on the surface of the brain having probably occurred. If the localising symptoms point to a lesion of the medulla or pons, the prognosis is almost as unfavourable. Early return of consciousness and slight alteration in temperature are favourable signs. Previous cerebral disease renders the prognosis worse. Lastly, the prognosis must be influenced unfavourably by any impairment of the organic functions of circulation and respiration, whether independent of or due to the cerebral lesion. Previous alcoholism, from its effect on the nutrition of all tissues, is especially serious.

TREATMENT.—The treatment of cerebral apoplexy must be guided by the indications of its cause. Where none can be obtained, it should be treated by a system that is a compromise. The chief causes are hæmorrhage and thrombotic softening of the brain. Embolism seldom causes pronounced apoplexy. The measures therefore must be such as are suitable to both states, those being excluded which, while they would do good in one, would do harm in the other. But such a compromise is seldom needed. Diagnostic indications can usually be traced with clearness, and as a rule also several

coincide distinctly in this direction, whilst those that do not point in this direction are neutral. Hence, as a matter of fact, there should seldom be the hesitation that interferes with the proper energy of treatment, and the statements often made in books are not true,—at any rate in the present day they should not be true. Stillness is the most important condition. The patient should be moved as little as possible, but placed in the recumbent posture with the head slightly raised. The neck should be freed from constriction. If the extremities are cold, warmth may be applied to them; and cold to the head if there is local heat or flushing. Sinapisms to the neck and extremities sometimes seem to hasten the return of consciousness. The administration of stimulants should be regulated by the state of the heart. In thrombosis or embolism the heart should be kept up to the normal by very careful administration of alcohol, ether, or ammonia. In hæmorrhage it may be allowed to fall a little below the normal, but indications of failing power should be watched for and counteracted. Where no causal indications exist, the latter is the wiser plan. Venesection and purgation are remedies of similar effect, but different in degree, and are indicated by high arterial tension and cephalic congestion, shown by incompressibility of the pulse and flushing of the face. Venesection is useful where the heart acts strongly, and the pulse is full as well as incompressible. Its effect is proportioned to the rapidity with which the blood is taken, rather than to the quantity removed. Purgatives remove serum from the blood, and lessen the amount of blood within the skull by causing an afflux to the capacious intestinal vessels. The best purgative is croton oil or calomel. With a failing heart and pale surface they should be avoided. When in doubt we should give a mild aperient that acts quickly, such as senna or a little solid extract of cascara rubbed up in water, and the action should be ensured by a glycerine enema. Diuretics may always be used to relieve the vascular tension. As the apoplexy clears, the nature of the case becomes evident, and the treatment of the several conditions is described elsewhere (*see* BRAIN, Hæmorrhage of, and Softening of). For treatment of the other causes of the apoplectic state, *see* ALCOHOLISM, POISONS, and URÆMIA.

W. R. GOWERS.

#### APPENDIX VERMIFORMIS, Inflammation, Ulceration, and Perforation of.

**DEFINITION.**—Inflammation of the appendix vermiformis from lodgment of hardened feces or a foreign body; leading to ulceration, which frequently ends in perforation of its coats; and to inflammation (localised peritonitis) and suppuration (en-

cysted suppurative peritonitis) of the adjacent tissues.

**ÆTIOLGY.**—The usual cause of this affection is a fecal concretion, or a foreign body (*e.g.*, fruit-pips or -stones, a small bone, pins, shot, small gall-stones, &c.) imprisoned within the cavity of the vermiform appendix. Congenital deformities, and also twisting of the appendix on itself from contraction in its mesentery or from adhesions, are likewise important factors in inducing attacks. Perforation has been recorded in typhoid fever and tuberculous disease.

**ANATOMICAL CHARACTERS.**—Before perforation takes place the appendix may be found distended with pus or mucus; a foreign body or concretion lodged within it; and the mucous membrane ulcerated. The concretion or concretions vary in size from a small pea to a bean; are usually brown and hard; and consist of layers of condensed feces, secretions, and phosphates, deposited around a small nucleus, which may prove to be a seed or other foreign body, or a piece of unusually inspissated feces. These concretions greatly resemble, and are often mistaken for, fruit-stones. The appendix may, however, be found perforated, and yet no foreign body can be discovered. Ulceration and perforation may occur at any part of the appendix, usually, however, at the extremity of the lower third. When twisted on itself, or when acutely bent by adhesions, the appendix may be found gangrenous, or may be so greatly distended by mucus (being large, round, and hard) as to seem as if it contained a concretion, with occlusion of its lumen. The appendix may be adherent to the cæcum, ileum, rectum, bladder, mesentery, or abdominal wall, with the signs of an ordinary local peritonitis; or there may be a circumscribed peritoneal abscess.

**SYMPTOMS.**—Pain, generally ill-defined, in the right iliac region, may be the only symptom to attract attention, but, as a rule, it is not until local peritonitis or typhlitis is set up that we suspect the nature of the disorder (*see* TYPHLITIS). Now and then, however, the course from the first is latent, and the mischief is suddenly revealed by perforation into the peritoneum, followed by the symptoms of general and rapidly fatal peritonitis. Adhesions formed in front of the slowly advancing ulceration tend to localise the consecutive inflammation and abscess in other instances.

The substance imprisoned within the appendix may be dislodged (*a*) by an abscess opening externally (such as through the anterior abdominal wall, thigh, or scrotum), or into the abdominal cavity; or (*b*) by the perforated appendix establishing a communication with the cæcum, bladder, rectum, &c. The diseased and distended appendix—especially when its lumen is occluded, and there is little or no consecutive peritonitis—

may be not infrequently felt as a hard elongated body deep in the right iliac region, and can often be discovered by rectal examination, especially in young subjects.

**DIAGNOSIS.**—Inflammatory affections of the appendix can rarely be clearly distinguished from those of the cæcum. Pains radiating to the testis or groin are more common in the form of 'typhlitis' than in the other varieties. The clinical and post-mortem observations of late years have clearly shown that peritonitis in the cæcal region—especially in its severe and relapsing forms—is more commonly the result of ulcerative inflammation of the appendix than of the cæcum. See CÆCUM, Diseases of; and TYPHLITIS.

**PROGNOSIS.**—General peritonitis from sudden perforation into the cavity of the peritoneum is the great danger, recovery from which is extremely rare. Inasmuch as this may occur at any time during the course of ulcerative inflammation of the appendix, a guarded opinion should always be given when there is suspicion of the existence of this affection. Continued uneasiness in the right iliac region without indications of fecal accumulation, should not be regarded lightly. Though this serious accident is less apt to occur after the formation of adhesions around the advancing ulceration, we must not forget that well-marked local inflammation of the peritoneum does not always prevent it, inasmuch as the adhesions which are thus formed may not be sufficiently strong to withstand the pressure of pus in the appendix.

**TREATMENT.**—When there is suspicion of trouble in the appendix the patient must be kept at rest in bed, hot poultices applied, and an unirritating, very restricted fluid diet allowed. Opiates, for the purpose of controlling pain, should be continuously administered; and if irritability of the stomach exist, they should be introduced by enema or suppository, or by subcutaneous injection. Perforation and peritonitis, or other complications, must be treated as they arise. See TYPHLITIS.

**Radical Surgical Treatment.**—It has recently been shown<sup>1</sup> that an offending appendix may be removed, or a deformity of it corrected, with the happiest results in cases of relapsing typhlitis. The operation, performed in the quiescent period after subsidence of inflammatory and other symptoms, has prevented further relapses, and possibly averted a fatal perforative peritonitis. See TYPHLITIS.

GEORGE OLIVER.

**APPETITE, Disorders of.**—In disease the desire for food may be either *lessened* or *increased*; or the appetite may be *perverted*, and a longing for various substances unfitted

for or incapable of digestion may be displayed.

**Loss of appetite.**—*Anorexia* accompanies fevers, almost all forms of congestion of the mucous membrane, and acute or chronic gastritis; and as these affections constantly coexist with other diseases, great variety as regards the desire for food is manifested in various complaints. In acute gastritis there is often not merely a loss of desire for, but a positive aversion to food, and the patient resolutely resists any attempt at obliging him to take either solid or liquid nourishment. In the more chronic forms of gastritis the distaste for food may be only slight; in some cases the appetite is increased, but is quickly satisfied as soon as a small quantity of food is taken. In chronic ulcer of the stomach the appetite, as a rule, remains good, and the patient is only prevented from indulging it by the fear of the pain that will result from so doing. Whenever the secreting structure of the organ is extensively diseased the appetite fails. Thus, in atrophy of the stomach the desire for food generally lessens along with the diminishing strength of the invalid. In cancer of the stomach there is always an extensive destruction of the glandular structure, and loss of appetite is a constant and prominent symptom. It must be remembered that a loss of appetite may be more apparent than real. The physician is constantly consulted on account of this symptom, when a little inquiry will show that the patient is really digesting as much as his system requires, but that by a habit of eating without allowing a proper interval between his meals, or by indulging in food of too nutritious a nature, or in an undue amount of alcoholic stimulants, hunger is prevented. For *Anorexia nervosa* see NEURASTHENIA.

**Increase of appetite.**—*Bulimia* usually occurs where there is a necessity for an increased supply of food. Thus it is common after all febrile diseases, where the stomach has been long inactive. Again, in diabetes, where a large portion of the food is passed off in the form of sugar instead of being converted into the material required to keep up the nutrition of the body, there is an unusually large appetite. A craving sensation is a common symptom in chronic catarrhal gastritis. It probably arises from the irritation set up by the mucus and fermenting substances long retained in the stomach, and is temporarily relieved by eating. The best treatment for such cases is to give alkalis about half an hour before the craving usually occurs, at the same time that the affection of the mucous membrane is combated by appropriate diet and remedies. In some persons the sensation of extreme hunger appears to arise from an irritable condition of the stomach, by which the food is passed into the duodenum before digestion is completed. The sensation is mostly complained

<sup>1</sup> See Treves, *Med.-Chir. Trans.*, vol. lxxi. p. 165, and *The Lancet*, Feb. 9, 1889.

of at night, and the writer has found it a good plan to let the patient have some beef-tea or meat lozenges, for example, either just before retiring to rest or during the night. In children a craving for food is a frequent symptom, and arises either from the irritation of worms, or from chronic catarrh of the mucous membrane of the small intestines.

*Perversion of appetite*—*Pica* is most common in pregnant or hysterical females. Articles, such as chalk, cinders, and slate-pencil, are sometimes swallowed. In the insane and in idiots articles of an indigestible nature are not infrequently introduced into the stomach, such as string, paper, cocoa-nut fibre, &c.

It is a matter of great importance that all persons, but especially dyspeptics, should accustom themselves to control their appetites. Whenever a larger amount of food is taken than the stomach is capable of digesting, the residue is apt to ferment and thereby to produce gastric catarrh. This is more especially the case where the digestive powers have been enfeebled by previous attacks of gastric inflammation. S. FENWICK.

**APYRETICS.**—See ANTIPYRETICS.

**APYREXIA** (*ἀ*, priv.; and *πυρέσσω*, I am feverish.)—This word literally means absence of fever. It is also used to denote the interval between paroxysms of intermittent fever. See FEVER.

**ARACHNITIS.**—Inflammation of the arachnoid membrane. See MENINGITIS.

**ARCACHON, West Coast of France.** Summer and autumn resort. Sheltered by pine-woods. Calm in winter. See CLIMATE, Treatment of Disease by.

**ARCUS SENILIS.**—SYNON.: Geron-toxon.

**DESCRIPTION.**—Arcus senilis is a crescentic or annular opacity just within the margin of the cornea, often seen in old persons. It usually begins as a greyish or whitish crescent at the upper part of the cornea, and subsequently a similar crescent makes its appearance in the lower part. These crescents gradually increase in opacity, in width, and in length, until their horns coalesce and form an *annulus* or ring. A true arcus is not quite continuous with the sclerotic, but is separated from it by a narrow rim of transparent cornea. The breadth of the crescent or ring generally ranges from one-twentieth to one-tenth of an inch. The opacity is more pronounced at its outer border, but fades insensibly towards the central portion of the cornea, which is of normal transparency.

**ETIOLOGY AND PATHOLOGY.**—The proximate causes of arcus are not known. Some families are especially prone to it, and it most frequently occurs in persons of fifty years of age and upwards. The opacity is

commonly believed to be due to fatty degeneration of the corpuscles and fibrillæ of the cornea, and to be indicative of degenerative changes within the heart and blood-vessels and other textures. There is, however, a growing consensus of opinion that both its pathological and clinical significance have been overrated. Certainly it is, in itself, no criterion of age, either as regards years or constitution. On the one hand it may occur in young persons, and on the other hand it may be absent in extreme old age. Again, it may exist in persons free from any appreciable signs of arterial or cardiac disease, and it may be absent from those who have pronounced degeneration of the heart and blood-vessels. It is, nevertheless, true that arcus senilis is due to degenerative changes within the cornea, and that it does sometimes co-exist with atheroma or with fatty heart. According to some investigations made by the writer, in conjunction with Mr. E. T. Collins, in the laboratory of Moorfields Eye Hospital, the change in the cornea consists of the presence of fine, highly refractive molecules, distributed along the course of the lymphatic spaces and channels of the superficial layers of the periphery of the cornea near the loop-endings of the capillaries of the conjunctival and episcleral blood-vessels. The greater portion of these molecules are not fatty, as is generally supposed; for, unlike fat, they are neither blackened by osmic acid nor dissolved by ether. They probably arise from mucoid degeneration of the protoplasm within the lymphatic channels and spaces of the cornea, and to some extent of the fibrillæ themselves. A few doubtful, blackened, fatty molecules may be seen here and there in sections stained by osmic acid. The fibrillæ are slightly wrinkled, and are more loosely held together than natural, and the spaces between the laminae are wider. In support of the non-fatty nature of arcus senilis it may be stated that wounds of the cornea, whether through the opacity, or to its inner side, or to its outer, and whether surgical or traumatic, heal in a natural way.

**TREATMENT.**—The condition is incurable, and is unaffected by any kind of treatment.

JOHN TWEEDY.

**ARDOR** (*ardor*, heat).—A sensation of heat, burning, or scalding. It may be felt either along the urethra during the passage of urine (*ardor urinae*); or in connexion with the stomach (*ardor ventriculi*).

**ARGYRIA** (*ἀργυρος*, silver).—The slate-coloured stain of the skin produced by the internal use of the salts of silver. See PIGMENTARY AFFECTIONS.

**ARKANSAS SPRINGS, in Arkansas, United States.**—Thermal waters. See MINERAL WATERS.

**ARSENIC, Poisoning by.**—Arsenic is classed as a metallic irritant poison, though its action is by no means limited to that of an irritant. It acts specifically on the gastrointestinal mucous membrane, whatever be the channel by which the poison gains access to the system. The most usual source of acute arsenical poisoning is the administration of white arsenic or arsenious acid; but the sulphides, various arsenites, and impure commercial articles, such as dyes, wall-papers, and pigments, may be fertile sources of arsenical poisoning. Poisoning by arsenic may be either *acute* or *chronic*.

**ANATOMICAL CHARACTERS.**—These are the same by whatever channel the poison has gained access to the system. There is marked petechial inflammation of the stomach and duodenum, usually of the small and large intestines also; but not uncommonly the inflammation is limited to the stomach, duodenum, and rectum, the intervening alimentary tract having escaped. If the poison has been administered in a solid form, white patches of the arsenical compound may be found embedded in yellow or bloody mucus and inflammatory exudation. Portions of the white arsenic are also sometimes converted by the sulphuretted hydrogen evolved during decomposition into the yellow sulphide. Ulceration of the stomach is rare. Neuritis may be observed. An ecchymosed condition of the heart is often observed; and fatty degeneration of the liver, as in poisoning by phosphorus, has been described.

**A. Acute Arsenical Poisoning.**—This is the usual form of poisoning ensuing on the nefarious administration of any preparation of arsenic, but usually the oxide (*arsenious acid*) is employed.

**SYMPTOMS.**—The symptoms do not, as in the case of corrosive poisoning, come on immediately after the administration of the poison. There is most commonly an interval of half an hour or an hour between the swallowing of the agent and the onset of prominent symptoms. The quantity of the noxious agent, and its state as regards solubility, have also an obvious relation to the commencement of symptoms. Most commonly, after a sense of faintness and depression, intense burning pain is felt in the epigastric region, accompanied by tenderness on pressure. Nausea and vomiting quickly supervene, increased by every act of swallowing. Unlike what occurs in an ordinary bilious attack, with which arsenical poisoning may be at first confounded, the pain and sickness are not relieved by the act of vomiting. The vomited matters are extremely varied, and present no characteristic appearances. At first they usually consist of the ordinary contents of the stomach, but at a later stage are largely charged with bile which has regurgitated into the stomach in consequence of the violence of prolonged emesis; and they

may be tinged with blood. Ordinarily vomiting is speedily followed by violent purging, and great straining at stool, the motions being often streaked with blood. Purging may, however, be entirely absent. Other prominent symptoms are great thirst, a feeble irregular pulse, and cold clammy skin. The patient, as a rule, dies within eighteen to seventy-two hours, in a state of collapse; but tetanic convulsions are not uncommon, and even coma and paralysis may close the scene.

**DIAGNOSIS.**—From an ordinary bilious attack, induced by improper diet or by decomposing food, arsenical poisoning is diagnosed by the persistence of the symptoms after the removal of the apparent cause; and not infrequently by the symptoms remitting and again supervening on the administration of food or drink of a particular kind, or given by a particular hand. From choleraic diarrhoea it is distinguished by the *sudden* onset of symptoms, thirty to sixty minutes after food or drink has been taken; by the absence of rice-water stools, or of lividity of the skin; and by the symptoms not yielding to treatment. Moreover, in poisoning by arsenic there is usually greater tenderness over the epigastrium; the diarrhoea is less passive, and accompanied with more tenesmus than in choleraic diarrhoea; the stools are more often bloody; and nervous symptoms may be more pronounced. The diagnosis is, however, often very difficult, except when aided by a chemical analysis of the matters ejected from the stomach or of the excreta, which should always be made in doubtful cases.

**PROGNOSIS.**—This must always be uncertain, since it is rarely possible to ascertain the quantity taken, or to ensure its entire evacuation from the stomach.

**TREATMENT.**—Emetics, diluents, and demulcents are the appropriate remedies. The stomach-pump may also be usefully employed. In administering emetics, tartar emetic should be avoided, as it increases the depression, and its presence complicates a chemical analysis. Moreover, tartar emetic sometimes contains traces of arsenic, and, in the event of an analysis being made, an unfounded suspicion may be raised. No confidence can be placed in the so-called antidote, *ferric hydrate*, but dialysed iron may be freely given.

**B. Chronic Arsenical Poisoning.**—This form of poisoning is not uncommon, and is, unlike the acute form, generally accidental. The inhalation of arsenical vapours in factories, or of arsenical dust, as from green and other wall-papers, and in the process of manufacturing artificial flowers, is a common source of chronic arsenical poisoning.

Those who are chiefly exposed to this form of poisoning are persons employed in the manufacture of pigments, especially green

pigments: paperhangers and decorators; artificial-flower manufacturers; milliners; persons exposed to the fumes of heated metals, particularly zinc and brass; manufacturers of dyes; and leather-dressers. In the process of depilating sheep-skins, previous to the tanning or the tawing process, a mixture of lime and orpiment (*sulphide of arsenic*) is used; and serious ulceration of the hands, scrotum, nose, and cheeks not infrequently results. Persons living in rooms the walls of which are covered with arsenical paper, especially bright-green papers containing arsenite of copper, are liable to suffer from chronic arsenical poisoning. It is uncertain whether this is entirely caused by the mechanical transfer of pigmentary dust to the air-passages, or is partly due to volatilisation of the arsenic, probably in the form of arseniuretted hydrogen. Many brown wall-papers also contain arsenic, and arsenious acid is sometimes added to the size; such papers have been known to produce the specific symptoms of arsenical poisoning.

That some persons can take arsenious acid internally with impunity in relatively large doses (arsenic-eating) is now a well-established fact.

**SYMPTOMS.**—The first symptoms of chronic arsenical poisoning are usually loss of appetite, præcordial pains, irritability of the bowels, and occasionally headache. Suffusion of the eyes, a peculiar and characteristic appearance of the conjunctiva, often amounting to actual conjunctivitis, and intolerance of light are early manifested. The muscular power of the limbs is impaired pretty constantly, and actual paralysis, extending upwards from the lower extremities, is occasionally observed (*see PARALYSIS, TOXIC*). A characteristic vesicular eruption on the skin (*eczema arsenicale*) is frequent, as well as irritation of the skin, especially over the neck, scalp, hands, and armpits. Males who handle arsenical preparations are liable to ulcerations of the scrotum and penis, obviously due to a mechanical transference of the poison to the genitals when these are touched. If the source of the disease be not removed, progressive emaciation, exfoliation of the cuticle, and nervous prostration supervene; and convulsions may precede the fatal termination. The effects of green arsenical pigments are sometimes manifested by bleeding from the nose.

**DIAGNOSIS.**—When a patient suffers more or less from the symptoms above described, and is also known to be exposed to any of the sources of danger from arsenical poisoning enumerated above, the diagnosis is not difficult.

**TREATMENT.**—The source of poisoning should invariably be removed. It is found that those who suffer from working in arsenic make no progress towards recovery until they are removed from contact with the

poison. Wall-papers which contain arsenic, and are suspected to be the cause of symptoms, should be taken away. Quinine, or other tonics, iron, and attention to the digestive organs will be needed. Removal to fresh country air is often productive of marked benefit. Soothing lotions to the skin, and careful attention to eroding ulcers, especially of the cheek, may be necessary. Shampooing and warm baths form the best treatment for paralytic lesions.

T. STEVENSON.

**ARTERIES, Diseases of.**—It is important to keep in mind the following anatomical facts in studying the morbid processes to which arteries are subject:—In immediate contact with the blood-stream in arteries lies the *endothelium*—a layer of flattened cells; outside this is the *tunica intima*, composed of elastic tissue, in longitudinal arrangement: together the endothelium and tunica intima constitute the *internal coat* of the older writers. Still more external we find the *middle coat*, made up of muscular fibre arranged transversely, in the larger arteries mixed with elastic tissue. And, most external of all, there is the *external coat*, consisting of longitudinally fibrillated connective tissue.

**1. Acute Arteritis.**—Acute arteritis, affecting a very limited portion of a vessel, and leading to ulceration, occasionally occurs. It is generally due to the propagation of an inflammation from the tissues of the vicinity to the external coat of the vessel, but in some cases it arises from the irritation caused by an embolus, which, becoming detached from a cardiac valve, blocks a distant artery. The late Dr. Moxon has specially drawn attention to its occurrence in the aorta, when the ascending portion of the vessel has been exposed to the impact of a hard, freely moving vegetation on one of the segments of the aortic valve. Dr. Moxon has also described, under the designation *inflammatory mollities*, the occurrence of softening and swelling of the arterial tunics in circumscribed spots, which become flabby and inelastic, and ultimately bulge outwards and form aneurysms. He believed that this condition depends on a peculiar general state, and is the great cause of aneurysm in young, hard-working men. Except such circumscribed inflammatory lesions, we do not meet with any condition of the arterial tunics to which the designation acute arteritis can be applied. A general inflammatory change has, indeed, been described, and the writers have seen the lining membrane of the ascending and transverse portions of the aorta of a bright vermilion hue, strongly suggestive of acute inflammation; but the best observers are agreed in believing that this appearance arises from staining by hæmatin.

2. **Endarteritis obliterans.**—This is an affection of the small arteries which causes narrowing of their calibre by thickening of the internal coat. It is not an independent disease, and is most commonly found in the cirrhotic kidney, where it is usually accompanied by periarteritis. See Gummatous Disease, page 115.

3. **Periarteritis.**—Periarteritis is mostly a chronic change, and consists in the production of diffuse or circumscribed thickenings of the arterial walls by a new formation of connective tissue in the external coat. It is to a periarteritis that Charcot and Bouchard ascribe the lesions which, in their opinion, eventuate in cerebral hæmorrhage. According to these physicians, cerebral hæmorrhage is not usually due to atheromatous decay of the vessels of the brain; but, in the vast majority of cases, to the rupture of miliary aneurysms, which in their turn have been produced by a morbid process beginning in the perivascular sheath surrounding the cerebral vessels, and which, proceeding from without inwards, ultimately involves all the coats of the vessels. See BRAIN, Hæmorrhage into.

4. **Atheromatous Disease.**—This disease, the *endarteritis deformans* of Virchow, is the arterial disease which is most frequently met with, and the one whose consequences are most serious. It may be divided, for purposes of description, into three tolerably well-defined stages:—(a) In the *first* stage we notice, when the vessel is slit open, greyish patches, by which the lining membrane is irregularly thickened; these patches seem to lie on the surface of the membrane, but this appearance is deceptive; the endothelium lies between them and the blood-stream, and is, at least at the beginning of the morbid process, unaffected. The material of which the patches consist is situated in the tunica intima; it is gelatinous, or semi-cartilaginous, in consistence; and is formed, according to Virchow, by an abnormally rapid multiplication of the deeper cells of the tunica intima,—the new growth pushing up this tunic with its superimposed endothelium, and so causing a bulging into the interior of the vessel. But, in addition to these proliferated elements, round cells, derived from the vasa vasorum, are often present in all three coats; and it would appear that the process often begins as an inflammation, and perhaps less commonly as a hyperplasia, of the tunica intima. (b) In the *second* stage the cellular elements of which the new growth is composed undergo a process of fatty degeneration; and in consequence it becomes yellowish in colour and pasty in consistence: it was the paste-like appearance of the mass in this stage which originally gained for the process the designation *atheroma* (*ἀθήρη* = meal). It not infrequently happens that the whole of the internal coat with its endothelium is involved in the softening, and gives way under the pressure of

the blood, leaving an excavation, the floor of which is formed by the middle and external coats of the artery. (c) In other instances, however, the pasty mass, instead of being washed away, becomes the seat of calcific deposit. This is the *third* stage in the process. The appearance of a vessel in which atheromatous disease has reached this stage is very striking: plates, which present to the naked eye the appearance but do not show the minute structure of bone, are observed at intervals in the walls of the vessel, and their sharp spicula project into its interior; in the aorta it is not uncommon to find such plates an inch long and half an inch broad, and in the smaller arteries the calcareous matter sometimes forms a ring round the vessel. In the latter the calcareous particles appear to be deposited in the patch while it is still firm, so that the second stage of the process is wanting.

The changes above described may be confined to the internal tunic, but the two outer coats are often implicated as well. In the media we may have round-celled infiltration, induration, fatty degeneration, and calcification, while the external coat may be thickened by newly formed connective tissue. Indeed according to Thoma, the primary change is a lesion (weakening or atrophy) of the middle coat. See AORTA, Diseases of, page 90.

Atheromatous disease sometimes invades both the aorta and the small vessels, but the aorta may be extensively diseased and the small arteries unaffected; or, on the other hand, the cerebral, temporal, and coronary arteries may be the seat of calcareous change while the great vessels are healthy; occasionally the disease is limited to a few vessels. Next to the aorta, the cerebral, coronary, and splenic vessels, and the arteries of the lower extremities, are prone to this form of arteritis.

**EFFECTS.**—The dangers to which an atheromatous state of vessels exposes the person in whom it exists are varied. The stream of blood is retarded by the projection of the new growth into the vessel, and still more by the destruction of the elasticity of its coats; and hence ensues a failure in the nutrition of the organ, which depends for its supply of blood on the diseased vessel—this is one cause of cerebral softening. When the paste-like mass is washed away it sometimes happens that the blood insinuates itself between the coats of the vessel, producing a dissecting aneurysm; or the portion of the vessel which has been weakened by the removal of the internal coat yields to the pressure of the current, and a sacculated aneurysm is originated; sometimes the diseased vessel bursts. Cerebral vessels, probably on account of the thinness of their walls, are specially liable to rupture when they are the seat of atheromatous change; and occasionally a diseased coronary artery has given way, filling the pericardium with blood. Arteries have been completely

occluded by the deposition of fibrin on the spiculated edges of calcareous plates: this is one of the causes of senile gangrene; and embolic plugging of distant vessels at times results from the detachment of such fibrinous clots, and the washing away of atheromatous débris. Rigidity of the larger arteries from atheromatous change is likewise one of the most frequent causes of hypertrophy of the left ventricle of the heart, on which increased work is imposed in consequence of the destruction of the elasticity of the vessels. Anasarca has not, so far as the writers are aware, been mentioned by any author among the consequences of diseased arteries; but some cases which have come under their observation have led to the conclusion that persistent anasarca, especially of the lower extremities in elderly men, is sometimes mainly due to a diseased condition of the arterial tunics. In the cases observed there was likewise present dilatation with hypertrophy and commencing fatty change of the left ventricle, itself a consequence of the arterial disease; but this seemed insufficient to account for the persistent œdema of the lower extremities.

**ÆTIOLOGY.**—The principal cause of endarteritis deformans is now generally admitted to be overstrain of the vessel. It was formerly thought, and is still held by many, that syphilitic impregnation of the system is a powerful favouring condition; but this opinion rests chiefly on observations made among soldiers, who, in addition to the syphilitic taint, were subject to other influences now known to be adequate in themselves to develop the disease. Intemperate habits and gout appear to be powerfully predisposing causes. They probably render the blood impure; and its passage through the capillary vessels being thereby retarded, the tension of the arterial system is increased. Besides violent exertion, which imposes a strain on the entire arterial tree, there are other influences which act upon certain vessels. Thus the renal arteries are kept overfull in the cirrhotic form of Bright's disease, owing to the destruction of the capillary tufts, and hence atheroma of these vessels is frequently present in that form of renal mischief. One of the writers has on two occasions found extensive calcareous formations in the cerebral vessels of persons in whom cerebral degeneration had followed excessive anxiety and mental effort. The pulmonary artery is very rarely invaded by atheroma; and only in cases in which it has been kept in a state of tension by hypertrophy of the right ventricle or disease of the mitral orifice.

**DIAGNOSIS.**—The diagnosis of atheromatous inflammation of the aorta is discussed in a separate article. The existence of the disease in the arteries of particular organs can only be a matter of reasonable presumption when the patient is past middle life;

when the ascertained causes of atheroma have been in operation; when symptoms of impaired nutrition of the organ are present; and when the organ (the brain or heart) is one the arteries of which are known to be prone to the disease. Calcification of the superficial arteries renders these vessels rigid and tortuous. The temporals when so affected attract the eye by their prominence, and may be felt hard and rigid beneath the finger; the brachial may equally be made the subject of examination; and, although the presence or absence of atheromatous change in such superficial vessels does not necessarily prove that the other arteries of the body are in a similar condition, it renders it more than probable that they are. Those who are not familiar with the resisting feel of the radial artery, when it is the seat of the change now under consideration, are liable to form a very erroneous estimate of the strength of the pulse: this may convey to the inexperienced finger an impression of a force which it does not possess. The error also is sometimes committed of inferring the existence of aortic regurgitation in these cases in consequence of the tortuous course and visible pulsation of the superficial vessels; but they do not collapse suddenly under the finger, as do the vessels during the receding wave in aortic patency. The sphygmographic tracing, moreover, is essentially different; in atheromatous disease of the artery the upstroke is vertical, and the summit of the tracing extended. The existence of such evidences of vascular mischief affords a fair subject for consideration to those who are called upon to form an opinion as to the eligibility of a life for assurance.

**TREATMENT.**—The treatment of endarteritis deformans is mainly preventive. It consists in the avoidance of all those influences to which we have adverted as causes of the disease, namely: indulgence in alcohol; causes originating a gouty state of the blood; excessive muscular efforts, especially in constrained positions; and postures which involve the long-continued contraction of muscles which surround arteries. As far as the brain and heart are concerned, all those states must be forbidden which favour overfulness of their respective arteries: in the case of the brain, excessive mental application, deficient sleep, and, the writers believe, prolonged periods of sexual excitement; in the case of the heart, *inter alia*, efforts which involve holding the breath, thus leading to distension of its right cavities, and imposing an obstacle to the return of blood from its walls.

**5. Fatty Degeneration.**—Fatty degeneration, unconnected with the atheromatous process, is sometimes found to affect arteries. Circumscribed opaque and velvety spots appear on the surface of the intima, and erosion ultimately occurs. Once this has taken place, the muscular coat, which

may itself be fatty degenerated, unable to bear the pressure of the blood-stream, fissures transversely; and the blood may rupture the external coat, or, insinuating itself between the middle and external coat, produce a dissecting aneurysm. The fatty change is met with in extreme anæmia and in senile marasmus, but has also been found in the arteries of persons who seemed otherwise quite healthy; it is 'a morbid change which is simply degenerative from the first, and of whose immediate cause we know nothing' (Rindfleisch). Fatty degeneration of the external coat of the smallest arteries has also been noticed; it appears to be a senile affection.

**6. Calcification.**—This disease of the arterial tunics also occurs unconnected with endarteritis, but more rarely than fatty degeneration. When this is the case, it is most frequently the middle coat of the medium-sized vessels that is the seat of the deposit, but all three tunics may be involved. The process is usually limited to the vessels in which muscular fibre is abundant; but these it may affect extensively, the superficial vessels and the arteries of the brain and of the extremities being the favourite seat. It is eminently a senile change.

**7. Gummatous Disease.**—In this condition the vessels present nodose swellings, and are thickened sometimes to three times their normal size by gummatous material infiltrating the outer coat. According to Heubner, the disease begins in the intima by a proliferation of the cells of the endothelium, which may be so excessive as to cause complete obliteration of the lumen of the vessel. The middle and external coats only subsequently become infiltrated with round cells. The lesion described by him, however, is common to all forms of endarteritis obliterans, and is not distinctively syphilitic. 'A random succession of nervous symptoms,' to use the words of Dr. Hughlings Jackson, affords strong grounds for suspecting syphilitic disease within the cranium; and one of the writers has himself seen three cases in which such symptoms disappeared under the use of perchloride of mercury and iodide of potassium, and in which it seemed to him that the supposition of arterial disease was much more probable than that of any other form of intracranial syphilis.

**8. Albuminoid Disease.**—This, when it attacks an organ, very commonly appears first in the walls of the small arteries, the muscular coat being the part affected.

**9. Hyaline Degeneration.**—This degeneration may attack the smallest arteries, and give rise to a continuous thickening of their coats, with ultimate occlusion of the vessel; or it may result in the production of isolated masses of hyaline material, which lie principally between the tunica intima and tunica media.

**10. Contraction.**—Contraction and final impermeability of an artery from atheromatous calcification, from the accumulation of fibrin on its rough inner surface, from pressure, or from other causes, occasionally occurs, leading to gangrene of the extremity which it supplied.

**11. Dilatation.**—Dilatation of arteries is in the majority of cases due to previous disease of their coats; but sometimes in the aged the arteries are found dilated without any degeneration of their tunics being present,—a state of affairs which Rindfleisch suggests may depend on atony of the muscular coat, and in some cases may possibly be connected with deficient innervation.

**12. Aneurysm.**—Aneurysm receives full consideration in a separate article. Here it is merely necessary to point out the ways in which atheromatous inflammation and the other morbid processes which have been described contribute to the production of dilatation and aneurysm. In some cases the course of events consists in the washing away of the diseased patch of the intima; when the middle coat either dilates, or, by separation of its muscular bundles, undergoes rupture, and the external coat yields before the pressure of the blood-stream. In other cases the dilatation occurs, not at the point where the endarteritis has invaded the vessel, but nearer to the heart. At the affected point there is narrowing of the canal of the vessel, and loss of elasticity in its coats; and, as a consequence, we have slowing of the circulation and deficiency in the supply of blood beyond, and increased arterial tension on the proximal side of the affected spot. The effect of this tension is more serious than would at first sight appear. In health the blood, propelled by each ventricular systole, enters contracted vessels, which, yielding before it, are uninjured by its sudden impact; but a vessel in a state of tension is exposed to the full violence of the column of blood discharged by the heart, and must gradually dilate before it.

**13. Arterial Disease in Insanity.**—According to Dr. J. Batty Tuke, and other physicians who have specially investigated the morbid changes in the brains of the insane, arterial disease is almost invariably present. It consists in such alterations as would result from obstruction in the ultimate ramifications of the vessels: thickening of the proper coats of the arteries, and of the sheath of connective tissue which surrounds the cerebral vessels; the deposition of fine molecular matter and crystals of hæmatoidin between the adventitia and the sheath; and extreme tortuosity of the vessels.

**14. Arterio-Capillary Fibrosis.**—This term was applied by the late Sir William Gull and Dr. Sutton to the hypertrophy of the walls of the small arteries found in the subjects of the cirrhotic form of Bright's disease.

It is admitted by all observers of repute that the walls of the blood-vessels of the kidney are greatly thickened in this malady; but it is by no means so universally admitted that the small arteries throughout the whole body are in all such cases hypertrophied. That they are hypertrophied in a certain proportion of the cases admits of no doubt; but there is a difference of opinion as to the nature of the thickening. Dr. George Johnson, who early called attention to this condition, considers that there is present a hypertrophy of all the tunics of the small arteries, especially of the muscular coat—a consequence of the obstruction which impure blood invariably meets with in the capillaries. Sir William Gull and Dr. Sutton, on the other hand, asserted that the thickening is due to a fibroid growth, especially seated in the external coat of the vessel; and they believed that the co-existing disease of the kidney is not the cause of the arterial change, but that both are parts of a general diseased process.

JAMES LITTLE. A. B. McKEE.

**ARTERIES, Examination of.**—See PHYSICAL EXAMINATION; and PULSE.

**ARTHRALGIA** (*ἄρθρον*, a joint; and *ἄλγος*, pain).—Pain in a joint. The term is more particularly applied to articular pain in the absence of objective disease. See JOINTS, Diseases of.

**ARTHRITIS** (*ἄρθρον*, a joint).—A term generically used to signify any disease whatever involving a joint, but more correctly confined to articular inflammation. It is also employed to designate inflammation of all the structures forming a joint, as distinguished from mere synovitis. See JOINTS, Diseases of.

**ARTHRODYNIA** (*ἄρθρον*, a joint; and *δδύνη*, pain).—Pain in a joint. See ARTHRALGIA.

**ARTICULAR RHEUMATISM.**—Rheumatism affecting joints. See RHEUMATISM, ACUTE.

**ARTIFICIAL RESPIRATION.**—The method of exciting and keeping up the movements of the chest, so as to supply air to the lungs, is a subject of the highest importance, since the hopes of recovery depend on its due performance in many cases of narcotic poisoning, in the apparently drowned or asphyxiated, and in the collapse of the advanced stage of the condition induced by anæsthetics.

**PRECAUTIONS.**—For its effective employment it is essential to see that no foreign body obstructs the air-passages. Children and old people are liable to swallow large pieces of meat or crust, which become impacted in the pharynx or œsophagus. A clot of blood may threaten life in operations about the mouth. These should, if possible, be dragged away with the finger or a spoon-handle, but they may require the use of a probang.

Tracheotomy is rarely necessary. A knife-handle held between the molar teeth is a ready and useful gag to keep the mouth open. A button-hook, in the absence of pharyngeal forceps, is sometimes very serviceable. Vomited matter should be quickly removed with a sponge or cloth twisted round a piece of wood. In treating the half-drowned the body should be inverted for a few minutes to favour the escape of water from the air-passages, but artificial breathing should be commenced even whilst the body is in this position.

**METHODS.**—In most cases the best method of commencing artificial respiration is to compress the chest and abdomen simultaneously, then remove pressure so as to allow air to enter the chest, and again repeat the pressure every two or three seconds. If the sound indicates that air is passing into and out of the lungs, this method may be continued for half a minute; but if we are not sure that the air is exchanged, and in all cases if the patient's condition is not decidedly improved in half a minute, we should resort to one or other of the following methods:—

1. *Howard's method.*—This method is so important that it is considered separately under RESUSCITATION.

2. *Sylvester's method.*—Place the patient on his back on the floor, with a block or pillow under his shoulders, and raise the arms upwards above his head, by grasping them above the elbow, and pulling firmly and steadily as long as there is any sound of air entering the chest. Some arrangement is needed to prevent the body from being dragged towards the operator. For this purpose the plan of raising the chest on a high cushion or box has been adopted, but as a condition of cardiac anæmia is often present, this is objectionable. It is better to effect the object by placing a book in front of the thighs while kneeling at the head of the patient. It may be needful to draw forward the tongue; but generally if the head falls back over a cushion placed behind the neck, this is not required. An artery-forceps, or a noose of string, or a handkerchief will enable an assistant to keep the tongue well forward.

As soon as the sound produced by the entrance of air into the chest ceases, the arms should be brought down a little towards the front of the chest, and pressed firmly and steadily against it for about one second after air is heard escaping. In cases of drowning it is enough to repeat this operation every four seconds, but in the collapse resulting from chloroform or other anæsthetics, the necessity for getting the vapour quickly out of the chest justifies a more rapid performance of the movements during the first five minutes. After this time the movements should be carried on more slowly, but they should be continued for half an hour at least, and even longer if the warmth of the surface

and diminution of lividity gives any reason to hope that the heart has not entirely ceased to act.

3. *Marshall Hall's ready method*.—This is performed by placing the body on one side, and alternately rolling it on its face to compress the chest, and on its back to allow the elasticity of the ribs free movement to draw air into the lungs. The plan is not nearly so effective as Sylvester's, but if no assistant is at hand it is the best mode of artificial breathing that can be adopted.

4. *Mouth-to-mouth insufflation*.—This method is not to be depended upon, because of the difficulty both of keeping the larynx open, and also of preventing the air going down the gullet.

Of the *instruments* introduced for the purpose of carrying on artificial respiration, mention should be made of those invented by Dr. Marcet, Dr. Richardson, and Dr. Vanderburg; but, except in the hands of the inventors or of those who had gained much experience in their use by practising upon animals, they would probably do as much harm as good. The objection to them all is that they interfere with the prompt imitation of the movements of respiration just described.

The administration of *oxygen* is indicated in most cases of artificial respiration, but the results of its use have not been satisfactory hitherto. Now that the gas can be had in a compressed state, and can be given by means of the laughing-gas inhaler, it is worthy of a further trial; but it is certain that in all cases of impending asphyxia time is of so much importance that anything which would delay the supply of oxygen would not be compensated for by giving it pure, instead of in the form of common air. Tracheotomy is not to be thought of in the first instance in any case in which air can be made to pass, even in very small quantity, through the trachea.

For supplemental and after treatment, see RESUSCITATION.

J. T. CLOVER. G. H. BAILEY.

### ASCARIDES.—See ENTOZOA.

**ASCITES** (*ἀσκίς*, a leathern sac: a large belly).—SYNON.: Dropsy of the peritoneum; *Hydrops peritonei vel abdominis*; *Hydro-peritonæum*. Fr. *Ascite*; Ger. *Die Bauchwassersucht*.

**DEFINITION**.—An accumulation of fluid within the cavity of the peritoneum, more or less serous in character; the accumulation being of the nature of a local dropsy, and not originating in inflammation. The amount of fluid varies much in different cases.

**ETIOLOGY AND PATHOLOGY**.—The chief matter relating to the causation of ascites is to point out the morbid conditions by which it may be produced, as it almost always follows, and is a consequence of certain pre-existing organic diseases, of which it becomes

a most important symptom and pathological phenomenon. The causes to which it has been attributed may be discussed according to the following arrangement:—

I. Direct mechanical obstruction affecting the portal circulation.

1. Obstruction of the trunk of the portal vein before it enters the liver, either from external pressure or an internal block.

2. Pressure upon or obliteration of the branches of the vein within the liver.

3. Pressure upon the hepatic veins, or upon the inferior vena cava after it receives these veins.

II. Cardiac or pulmonary diseases obstructing the general venous circulation.

III. Disease of the kidneys.

IV. Morbid conditions of the peritoneum.

V. Miscellaneous.

I. Any direct obstruction interfering with the portal circulation must necessarily lead to congestion and over-distension of its tributaries, one of the consequences of which is excessive transudation of the fluid portion of the blood into the peritoneal cavity, while absorption is checked. The ascites is, under such circumstances, in short, merely a localised dropsy, resulting from mechanical congestion. The impediment may affect either the portal trunk before it enters the liver; its branches in the substance of this organ; or the hepatic veins or the inferior vena cava near its termination.

1. The portal trunk may be pressed upon, as it lies in the fissure, by prominences from the liver itself, enlarged absorbent glands in its vicinity, a neighbouring tumour (as cancer of the pancreas, or a growth in the small omentum), a hepatic aneurysm, or inflammatory thickening resulting from peri-hepatitis. The pressure may absolutely close up the vessel, but it more commonly causes a local clot to form, and thus its channel is blocked up. A thrombus is also in exceptional instances produced in connexion with a diseased condition of the portal vein, such as inflammation or calcification; obstruction to the circulation within the liver; or feebleness of the circulation, with an abnormal tendency to coagulation of the blood.

2. Pressure upon, or obliteration of the branches of the portal vein within the liver, can only arise as a consequence of some morbid condition involving the actual substance of this organ. The hepatic disease which by far most commonly leads to this result, and which is one of the most frequent causes of ascites, is cirrhosis. Occasionally it accompanies syphilitic and other forms of contracted and indurated liver, or it may be associated with infiltrated cancer. The extent of the obstruction thus set up will necessarily vary with that of the morbid changes in the organ. Occasionally a mass within the liver obstructs a considerable branch of the portal vein.

3. Obstruction of the hepatic veins, or the upper end of the inferior vena cava, is a rare event, but may arise from the pressure of a growth connected with the liver itself, or of some neighbouring tumour.

II. Diseases of the lungs or heart which impede the general venous circulation must necessarily exercise a speedy and direct influence upon the hepatic circulation, and may thus lead to ascites. Usually, however, in cases of this kind the legs are the seat of considerable anasarca before peritoneal dropsy is observed. In course of time the continued venous congestion originates serious organic changes in the liver, its vessels being more or less obliterated, and consequently it is at this period that ascites is particularly liable to set in, and it may even become the most prominent form of dropsy.

III. Ascites may constitute a part of the dropsy which so often accompanies renal disease. It is, however, of comparatively infrequent occurrence to any great extent under these circumstances, the amount of fluid being not considerable as a rule, and the ascites being but a subsidiary part of a general dropsy.

IV. More or less serous effusion into the peritoneal cavity is a pathological result of peritonitis; but, in accordance with the definition of ascites given above, this does not come strictly within the present article. Exceptional cases do occur, however, in which a large effusion rapidly collects in the peritoneum, and it is difficult to say whether it is a mere dropsy or the product of an acute inflammation. Moreover, true ascites is observed occasionally as a sequel of peritonitis, in consequence of the morbid conditions which it leaves behind. Chronic peritonitis may also occasion a simple local dropsy; but this is particularly liable to be set up in connexion with morbid formations in the peritoneum, such as cancer or tubercle. The immediate causes of ascites associated with diseases of the peritoneum may be active congestion; implication of the capillaries or minute veins, or even of the larger veins, leading to mechanical congestion; obstruction of the lymphatic orifices, and consequent impairment of absorption; or undue activity of the secreting structures.

It may be mentioned in this connexion that ascites is sometimes associated with a tumour which does not directly interfere with the portal circulation; probably it is then due to irritation of the peritoneum.

V. Among the chief *miscellaneous* causes to which ascites has been attributed may be mentioned exposure to cold or wet; the sudden suppression of habitual discharges, or the rapid cure of chronic cutaneous affections; and extreme anæmia and debility. These causes are supposed to originate this symptom either by inducing active internal congestion; or by disturbing the renal functions; or in

consequence of the abnormal state of the blood and tissues. It is very doubtful, however, whether either of them can actually of itself occasion ascites, though they may help in its production. Fluid may collect within the peritoneum as the result of the rupture of a cyst within the abdomen, especially an ovarian cyst.

It must be remembered that ascites may be due to a combination of two or more of the causes which have been indicated in the preceding remarks. For instance, there may be obstruction affecting the portal circulation within the liver and outside this organ at the same time; or the different organs may be involved simultaneously.

*Predisposing causes.*—Whatever tends to set up either of the morbid conditions which originate ascites, may be regarded as a predisposing cause. It may be met with at any age, but is most common during middle life. The hepatic form is much more frequent in males than females. An anæmic condition of the blood and weakness of the tissues predispose to peritoneal dropsy, as they do to dropsy in other parts.

*ANATOMICAL CHARACTERS.*—The essential anatomical character of ascites is the accumulation of a serous fluid within the peritoneal sac. Its amount may range from a few ounces to some gallons. As regards physical characters, the fluid is generally thin, limpid, and watery in consistence; colourless or slightly yellow; clear and transparent; and of alkaline reaction. In exceptional instances, however, it may be coloured by blood or bile; or more or less turbid and dirty-looking or milky (chylous); or of unusually thick and somewhat gelatinous consistence. Soft fibrinous masses occasionally float in the fluid, or these may form spontaneously when it is allowed to stand. Very rarely the reaction is neutral or acid. The specific gravity varies considerably. Chemically the fluid consists of water holding in solution albumen and the usual salts which are found in dropsical fluids; but their proportion is very variable, though the albumen is generally in good quantity, which is evidenced by the degree of coagulation which takes place when the fluid is boiled. Occasionally it contains fibrin, cholesterolin, bile-elements, or, in cases of renal dropsy, urea.

The effects of the accumulation upon surrounding structures are to distend and macerate them more or less, or to compress them. Of course along with the ascites there will be the signs of any morbid condition upon which it depends; and there may also be indications of anatomical changes resulting from long-continued pressure of the fluid upon certain structures.

*SYMPTOMS AND SIGNS.*—Ascites usually sets in very gradually, being chronic in its progress, but advancing steadily. Occasionally, however, the fluid collects with considerable rapidity. The clinical phenomena associated

with this pathological condition differ in different cases, both in their exact nature and their degree, according to its cause, the amount of fluid collected, and other circumstances; but they may conveniently be considered under the following heads, namely: 1. *Physical signs*. 2. *Mechanical effects of the dropsical accumulation*. 3. *General symptoms*.

1. *Physical signs*.—Physical examination constitutes one of the most important parts of the clinical investigation of cases of ascites, and it will be requisite to discuss in some detail the signs thus recognised.

(a) If fluid collects in the peritoneum in any quantity, the abdomen presents more or less general enlargement. This is often the first change which attracts the patient's attention, and it may also have been noticed that the increase in size commenced below. The degree of enlargement depends upon the amount of fluid, but it may become extreme, so that the skin is tightly stretched and thinned, presenting a smooth and shining appearance, or sometimes white lines—*lineæ albicantes*—due to laceration of its deeper layers. The umbilicus becomes affected in a characteristic manner, being more or less stretched and everted, and finally obliterated, or in some cases more or less pouched out, when it may form a considerable prominence. Should there happen to be a weak portion of the abdominal walls, such as a hernial sac, this will be unduly protruded. The important characters of abdominal enlargement, due to uncomplicated ascites, are that it is of a rounded form, though tending to be more prominent or to bulge towards the hypogastric region or in the flanks, according to the posture of the patient; that it is quite symmetrical in shape, when the patient stands or lies on his back, but that the form alters considerably with a change of position, the abdomen becoming then more prominent in the dependent region, in consequence of the gravitation of the fluid in this direction, and it may actually be seen to move as the posture is changed. In contrast with the enlarged abdomen, the chest often looks small and depressed, and the fluid may cause the margin of the ribs to become everted, or it may push forwards the xiphoid cartilage. Mensuration is of service for giving more accurate information as to the size of the abdomen in cases of ascites, and for determining their progress.

(b) The abdomen feels perfectly smooth and even over its entire surface. It usually gives a sensation of tension of the walls, without any hardness underneath. In some instances an obscure feeling of fluctuation is experienced on palpation with the fingers.

(c) The tendency of ascites is to interfere with the abdominal respiratory movements, if it is at all considerable, by preventing the diaphragm from acting properly. At the

same time the writer has not uncommonly observed that, even in cases where the accumulation of fluid has been very considerable, abdominal respiration did not seem to be obviously impeded.

(d) *Percussion* affords some of the most important signs of peritoneal dropsy; and when the fluid is present only in small quantity, this is the only mode of examination that can be relied upon for its detection. In the first place, marked dulness is elicited over the seat of the fluid; while a tympanitic sound, which is often abnormally clear and distinct, is heard over the intestines. When there is but little fluid, it may be impossible to detect any abnormal dulness as the patient lies in the recumbent posture; but on placing him on his hands and knees, the fluid gravitates towards the front of the abdomen, and dulness may then be noticed in the umbilical region. In most cases, however, there is no difficulty in making out the dulness, and this sign is observed in those regions towards which the fluid naturally gravitates. Hence, when the patient lies on his back, the lower part and sides of the abdomen are dull, while its upper and front part is tympanitic. As more and more fluid collects, so the dulness increases in extent, gathering in, as it were, from below and from the sides, until finally the entire abdomen may be dull, except the umbilical region, which usually remains tympanitic to the last. The boundary line between the dulness and tympanitic sound is usually well-defined. As the posture is changed, so will the site of the dulness vary, the part which is undermost presenting this sign, while that which becomes highest is tympanitic, and thus the relative situation of these two sounds, as well as the shape of the dulness, can be altered in a variety of ways. When the patient sits up, the prominence between the recti muscles gives a tympanitic sound on percussion. In exceptional instances a distended colon gives rise to a tympanitic sound along each side of the abdomen, even when there is abundant fluid present.

Another important sign brought out by a form of percussion is the sensation specially termed fluctuation, which is the peculiar wave-like movement realised on placing the fingers of one hand over one side of the abdomen, and flipping or tapping the opposite side with the fingers of the other hand. This sensation is very easily brought out if there is much fluid present, provided it is free to move, and sometimes the motion is actually visible. Change of posture will modify the situation over which fluctuation can be produced.

(e) *Auscultation* yields negative results in cases of ascites, there being no sound of any kind heard over the abdomen.

(f) In the large majority of cases ascites is clearly revealed by the physical signs already described. In exceptional instances,

however, when the diagnosis is obscure, it is requisite to resort to a *digital* examination through the *rectum*, and in females through the *vagina*. The fluid collects in the rectovesical pouch, and on examination *per rectum* the finger detects the sensation yielded by this fluid through its anterior wall. The vagina is usually felt to be shortened, while the uterus is pushed down and flexed. In extreme cases of ascites the posterior wall of the vagina, or even the uterus itself, may protrude through the vulva.

(g) Now and then it is requisite to make use of the aspirator or a small trocar, by the aid of which not only can it be determined whether fluid is present in the abdominal cavity, but its nature can also be ascertained. This method of examination is further useful when ascites is associated with some other morbid condition within the abdomen, which frequently cannot be made out so long as the fluid remains in the peritoneum.

It must be borne in mind that the ordinary physical signs of ascites will be materially modified or obscured under certain circumstances. For example, the quantity of fluid may be so small that most careful examination is required in order to detect its presence; on the other hand, it may be so abundant that dulness is observed over the entire abdomen, and fluctuation may be very indistinct. The existence of peritoneal adhesions—for instance, those which may be formed as the result of repeated paracentesis—also renders some of the most characteristic signs of ascites very ill-defined. Again, the association of peritoneal dropsy with some other abdominal morbid condition, such as a new growth, an enlarged liver or spleen, or an ovarian tumour, will also modify the signs elicited. The mesentery may be abnormally short, or the intestines may be adherent, and thus prevented from floating forwards, so that the usual relative positions of dulness and tympanitic sound are not observed.

**2. Mechanical effects of the dropsical accumulation.**—The clinical phenomena resulting from the mechanical effects of ascites are both subjective and objective. The patient often experiences a feeling of uneasiness and discomfort in the abdomen, as well as more or less tension and fulness, if there is much fluid present; while there may be a sense of fatigue and aching about the loins or abdominal walls. As a rule no particular pain is felt, but colicky pains are liable to occur from time to time, and extreme distension of the structures constituting the abdominal wall may also cause painful sensations. In exceptional instances peritonitis is set up. When the fluid is abundant, the patient is conscious of its weight when he walks; and during progression he throws the head and shoulders back, at the same time keeping the legs apart. Symptoms connected with the

alimentary canal are of common occurrence, but these are often to a great extent due to the same cause which originates the ascites, though the fluid must necessarily tend to interfere with the functions of the stomach and intestines. The bowels are usually constipated, but in some instances diarrhoea or dysenteric symptoms may arise. Flatulence is very commonly complained of, even a small amount of gaseous accumulation in the intestines being felt unduly, producing much discomfort, and increasing the enlargement of the abdomen temporarily. Occasionally vomiting occurs, in consequence of interference with the stomach. When considerable fluid has remained in the peritoneum for some time, it presses upon the inferior vena cava and prevents the return of blood through this vessel, and may thus lead to anasarca of both lower extremities, with enlargement of the superficial abdominal veins. Exceptionally the anasarca attracts attention at an early period. The flow of blood through the renal veins may also be obstructed, inducing mechanical congestion of the kidneys, with consequent diminution in the quantity of urine and sometimes albuminuria. In rare instances the fluid has been known to accumulate to such an extent as to rupture some part of the abdominal wall.

Ascites also frequently interferes with the thoracic organs. The bases of the lungs are more or less compressed, and the breathing becomes chiefly upper-costal, while a sense of dyspnoea is experienced, especially in the recumbent posture and after taking food, the breath is short on exertion, and the respirations are often hurried and shallow. The heart is likewise liable to be disturbed in its action, as evidenced by palpitation, irregularity, or a tendency to faintness. This organ may also be displaced, so that its apex-beat is raised and too far towards the left, and in rare instances a basic systolic murmur has been originated as a result of this displacement.

**3. General symptoms.**—The general system is frequently seriously affected in cases in which ascites is a prominent symptom, but this usually depends upon the cause or causes which have originated the dropsy, though it may itself induce more or less debility, wasting, anæmia, and other general effects. The loss of fluid in this way has also been supposed to lead to deficient perspiration, and consequent dryness of the skin; as well as to diminution in the quantity of urine.

**DIAGNOSIS.**—The first matter bearing upon the diagnosis of ascites is to determine whether this morbid condition actually exists. The presence of fluid in the peritoneum, as well as its amount, can only be positively made out by physical examination, and in the great majority of cases the signs thus elicited are quite characteristic. When the fluid is small in quantity, as well as under

other circumstances in which the physical signs are obscured or modified, the diagnosis may be difficult and uncertain, but it may then be aided by a knowledge of the existence of some disease likely to give rise to ascites, of which it is really but a symptom. That the accumulation of fluid is of a dropsical nature, and not due to acute or chronic peritonitis, is usually sufficiently obvious from the history of the case, and the collateral symptoms, while the local signs are also of a different character (*see* PERITONEUM, Diseases of). The remaining abdominal enlargements from which ascites has to be more commonly distinguished are those due to flabby relaxation of the walls of the abdomen, combined with flatulence; accumulation of fat in the subcutaneous tissue and in the omentum; abundant subcutaneous œdema, which may be associated with and obscure ascites; an ovarian cyst; or a pregnant uterus. Among the more rare conditions with which ascites is liable to be confounded may be mentioned a greatly dilated stomach; colloid disease of the omentum; distension of the uterus with fluid; great accumulation of urine in the bladder; a very large hydatid tumour, usually connected with the liver; extreme cystic enlargement of the kidney; and the so-called 'phantom tumour.' Most of these conditions are described in other parts of this work, and their several diagnostic characters, need not be discussed here; but a consideration of the history and existing symptoms of each particular case, combined with the results of an adequate physical examination, constitute the data upon which the diagnosis is founded. It must be remembered that ascites may coexist with other morbid conditions in the abdomen, their physical signs being combined. Should there be an enlarged organ or other solid mass, it may often be recognised by making sudden firm pressure with the fingers over the corresponding part of the abdomen, when the fluid is pushed aside, and the underlying resistance can be felt; or paracentesis may be performed, and further examination carried out after the evacuation of the fluid.

Another most important point in the diagnosis of ascites is to make out its cause. For this purpose all the facts bearing upon the case must be taken into account and carefully weighed, special attention being paid to the liver and the structures in its vicinity, to the heart, and to the kidneys. The amount of the ascites, and its relation to other forms of dropsy, afford considerable aid in the diagnosis. If it results from cardiac or renal disease, ascites always follows dropsy in other parts of the body, to which it is also generally subordinate; when it is due to hepatic disease or some neighbouring morbid condition, the peritoneal dropsy appears first, and is, as a rule, throughout most prominent. Should the

vena cava inferior be obstructed at its upper part, anasarca of the legs will be observed simultaneously with, or even before, the ascites.

**PROGNOSIS.**—The prognosis of ascites will mainly depend upon its cause; the amount of fluid present; the state of the patient; the condition of the principal organs; and the results of treatment. In some cases this symptom is in itself attended with urgent danger, on account of the mechanical effects of the dropsical accumulation, especially upon the thoracic organs, and still more if these organs are in a diseased condition. In other instances it tends to reduce the patient, and thus to bring about a fatal termination. When ascites is, due to local interference with the portal circulation, great relief can unquestionably be afforded in a considerable number of cases, and life may be prolonged by appropriate treatment; while, if the local cause is not such as in itself to lead to a fatal issue, the ascites may not infrequently be permanently cured.

**TREATMENT.**—The principles of treatment applicable to cases of ascites are: (*a*) to treat the conditions upon which the dropsy depends, and thus endeavour to get rid of its cause or causes; (*b*) to promote absorption of the fluid; (*c*) to improve the constitutional condition and the state of the blood, if necessary; (*d*) to remove the fluid by operation, if absorption cannot be effected; and (*e*) to treat any symptoms needing special attention.

(*a*) As an important part of the treatment directed to the causes of ascites, particular attention must be paid to the state of those organs which are most commonly accountable for this symptom, though unfortunately but little effect can, as a rule, be produced on the dropsy in this way.

(*b*) Absorption of ascitic fluid is chiefly promoted by acting freely upon the bowels, skin, or kidneys. The class of remedies indicated will vary in different cases, and must be adapted to the state of the different organs; but, as a rule, active hydragogue purgatives are most efficient in relieving ascites, especially when due to local causes. The most useful are compound jalap powder, cream of tartar, elaterium or compound elaterin powder; but calomel, gamboge, resin of podophyllum, and saline purgatives may be of service in particular cases. These agents must, however, be administered with caution. In certain instances balsam or resin of copaiba has proved useful as a diuretic in the treatment of peritoneal dropsy. Assistance may be derived in certain forms of ascites from acting upon the skin by means of various diaphoretic baths. Digitalis and squill may be of service as diuretics; the application of poultices of digitalis leaves over the abdomen is occasionally attended with benefit. The administration of iodide of potassium also seems to aid absorption in some cases, more

especially if there be any tendency to syphilitic disease of the liver. It has been recommended to treat ascites merely by a skim-milk diet, without any medicine. Two marked cases were thus treated by the writer with complete success, the fluid being entirely absorbed. In other cases, however, no effect was produced. The employment of galvanism in connexion with the abdominal walls has been found efficacious in producing absorption of peritoneal dropsy in some instances.

(c) Treatment directed to the general condition of the patient, and to the state of the blood, is essential in many cases of ascites. Tonics are often of decided service, and preparations of iron are specially indicated for improving the quality of the blood, if there is a tendency to anæmia. Not only do these remedies sustain the patient, but they may also have an influence in promoting the process of absorption. The diet must be adapted to the circumstances of the case, but usually needs to be of a nutritious character.

(d) In a considerable proportion of cases, however, no effect is produced upon the dropsical accumulation by any of the measures thus far considered. Then it becomes necessary to determine whether it is desirable to remove the fluid by operation. The fluid may be taken away either by means of the aspirator, by the ordinary trocar and cannula, by Southey's trocars, or by a special apparatus. The advisability of having recourse to this plan of treatment must depend upon circumstances. The ascites is frequently not sufficiently abundant to justify paracentesis, and when the condition is of cardiac or renal origin, the operation can, in the majority of cases, only afford temporary relief, so that there is no object in resorting to it unless the mechanical effects of the accumulation are such as to cause troublesome or dangerous symptoms, and it had better be delayed as long as possible. When ascites is a local dropsy, the fluid is often so considerable in amount as to necessitate its removal for the mere purpose of giving relief for the time. In cases of ascites associated with malignant disease, for instance, this is all that can be hoped for, as the fluid will certainly collect again. When, however, the condition is due to some local disease which is not in itself fatal, and especially to cirrhosis of the liver, the writer has found signal benefit result from the repeated performance of paracentesis, and has for a long period advocated this plan of treatment as a curative measure, so far as the ascites is concerned. Rarely does the operation give rise to any immediate ill-effects, and it is frequently found that remedies will act much more efficiently after the removal of the pressure caused by the fluid than they did previously. In the writer's experience paracentesis, repeated as often as the fluid re-accumulated, has ultimately led to a complete cure in several

instances; in others the cure was partial, a certain quantity of fluid remaining in the peritoneum, limited by adhesions; while in others still, life has been greatly prolonged, and much comfort afforded. The repeated accumulation does not seem to affect the system materially by reason of 'the drain upon it, and frequently not at all. Of course due care must be exercised in the performance of the operation, and in the subsequent treatment. In a few days after the removal of the fluid, the application of a bandage firmly round the abdomen, so as to exert even pressure, may prove of service in aiding the absorption of what remains, and preventing the recurrence of the ascites; and this measure may also be useful when a certain amount of fluid continues after the repeated performance of paracentesis. When the ascites is not cured by repeated tapping, advantage has been found in some instances from allowing the fluid to drain away continuously, and this measure may be worthy of trial.

(e) The symptoms resulting from ascites which are likely to require attention are those connected with the alimentary canal; dyspnoea; and cardiac disturbance, or a syncopal tendency. These should be treated on ordinary principles; but it must be observed that marked dyspnoea or cardiac disorder, if evidently due to the fluid, is an indication for the immediate performance of paracentesis.

FREDERICK T. ROBERTS.

**ASHEVILLE**, in North Carolina.—Situating between the Blue Ridge and Alleghany Mountains, in 35° 36' N. lat., 2,250 feet above the sea. A mild winter and cool summer climate; of repute in the treatment of phthisis. Temperatures: summer, 70-7°; winter, minimum has never fallen below 8° Fah.; range 80°. Rainfall, 40 inches. Possesses a sanatorium. See CLIMATE, Treatment of Disease by.

**ASIATIC CHOLERA**.—See CHOLERA, ASIATIC.

**ASPHYXIA** (ἀ, priv.; and σφυξις, pulse).—SYNON.: Apnoea; Fr. *Asphyxie*; Ger. *Erstickung*.

**DEFINITION**.—The term *asphyxia*, though literally signifying *pulselessness*, is generally understood to mean the condition that supervenes on interruption of the function of respiration. The term *apnoea*, preferred by many as a more exact one, has the disadvantage of being employed by physiologists in a totally different sense, viz. the cessation of the respiratory movements consequent on artificial hyperoxygenation of the blood. There is therefore no advantage to be gained by substituting the term *apnoea* for the well-understood and older one, *asphyxia*.

**ÆTIOLGY**.—Asphyxia may result from many causes which obstruct or interrupt the

respiration. They may be divided into two categories, internal and external.

*Internal.*—These include paralysis of the respiratory nerve-centres by disease or injury of the medulla oblongata; paralysis of the nerves or muscles of respiration; a rigid fixation of the respiratory muscles; collapse or disease of the lungs; occlusion of the air-passages by organic disease or spasm of the glottis, pressure of tumours, and the like.

*External.*—To this group belong occlusion of the air-passages by foreign bodies; pressure on the chest not capable of being overcome by the muscles of respiration; closure of, or external pressure on, the air-passages, as in suffocation, strangulation, or hanging. These are all cases of obstruction of the respiratory movements in a medium capable of supporting life. To these external causes are to be added those conditions in which, though the respiratory movements are free, the surrounding medium is incapable of oxygenating the blood, viz., submersion in a liquid medium (drowning); or being surrounded by a medium devoid of oxygen, such as nitrogen or hydrogen. These gases have a purely negative effect; but many other gases which are classed as asphyxiants, such as carbonic oxide, sulphuretted hydrogen, chlorine, chloroform vapour, &c., have positive poisonous effects, and should therefore be called by some special name, such as *toxic asphyxiants*, to distinguish them from those which have no such properties.

*PHENOMENA.*—When an animal is placed in an atmosphere devoid of oxygen, or not containing a sufficient quantity of this gas (under 10 per cent.) to maintain the respiratory process, or if the mechanism of respiration is simply obstructed, it begins to show signs of agitation, and to make powerful inspiratory and expiratory efforts, in which the accessory muscles of respiration are all brought into action; the arterial tension increases; and the superficial veins become distended and livid.

After a variable period these dyspnoic efforts pass into general convulsions, in which the muscles of expiration are more especially in action, during which the sphincters are forced, and the excretions voided. On these there follows a calm, during which the animal lies insensible, with dilated and immovable pupils, and with reflex excitability abolished generally. At the same time the pulse is small, feeble, and almost imperceptible. All muscular movements cease except those of inspiration, which are repeated at intervals. As death approaches, the respiratory movements become shallower and less regular, and are succeeded by stretching convulsions, during which the back is straightened, the head is thrown back, the mouth gapes, and the nostrils dilate. The heart still continues to beat after other

movements have ceased. The heart ultimately stops in the state of diastole. Death is then complete and final.

*COURSE AND TERMINATION.*—The time necessary to bring about a fatal termination varies in different animals, and in the same animal under different conditions. It has been noted that the young of some animals resist asphyxia longer than the adults. The late M. Paul Bert has shown that these differences are all explicable in accordance with the law that the more active the vital combustion, the greater the gaseous interchange, and therefore the more rapidly fatal the obstruction of the respiratory process. Excluding special considerations of this kind, it may be stated, as the result of the experiments of the Medical and Chirurgical Committee on Suspended Animation (*Med.-Chir. Trans.* vol. xlv. 1862), that when the respiration of a warm-blooded animal is totally obstructed, all external movements cease in from three to five minutes, and the heart stops within ten minutes. Certain modifications occur according to the method in which asphyxia is produced. See DROWNING.

*ANATOMICAL CHARACTERS.*—The blood is of a dark colour, owing to complete reduction of the hæmoglobin, and the proportion of carbonic acid is greatly increased. Owing to the excess of carbonic acid, the blood coagulates slowly or imperfectly; hence it remains long fluid, or forms few and soft coagula. The venous side of the heart, the great venous trunks, and the pulmonary artery are distended with dark blood; whilst the left side is empty, or contains only a small quantity of dark blood.

The appearance of the lungs is not constant. These organs are by no means always congested, as is very generally stated, being more often pale and anæmic. The posterior and dependant parts become hypostatically congested *post mortem*. The abdominal viscera are usually congested. The appearance of the brain varies, this organ being either anæmic or more or less congested. Special signs characterise special modes of causation of asphyxia.

*PATHOLOGY.*—Inasmuch as the cessation of respiration means both oxygen-starvation and accumulation of carbonic acid, the question is whether the phenomena of asphyxia depend on the one or the other, or on both. Various opinions have been entertained on this subject, but the experiments of Rosenthal and Pflüger would seem to show that the deprivation of oxygen is the chief factor. That the accumulation of carbonic acid has no effect at all cannot, however, be maintained, for it is demonstrable that carbonic acid has a distinct toxic effect on living tissues.

The circulation of non-oxygenated blood through the lungs and the respiratory centre in the medulla oblongata is the cause of the

powerful respiratory efforts in the first stage—directly, by stimulation of the respiratory nerve-centres; indirectly, by peripheral irritation of the pulmonary branches of the vagi. The respiratory movements increase in force, and the irritation irradiates into the centres of other movements besides those directly concerned in respiration, giving rise to the expiratory convulsions which have been ascribed by some to excitation of a special 'convulsion-centre' (*Krampfcentrum*).

The respiratory centres ultimately become paralysed, but subsequently to those of conscious activity—the brain; and of reflex action—the spinal cord.

The circulation of non-oxygenated blood likewise causes contraction of the arterioles from irritation of the vaso-motor centre. Increased resistance is thus offered to the heart, and this is intensified by the convulsive muscular efforts. The arterial tension rises. The resistance to the flow through the capillaries by contraction of the minute blood-vessels occurs not only in the systemic, but also in the pulmonary circulation at an appreciable period after its occurrence in the systemic vessels. The ventricles become distended, and the heart's action laboured. The heart becomes enfeebled by the circulation of non-oxygenated blood in its walls, the diastolic intervals become longer, until the organ finally stops in a state of diastole, with the right side full, while the left may have succeeded in emptying itself. Owing to the obstruction of the pulmonary circulation from this cause, the systemic blood-pressure falls, and the pulse becomes small and feeble.

**TREATMENT.**—Resuscitation from pure asphyxia is possible so long as the heart continues to beat. After cessation of the heart's action treatment is unavailing, except in cases of cessation from mere over-distension, in which bleeding from the external jugular vein may be resorted to with success. The chief indication in the treatment of asphyxia is to effect oxygenation of the blood by the introduction of air into the lungs. If the medium by which the patient is surrounded is incapable of supporting respiration, he must be immediately removed, or the atmosphere changed. If the air-passage is obstructed by a foreign body, this must be extracted; if this is impossible, or if the obstruction result from disease, tracheotomy or laryngotomy must be resorted to. Means must be adopted of exciting the respiratory centres or respiratory muscles to action; or if these are paralysed and non-excitabile, the natural movements of respiration must be imitated artificially, or air introduced by insufflation.

The respiratory centres, if not absolutely paralysed, may be excited reflexly by stimulation of cutaneous nerves, especially those of the face and thorax. This may be effected

by the stimulus of sudden cold, or better by alternate dashing of hot and cold water on the face and chest, or by flicking the skin with a towel. These reflex stimuli are often of themselves sufficient to excite respiratory movements; if not, they are powerful subsidiary aids to artificial respiration. The diaphragm may be stimulated to contraction by galvanisation of the phrenic nerve, one pole being placed on the nerve as it crosses the scalenus anticus at the root of the neck, the other on the epigastrium. Chief reliance, however, is to be placed on the methods of *artificial respiration*, which, after all, are the most simple and the most effectual, and possessed of the pre-eminent advantage of being always available. See ARTIFICIAL RESPIRATION.

Air may also be introduced into the lungs directly by properly constructed *insufflation-apparatus*, either by the insertion of a tube within the larynx (an operation requiring dexterity, but without danger if skilfully performed, though there is always risk of rupture of the air-vesicles from over-distension); or more easily by the insertion of the tube in one nostril, the other nostril and the mouth being closed (Richardson's apparatus). *Mouth-to-mouth-insufflation* is sometimes of advantage, especially in infants. The operator must close the nostrils of the patient, and, applying his mouth directly to that of the patient, inflate the lungs by his own expiratory efforts. The tendency to inflation of the stomach is considerably counteracted by backward pressure on the larynx.

D. FERRIER.

**ASPHYXIA, Local.**—A synonym for Raynaud's Disease. See RAYNAUD'S DISEASE.

**ASPIRATOR.**—**SYNON.**: Fr. *Aspirateur*. The operation of pneumatic aspiration was introduced into practice by Dieulafoy in 1869. Before this period various instruments had been used under the name of 'suction-trocars,' but to Dieulafoy is due the credit of fully appreciating their value.

**DESCRIPTION AND MODE OF EMPLOYMENT.** The aspirator consists of a glass syringe, having at its lower end two openings provided with stopcocks. When the piston is raised and the cocks are closed, a vacuum is created in the syringe, which can be maintained by fixing the piston in the withdrawn position. An india-rubber tube is fitted into each of the two openings, and these must be provided with coils of wire inside to prevent them from collapsing. At the end of one tube is fixed a fine hollow needle. The needle should have only one opening, at the point, and not, as is often seen, another at some distance from it. The instrument is thus used:—A vacuum having been created in the syringe by raising the piston whilst both stopcocks are closed, the needle is introduced into the part to be operated upon. As soon as the

opening of the needle is beneath the skin the stopcock leading to it must be opened. The vacuum will then extend to the point of the needle; and consequently, if it be gently pushed onwards, the moment it encounters fluid this will jet up into the glass syringe, when its nature may be ascertained. This mode of operating with what Dieulafoy calls the 'previous vacuum' is the essential difference between aspiration and suction. In aspiration it is impossible to pass the needle through a collection of fluid without discovering it; whereas without the 'previous vacuum' in the needle this might readily be done. If the fluid is sufficient in amount to fill the syringe, the stop-cock leading to the needle is to be closed and the other opened, by which the fluid may be discharged. The vacuum may then be re-established, and the operation repeated as often as is necessary. By opening both stopcocks at once and allowing the discharge-tube to hang down, the aspirator may be converted into a syphon. The action of the instrument may also be reversed, and it may be employed for injecting fluids. Other varieties of aspirator are in use besides the one above described. In Weiss's the receiver is a glass bottle, from which the air is exhausted by means of a separate exhausting syringe. The objection to this is that if the needle becomes choked, it cannot be cleared by pushing down the piston and driving some of the fluid back through it. Other kinds of aspirator cannot be converted into syphons, and these are objectionable. Weiss's has the advantage of being less liable to get out of order, as the fluid does not touch the exhausting syringe. The needles employed vary in size. Dieulafoy recommends that they should be about  $\frac{1}{50}$ ,  $\frac{1}{25}$ ,  $\frac{1}{20}$ , and  $\frac{1}{15}$  of an inch in diameter, and calls them Nos. 1, 2, 3, and 4 respectively. The aspirator may also be applied to trocars, but then its distinctive feature is gone, and it becomes but a 'suction trocar.'

The following rules must always be observed in using the aspirator:—1st. See that the needle is pervious and clean and the syringe in order before using it. It is advisable to wash the needle and syringe well in strong carbolic acid solution. 2nd. The needle must be pushed straight on in one direction only. Its course should not be altered while the point is under the skin. If no fluid is found, it may be withdrawn and reinserted. It must be held as steady as possible during the aspiration. 3rd. If the fluid will not flow with the pressure of the atmosphere, it is of no use squeezing and pressing the part. This can only do harm. 4th. Aspiration must cease at once when blood comes in any quantity, especially in abscesses. 5th. Keep up the vacuum during the withdrawal of the needle, lest some of the morbid fluid be left in its track. 6th. If the needle becomes choked, force a little of

the fluid back through it in order to clear it. 7th. After use the whole apparatus must be thoroughly cleaned with plain water to completely remove pus, blood, and other albuminous fluids, which would be coagulated by carbolic acid. After this is thoroughly done, the needle, tubes, and syringe should be disinfected with carbolic lotion (1 in 20). The leather of the piston may be greased with carbolised oil or vaseline.

USES.—Aspiration is used for purposes of diagnosis and treatment. Dieulafoy asserts that with the No. 1 needle it is possible to search for fluid without danger, whatever may be its seat or its nature; and experience has proved this to be practically true. In treatment it has been employed in the following affections:—

*Abscesses.*—In acute abscesses simple aspiration is usually of little value, as the pus soon re-accumulates. In chronic abscesses connected with diseased bone it usually fails; and often it is impossible, from the presence of cheesy matter in the pus. If performed with proper antiseptic precautions, however, it can do no harm, and may, if repeated, diminish the size of the sac before other means are adopted, or even effect a cure. A modification of simple aspiration has been recently introduced by Billroth, which not infrequently cures even chronic abscesses undoubtedly connected with diseased bone. Instead of the small needle, a large trocar, from  $\frac{1}{4}$  to  $\frac{1}{2}$  inch in diameter, is fitted to the aspirator. If necessary a small incision is made in the skin to facilitate the introduction of the trocar. The abscess having been emptied of its contents, the cavity is distended with a solution of perchloride of mercury (1 in 4,000) which is again withdrawn by the aspirator. This must be repeated again and again, till the fluid returns perfectly clear. Finally, an emulsion of iodoform and glycerine (1 to 10) is injected, the trocar is withdrawn, and the opening sealed with collodion. Most commonly the abscess fills again in a few days, after which the fluid may be gradually absorbed, but it is frequently necessary to repeat the operation more than once. There is no doubt that many chronic abscesses have been cured in this way, but the exact value of the treatment has yet to be determined. *Diseases of the Liver.*—Hydatid cysts have been successfully treated by aspiration. In many cases the fluid becomes purulent after one or two aspirations, and in some the cyst has opened externally. For purposes of diagnosis the small needles may always safely be introduced into the liver. Abscess of the liver has been successfully treated by the aspirator. *Retention of Urine* may be safely relieved by using No. 2 needle above the pubes. *Ovarian cysts* may be diagnosed and treated in the same way. *Hernia.*—It has been asserted that strangulated hernia may be, in the great majority of cases, re-

lieved by the use of the aspirator. The finest needles only should be used. They remove first the fluid from the sac, after which some gas may be obtained from the strangulated gut, but fecal matter rarely fails to choke the tube. Experience has, however, shown that this mode of treatment is both dangerous and inefficient. *Diseases of Joints*.—Aspiration is occasionally useful in acute synovitis. Great care must be taken not to scratch the inside of the joint with the needle, as this has been said to lead to acute arthritis and suppuration. No. 1 or 2 needle should be used. *Hydrocephalus* and *Spina bifida* may be aspirated with safety with No. 1 needle. In hydrocephalus it is to be passed through the anterior fontanelle. No case has hitherto been cured by this treatment. In *Pleurisy* and *Ascites*, except for purposes of diagnosis, in the writer's opinion the aspirator presents no advantages over a trocar properly constructed so as to exclude air (*see* PARACENTESIS). *Pericarditis*.—The operation of aspiration has frequently been successfully performed for pericardial effusion. It is thus carried out:—A spot is chosen 2 to 2½ in. (5 or 6 centimetres) beyond the left edge of the sternum, in the 4th or 5th interspace. No. 2 needle is then passed obliquely upwards and inwards, taking care to turn on the vacuum as soon as the eye is covered. The moment the fluid jets into the syringe the needle must be held steadily till the flow ceases. If this be done there is no danger of wounding the heart. If there is any doubt as to the existence of fluid, No. 1 needle must be employed, with which the heart may be punctured without great danger.

MARCUS BECK.

**ASTHENIA** } (*ἀ*, priv.; and *σθένος*,  
**ASTHENIC** } strength).—Terms signifying want of strength. As applied to the entire system, they indicate considerable general debility; in connexion with particular diseases, they imply that these are attended with marked weakness. *See* DEBILITY.

**ASTHENOPIA** (*ἀ*, priv.; *σθένος*, strength; and *ὄψ*, the eye).—Weakness of sight. *See* VISION, Disorders of.

**ASTHMA, SPASMODIC** (*ἄσθμα*; from *ἄω*, I blow).—SYNON.: Bronchial Asthma; Fr. *Asthme*; Ger. *Bronchial-Asthma*.

**DEFINITION**.—An affection characterised by severe paroxysmal dyspnoea, recurring at more or less well-marked periods, generally in the night, the dyspnoea being due to spasmodic contraction of the bronchi, produced by a variety of causes.

**ÆTIOLOGY**.—The causes that induce an attack of asthma are very various, and may be roughly classed according to their action, *direct* or *indirect*, on the respiratory organs. In the former class the exciting cause im-

mediately affects the mucous membrane; in the latter it does so in a more circuitous manner, as through the blood or the nervous system generally:—

Direct	}	Dust.	}		
		Vegetable irritants.			
		Chemical vapours.			
		Animal emanations.			
Indirect	}	Climatic influences.	}	Centric.	
		Bronchial inflammation.			Excito-motor.
		Through the nervous system			Gout.
		Through the blood			Syphilis.
		Heredity.		Skin-diseases.	
				Renal diseases.	

**Direct causes**.—Common roadside dust; fluff from woollen clothing; the dust of mills, threshing-floors, or bakehouses; and any mechanical particles when inspired, will produce in some persons an asthmatic seizure; dust of low specific gravity being more apt to have this effect than heavy particles like coal, steel filings, &c., from which arise lesions in the lung of a more permanent and serious character.

The odour evolved by certain vegetables, such as ipecacuanha; the pollen of many grasses and plants (*see* HAY-FEVER); certain chemical vapours, as that of pitch, sulphurous acid, and phosphorus fumes; the peculiar smell of some animals, as dogs, cats, horses, and hares, may each provoke a spasm in individual cases.

A still more powerful cause is climatic influence, the action of which on different patients cannot, unfortunately, be reduced within the limits of a law, but depends mainly on the idiosyncrasy of the individual. Extremes of temperature, or excessive dryness or dampness, may produce an asthmatic seizure, but in the largest number of cases one of two elements appears as the chief factor. One large class of sufferers trace the attack to *dampness*, whether of soil or of atmosphere, in combination with either heat or cold; another to *closeness* of atmosphere and a want of proper circulation of air, such as is found in deep valleys and thick forests, and during thundery weather—this last class experiencing great relief when a breeze springs up. Malaria plays an important part occasionally in the causation of asthma.

Far more general and intelligible in its action is bronchial inflammation, which is the cause in 80 per cent. of asthmatic cases. It frequently happens that after whooping-cough, measles, or infantile bronchitis the tendency towards asthma begins to appear. These diseases, implicating as they do both bronchial muscle and nerve supplying it, leave their mark behind, either in irritability of the mucous membrane; in induration of some portion of the lung, generally at the root; or in enlargement of the bronchial glands, causing pressure on the pneumo-

gastries, or on some of the branches of the pulmonary plexuses (*see* BRONCHIAL GLANDS, Diseases of); and thus lay the foundation of asthma in after-life. Asthma following on arrested phthisis is not rare in adults, probably from enlargement of the bronchial glands by absorption of tubercle and caseous material. In other instances the indirect cause may be found in morbid states of the nose or throat.

*Indirect causes.*—This class of causes includes those acting through the general nervous system; those acting through the blood; as well as the more or less structural one of heredity.

The *centric* subdivision embraces attacks arising from emotion, anger, or fright; as well as the curious alternations of asthma, neuralgia, angina, and gastralgia due to some centric irritation in the medulla, involving the origins of the fifth and eighth pairs of nerves, and manifesting itself by affecting first one branch and then another of these nerves.

*Excito-motor* causes may be illustrated by indigestion or costive bowels giving rise to a paroxysm of asthma. In the first case, irritation of the medulla is induced through the gastric branches of the pneumogastric, and a motor effect is reflected through the pulmonary branches. These *peptic* attacks, as they are called, occur more frequently after suppers than dinners, probably because reflex irritability is always exalted by sleep, as we know to be the case in epilepsy and the teething convulsions of childhood.

Gout, towards old age, often takes the form of asthmatic seizures, which alternate with the articular affections. In like manner attacks of the disease have been attributed to syphilis, but here the poison generally acts by enlarging the bronchial glands.

The connexion between asthma and various kinds of skin-disease is intimate: the subsidence of eczema, of urticaria, or of psoriasis, has often been accompanied by fits of spasmodic breathing, which have ceased on the reappearance of the eruptions. Here, again, the state of the blood is presumed to be the origin of both maladies, as in disease of the kidneys, which will be presently referred to.

*Heredity* can be traced in about 40 per cent. of asthmatics, though the tendency often does not show itself till late in life. The characteristic form of chest is frequently transmitted from parents to children; and even when this is not so, a disposition towards spasmodic symptoms in catarrhal attacks is often seen in the children of some asthmatics.

*SYMPTOMS.*—The patient may retire to bed with few or no premonitory symptoms, and sleep for some hours, but is disturbed in the late night or early morning—2 A.M. is a common time—by a feeling of oppression approaching to suffocation, referred either to the throat, sternum, or epigastrium, which obliges him

to sit up in order to breathe. Sometimes the onset is more gradual: the patient, having fallen asleep in spite of uneasy sensations, begins to wheeze during sleep, and is only aroused when the dyspnoea becomes severe. The breathing is accompanied by a humming sound, which gradually develops into a great variety of discordant noises.

In order to increase the capacity of his chest to the utmost, the asthmatic patient sits up and fixes his shoulders, either by placing his hands on either side of him, or by supporting his elbows on his knees; or sometimes he stands leaning over the back of a chair or other support. In one or other of these positions he remains immovable, with chest, back, shoulders, and head fixed; unable to speak or even to move his head; the lips being parted; the face pale, anxious, and, if the dyspnoea continue, livid; and the eyes prominent and watery. Every muscle of respiration, ordinary and extraordinary, is brought into requisition; those passing from the head to the shoulders, to the clavicles, and to the ribs become rigid, and, in place of moving the head and neck, act the reverse way, being used from fixed points to raise and dilate the thorax. The trapezii and levatores anguli scapulae by their contraction elevate the shoulders, in order that the muscles connecting these with the ribs may act as elevators to the latter. Even the muscles of the back are pressed into the service, and they almost cease to support the back; consequently the patient stoops. At each inspiration the sterno-cleido-mastoids stand out like cords, leaving a deep hollow between their sternal attachments; the diaphragm is contracted, and hence the stomach, liver, and heart are somewhat displaced. With all this display of muscular force, the chest remains almost motionless, being expanded to a variable extent.

In spite of the great dyspnoea, respirations are not proportionately frequent, seldom exceeding thirty, and sometimes falling to nine a minute. The expiration is prolonged, being generally two or three times as long as the inspiration. The pulse is usually slow and feeble; the temperature rarely exceeds 99° Fah., and is often below 98° Fah. Analysis of the expired air shows the oxygen to be almost entirely replaced by carbonic acid, which may increase from the normal to as much as 11 per cent., the nitrogen varying from 89 to 93 per cent. The arrest of expiration is probably the cause of the accumulated carbonic acid, but the total disappearance of the oxygen is hardly to be explained, for that oxidation of the tissues does not proceed is shown by the pale urine passed after the fit.

*PHYSICAL SIGNS.*—These reveal less than might be expected. The percussion-note is somewhat raised over the whole chest, most so in the posterior regions, where a drum-like

sound occasionally prevails; this hyper-resonance is probably due to accumulation of air induced by obstructed expiration, and in incipient cases passes off with the attack. The normal areas of cardiac and hepatic dullness often disappear during the attack, owing to the emphysema temporarily induced. When the attack has passed off, careful examination will sometimes detect dullness in the supra-scapular or inter-scapular regions, on one or both sides of the chest, indicating probable enlargement of the bronchial glands.

Auscultation shows an entire abolition of the normal breath-sounds, and the existence of dry sibilant or sonorous rhonchi, everywhere varying in tone according to the calibre of the bronchial tubes; the smaller tubes giving the high notes, and the larger ones the deep notes. These sounds continually change their position, springing up under the listening ear, and as quickly vanishing again, to give place to profound silence.

**PROGRESS AND DURATION.**—A paroxysm of asthma, when once established, lasts from half an hour to several days, and generally terminates with expectoration—thin and transparent if the seizure be short; but abundant and more or less opaque if the fit be prolonged, or if the case be chronic. The sputum has also been found to contain, in greatest abundance at the close of the attack, (1) the so-called Charcot-Leyden crystals, which are minute octahedra of unknown composition, but soluble in warm water; also (2) Curschmann's spirals, which consist of closely packed epithelial cells, arranged in a spiral form surrounded by mucoid material. These are not, however, pathognomonic of asthma. The urine is light-coloured and plentiful; the surface is bathed with perspiration; and flatus is often expelled from the bowels. Little or no food is taken during the attack, at the close of which the patient falls asleep.

The recurrence of the attacks when once they have been excited is generally periodic; but much depends on the presence or absence of the exciting cause. In many cases the patient is quite free from wheezing and dyspnoea in the intervals, and feels and acts like other people; but when the attacks follow each other closely, a more or less wheezy condition remains behind, and a few signs of obstructed breathing are generally to be detected in the interscapular regions.

**COMPLICATIONS AND SEQUELÆ.**—If the asthmatic attacks become habitual, their effects are seen on the patient's frame and on the organs implicated. The shoulders become raised, the head being buried between them; the muscles of the back, owing to their being called on to act as extraordinary muscles of respiration, are diverted from their use as erectors of the spine, which, accordingly, yields in the anterior direction, and the patient stoops. The frequent occurrence of

spasmodic contraction of the bronchi causes hypertrophy of their muscular coat; and this, with or without the congestion of the mucous membrane accompanying it, leads in time to thickening of the tubes and permanent narrowing of their calibre. The more common result of asthma is emphysema, arising from the difficulty of expiration. The emphysema, at first temporary, becomes in chronic cases permanent, and gives rise to displacement of the adjoining organs (*see EMPHYSEMA*). Contraction of the bronchi largely influences the pulmonary vessels, and considerable obstruction of the pulmonary circulation is the result. The vessels become gorged, and the lungs sometimes œdematous. If the emphysema be extensive, we may in time expect dilatation of the right side of the heart and marked prominence of the veins of the breast and neck, and the effects may be carried so far as to cause œdema of the lower extremities with albuminous urine, as the writer witnessed in a case where these symptoms disappeared on the subsidence of the asthma.

**PATHOLOGY.**—Patients rarely, if ever, die of spasmodic asthma, though death may ensue from some of its complications and sequelæ; and the disease, being a functional one, cannot be said to have any morbid anatomy. The onset and departure of the attack, and the ever-changing physical signs, led Laennec to think that, whatever obstruction in the bronchial tubes caused the phenomena, must be of a spasmodic and transitory nature. He therefore concluded that asthma was due to a spasm of the bronchial muscles which had been described by Reisseissen. Laennec also showed that an asthmatic sufferer could sometimes, after holding his breath, actually breathe naturally for one or two respirations, thus clearly demonstrating that the spasm was capable of momentary relaxation. Other theories were put forward, and doubt was thrown on the existence of muscular fibres in the bronchi, until Dr. C. J. B. Williams proved their existence by his experiments on the lungs of oxen, dogs, rabbits, and other animals, when he caused contraction of the trachea and bronchial tubes by the application of electrical, chemical, and mechanical stimuli. The muscular coat was shown to be more abundant in the smaller tubes than in the larger, the former contracting sufficiently to entirely obliterate their passages. In asthma, excitation of the muscles probably takes place through the anterior and posterior pulmonary plexuses, which are made up of branches from the pneumogastriæ, recurrent laryngeals, the spinal nerves, and the ganglia of the sympathetic, thus giving the bronchial tubes a very wide area of connexions. The branches of the pulmonary plexus form a network round the bronchial tubes, and contain some minute ganglia. When the cause is direct, as dust of any

kind or climatic influence, the spasm may be induced by reflex action through these small ganglia, or through the pulmonary plexus, though it soon extends deeper into the nervous system, involving the pneumogastrics, and causing a motor effect on the thoracic muscles through the upper cervical, phrenic, and dorsal nerves. Where emotion, fright, or laughter starts the fit, the irritation is centric, and causes a motor effect on the pulmonary plexus through the pneumogastrics. Where, again, indigestion excites it, the sensation passes through the gastric branches of the pneumogastric, and is reflected by the motor filaments of the pulmonary plexus. Lastly, where gout, syphilis, albuminuria, skin-disease, and heredity are the excitants, we may regard the blood itself as causing the local irritation. Spasmodic asthma may, therefore, be considered as a neurosis of the pulmonary branches of the plexus of that name, similar to other neuroses, as hemicrania and sciatica, and giving rise, through the motor nerves of the plexus, to spasm of the bronchial muscle.

**DIAGNOSIS.**—Asthma is distinguished from *bronchitis* by the fugitive physical signs; by the spasmodic character of the dyspnoea; and by the scant expectoration. The breathing in bronchitis, when at all difficult, is hurried; in asthma it is slow, wheezy, and prolonged; this feature also contrasting strongly with the gasping, panting dyspnoea generally accompanying pneumonia, pleurisy, and some forms of heart-disease. From *croup* it is recognised partially, but not entirely, by the age of the patient; and partly by the character of the dyspnoea, which in croup is inspiratory, whereas in asthma it is mainly expiratory. This characteristic also distinguishes asthma from spasm of the glottis and the various forms of laryngeal dyspnoea. The diagnosis from *emphysema*, which is so often mixed up with spasmodic asthma, is founded chiefly on the paroxysmal character and violence of the asthmatic dyspnoea, and on the complete freedom of the intervals, the dyspnoea of emphysema being more or less permanent.

*Aneurysm of the aorta* and other *mediastinal tumours* often give rise to symptoms so exactly simulating spasmodic asthma as to make the diagnosis difficult, and this is to be accounted for by these tumours pressing on the pneumogastric and its branches, and thus inducing an asthmatic spasm. These cases are, as a rule, however, accompanied by a certain amount of stridor arising from laryngeal spasm, not present in asthma, and this symptom is often of great diagnostic value in obscure cases. As the tumour enlarges, it causes greater pressure on the lungs, trachea, œsophagus, sympathetic ganglia, or other structures, and produces shrill cough, dysphagia, difficulty of inspiration, pain in the chest, impulse in the thoracic

wall, and other noted aneurysmal symptoms. Moreover, certain physical signs become evident, *e.g.* dulness over the first portion of the sternum or to the right of it, or between the scapulæ; tubular sounds and bronchophony close to the sternum, or above one or both scapulæ; or some form of bruit or murmur in the course of the aorta. These and other symptoms and signs contrast sufficiently with those of spasmodic asthma, to make the diagnosis from developed aneurysm comparatively easy.

In some cases of *renal disease* in which albumin appears in the urine, a form of dyspnoea appears, which is occasionally marked by paroxysmal features, and has been mistaken for spasmodic asthma. Renal dyspnoea differs, however, as a rule, in being more continuous; and in having for its origin œdema of the lung rather than bronchial spasm. See RESPIRATION, Disorders of.

**PROGNOSIS.**—The question of recovery in cases of asthma depends, to a certain extent: first, on the possibility of the removal of the exciting cause; secondly, on the age of the patient; thirdly, on whether the attacks increase or not in frequency; fourthly, on the condition of the lungs and the breathing in the intervals.

If the patient be young (say, under fifteen), the chest well-formed, the attacks tending to diminish in frequency and intensity, and the lungs free in the interval, a most hopeful prognosis can be given. If, on the other hand, the patient be middle-aged, the attacks increasing in number and severity, and the breathing more or less short in the interval, we may conclude that there exists a considerable amount of permanent emphysema, which renders the prognosis of an unfavourable character. In every instance the detection and removal of the exciting cause or causes, as the case may be, exercise a chief influence over the prognosis.

**TREATMENT.**—The principal difficulty in the treatment of asthma lies in clearly ascertaining the nature and origin of the exciting cause. When this is discovered two great principles should guide us, namely: first, to avoid or remove the exciting cause; secondly, to allay and prevent the spasm.

Many of the cases arising from direct causes, as from dust and chemical vapours, are cured by simple avoidance of the exciting cause. Where bronchial inflammation induces the spasm, the inflammation must be subdued by salines and expectorants, combined with some anti-spasmodic, as belladonna, henbane, or stramonium. In more chronic instances, where some thickening of the walls of the larger bronchi and enlargement of the bronchial glands exist, iodide of potassium in doses of grs. v. to xv., or iodide of sodium in doses of gr. v., or a combination of both iodides, has been found very beneficial when persisted in for long

periods. Symptoms of iodism may be generally averted by temporarily diminishing the dose and diluting more largely with water. Inunction of preparations of iodine, such as the *linimentum potassii iodidi c. sapone*, or the *unguentum potassii iodidi*, or painting the skin with iodine-tincture, have not proved so successful in the writer's hands. The waters of Woodhall and Purton in England, of Kreuznach in Germany, and of Saxon in Switzerland, contain iodides largely diluted, and may be beneficially administered in asthma. Affections of the nose or throat will require attention. Where the attacks depend on gout, syphilis, or renal disease, or are connected with skin-disease, treatment must be directed to the condition of the blood. Arsenic proves of signal service in asthma co-existing with eczema, psoriasis, or other skin-affections.

Where heredity is the predisposing cause, the origin of the disease lies generally in defective development of the frame or of the lung-structure of the patient. For such persons gymnastic exercises, carefully conducted swinging on the trapeze, and other means of expanding the upper part of the chest and correcting the asthmatic stoop are to be employed, combined with tepid or cold sponging and as much outdoor life as possible, with walking and riding without fatigue. The tendency to catarrh is thus lessened, and the frame of the patient developed and fortified. In a large number of cases—as, for instance, those arising from climatic influences—we have to treat a simple neurosis, and to allay the spasm either by climatic or by medicinal means, of which the former is often the more important, and, owing to the leading part played by the idiosyncrasy of the patient, generally the most difficult. In obstinate cases the doctrine of contrasts appears the only safe one. Where the disease has been contracted in a moist climate, a dry one must be tried; if in an inland district, the seaside must be resorted to; but for by far the majority of asthmatics the atmosphere of large towns is suitable, and the smokier the air and the closer the streets the more good do the sufferers appear to receive. London, Glasgow, Birmingham, and Bristol are all favourable resorts for cases of neurotic asthma; and the points of difference between their atmospheres and that of the country consist, (1) in dryness; (2) in deficiency of oxygen; and (3) in excess of carbonic acid and carbon: all of which peculiarities appear to exercise a sedative effect on the neurosis. Damp, whether of soil or atmosphere, is usually found to be hurtful, and is one of the chief exciting causes of asthma. Nothing exemplifies this more completely than the excellent results in cases of asthma produced by climates of which the atmosphere is dry.

The medicines most useful in asthma are

antispasmodics, either stimulant or sedative. The former, including alcohol, strong coffee, nitro-glycerine, ether, and nitrite of amyl, will prove more efficacious where emphysema is present; the sedative comprise stramonium, belladonna, Indian hemp, lobelia, datura tatula, tobacco, opium with its alkaloids, and drugs of a similar action. These may be taken internally in the form of extracts or tinctures; or smoked in pipes or as cigarettes; or inhaled as vapour diffused through the room by burning powders or pastilles containing them. A popular and often reliable remedy is the vapour arising from the combustion of nitre-paper, and other useful forms of powder may be devised; but these should be recommended with caution, lest an unfortunate habit of indulgence in fuming preparations be acquired. In the severest attacks the patient can neither smoke nor swallow, and in this difficulty of introducing medicines into his system we find the hypodermic injection of atropine (gr.  $\frac{1}{60}$ ), morphine (gr.  $\frac{1}{4}$ – $\frac{3}{4}$ ), and chloral, or suppositories of morphine and belladonna *per rectum*, prove effectual; but if albuminuria or emphysema be present, or if the pulse be weak, they must not be used. Chloroform often acts like a charm in the worst case, and after inhaling 20 to 60 minims the patient will gain the sleep which has been denied to him for hours, or even days; but as the effect is generally transitory, and the use of the remedy not free from risk, it should be given, if at all, in capsules containing a measured dose. Iodide of ethyl ( $\text{M v-x}$ ) can be inhaled with advantage. Ether is safer, but not so effective. Chloral hydrate in doses of 15 to 20 grs., repeated every four hours until the spasm subsides, has produced not only temporary, but even permanent good in a large number of asthmatic cases, and, if watched, may be persisted in for some time. In the writer's hands it has proved a most successful remedy. In the use of antispasmodics we must avoid judging the effects of one from the failure of another of the same class; but in difficult cases we must try each in succession—for it often happens that the successful remedy is only arrived at after repeated trials.

The mineral waters of Mont Dore and La Bourboule contain arsenic, and appear to act favourably in the more purely neurotic cases of asthma, while those of the Pyrenean sulphur springs of Eaux Bonnes, Eaux Chaudes, and Caunterets, are reputed to exercise a beneficial influence over the malady, but they have not been successful in the writer's experience, and it is more probable that they relieve by reducing the catarrhal symptoms than that they either allay the spasm or prevent its recurrence.

Compressed air baths at pressures varying from half to two-thirds of an atmosphere, and lasting two hours, are of the greatest benefit to asthmatics, and appear to reduce the sen-

sitiveness of the pulmonary plexus and to diminish cough and spasm, but they must be taken in numbers varying from 24 to 50 to be of permanent value. *See* AIR, Therapeutics of.

The *dietetic* treatment varies in individual cases, but as a rule asthmatics should dine early, and for the rest of the day limit themselves to liquid food, such as beef-tea, soups, and milk, combined with such an amount of stimulant, in the form of a pure spirit, as may be necessary, thus avoiding any distension of the stomach and intestines before retiring to rest. The diet should consist of brown bread, dry toast, and biscuits—excess of starch in any form being studiously avoided, a fair supply of plainly cooked meat, fish or poultry, and a limited amount of vegetables and fruit, care being taken to select only the most digestible of each class. Stimulants may sometimes be taken, but with discernment.

C. THEODORE WILLIAMS.

**ASTIGMATISM** (*ἀ*, priv.; and *στίγμα*, a spot or point).—*SYNON.*: Astigmism.—Want of symmetry in the anterior refracting surfaces of the eyeball, in consequence of which rays of light proceeding from a point are not brought to a focus upon the retina as a point, but only as a diffused spot. *See* VISION, Disorders of.

**ASTRINGENTS**.—*DEFINITION*.—Medicines which cause contraction of tissues.

*ENUMERATION*.—The chief astringents are: Nitrate of Silver; Sulphate of Copper; Sulphate of Zinc; Acetate of Lead; Perchloride of Iron; Alum; Tannic and Gallic Acids, and vegetable substances containing them, such as Oak Bark, Galls, Kino, and Catechu; and Dilute Mineral Acids. Some authors also include in this class of remedial agents such articles as Ergot of Rye, which contracts the blood-vessels and lessens hæmorrhage after it has been absorbed into the blood, although it has no local astringent action.

*ACTION*.—With the exception of gallic acid, the substances already mentioned coagulate or precipitate albumin. Dilute mineral acids do not coagulate albumin, but precipitate many albuminous bodies from the alkaline fluids by which they are held in solution. When applied to a surface from which the epidermis has been removed, the other astringents combine with the albuminous juices which moisten this surface, as well as with the tissues themselves, and form a pellicle more or less thick and dense, which in some measure protects the structures beneath it from external irritation, at the same time that they cause the structures themselves to become smaller and more dense. On a mucous membrane they have a similar action, and they lessen its secretion. It was formerly supposed that their action was partly due to their causing the blood-vessels going to a part of the body to contract, thus lessen-

ing the supply of fluid to it; as well as to their effect on the tissues themselves. But experiment has shown that, while nitrate of silver and acetate of lead possess this power, perchloride of iron and alum do not, and that tannic and gallic acids actually dilate the vessels. The astringent action of these latter drugs must therefore be exerted upon the tissues.

*USES*.—Astringents may be employed *locally* in various forms. In the solid form, as a powder, or in various preparations such as lotions, ointments, plasters, glycerines, &c., they are applied, especially the metallic astringents, to wounds and ulcers for the purpose of reducing the size and increasing the firmness of exuberant granulations, as well as of protecting the surface by forming a pellicle over it. They are used to lessen congestion and diminish the secretion of the various mucous membranes: as a lotion to the eye and mouth; as a gargle or a spray to the throat; in the form of an injection to the nose, urethra, and vagina; and as a suppository to the rectum. Administered internally, several astringents have a powerful effect in checking diarrhœa, and certain of them may have a local action upon the stomach and intestines.

The *remote* action of such astringents as acetate of lead and gallic acid, when absorbed into the blood, in lessening hæmorrhage, is made available in the treatment of hæmoptysis, hæmatemesis, hæmaturia, and loss of blood from other parts of the body.

T. LAUDER BRUNTON.

**ASTURIAN ROSE**.—The rose or erythema of the Asturias; one of the numerous synonyms of pellagra—*Mal de la rosa*; *Lepra asturiensis*; *Elephantiasis asturiensis*. *See* PELLAGRA.

**ASYSTOLE** (*ἀ*, neg.; and *συστολή*, a contraction).—*SYNON.*: Fr. *Asystolic*; Ger. *Mangelnde Zusammenziehung*.—The most advanced stage of progressive failure of the heart, in which the ventricular walls have become so feeble that the systole is incomplete and highly irregular, and the cavities are dilated from over-distension with undischarged blood. *See* HEART, Dilatation of.

**ATAVISM** (*atavus*, a grandfather).—The inheritance of a disease or constitutional peculiarity from a generation antecedent to that immediately preceding. *See* DISEASE, Causes of.

**ATAXIA** } (*ἀ*, priv.; and *τάξις*, order).—  
**ATAXIC** }  
Terms which originally meant any irregularity or disorder, but are now specially applied to irregularity of associated or co-ordinated muscular movements. The noun is frequently used as synonymous with the disease known as locomotor ataxy. *See*

Co-ordination, Disorders of; and LOCOMOTOR ATAXY.

**A TELECTASIS** (*ατελής*, imperfect; and *έκτασις*, expansion).—Absence or imperfection of the expansion of the pulmonary alveoli which normally takes place at birth, the lungs thus remaining more or less in their fetal condition. See LUNGS, Collapse of.

**ATHEROMA**.—See ARTERIES, Diseases of; and AORTA, Diseases of.

**ATHETOSIS** (*ἀθετος*, without fixed position).—DEFINITION.—A name given by Dr. Hammond, of New York, to a condition in which the hand and foot are in continual slow, irregular movement, and cannot be retained in any position in which they may be placed.

DESCRIPTION.—The special character of the movements in athetosis is that they are slow and deliberate. They usually affect the arm and leg on one side only. Voluntary power is retained, but is interfered with by the slow spasm. The fingers are irregularly flexed and extended: at one moment they spread wide apart, the thumb being over-stretched; thereafter, first one, then another, is bent into the palm, and again extended. The movement can be arrested for a moment in certain positions by the will, but is renewed with increased force. The foot is usually inverted; the toes being flexed or extended, but in less constant movement. The spasm may cause pain. The muscles sometimes become hypertrophied. The movements in some cases cease during sleep, in others they do not. Sensation is often, but not always impaired.

The onset of this condition has been sudden in a few cases, but usually the movements develop gradually after a unilateral seizure, whether a slight, sudden hemiplegia, or a convulsion. The subjects have been generally in middle life.

Athetosis differs from the spastic contracture so common after hemiplegia in children, only in the slowness of the movements, and in the fact that they are spontaneous and not limited to voluntary movement. But these distinctions are not absolute. The disorder of movement after hemiplegia may be identical in character with athetosis, and both are essentially the same in relations. In what is termed athetosis the cerebral lesion is so placed as to cause no distinct hemiplegia in most cases; but its occurrence is indicated by the sudden symptoms which precede the slow movement. It cannot be regarded as a distinct disease or even a distinct symptom. Typical athetosis may succeed hemiplegia.

**PATHOLOGY**.—It is probable that, as Dr. Hammond suggests, the seat of the lesion in athetosis is generally the optic thalamus or its neighbourhood. The sudden onset of the

disease and the slight affection of sensation are in favour of this explanation of cases in which there is no distinct hemiplegia. Charcot believes that all post-hemiplegic chorea-like movements depend on the implication of fibres outside the optic thalamus. In a case of simple ataxy after hemiplegia—an analogous condition—the writer has found a cicatricial sclerosis extending across the optic thalamus, and probably left by a patch of softening. The symptoms, perhaps, may be produced in various ways, since the regulation of the motor processes in the cortex is complex. One cause of them may be a state of perverted action of the motor cells of the cortex, due to a deficiency of the upward influence from the cerebellum. Another may even be partial damage to the motor centres themselves.

**PROGNOSIS**.—This is unfavourable; but the slighter cases improve and may even approximately recover.

**TREATMENT**.—Nervine tonics and sedatives are the remedies chiefly indicated. Of the former arsenic, and of the latter Indian hemp and bromides, may be given. The continuous current has been known to lessen the spasm, but, as a rule, it fails. The positive pole may be placed on the spine or brachial plexus, the negative on the muscles involved. The action of the continuous current is probably in part direct, in part reflex, lessening by the peripheral impression the over-action of the centre.

W. R. GOWERS.

**ATONY } (*ἀ*, priv.; and *τόνος*, tone).—**  
**ATONIC }**

Terms implying want of tone, power, or vigour, associated either with such a condition of the system generally, or of particular organs, especially those which are contractile. See TONE, Want of.

**ATRESIA** (*ἀ*, priv.; and *τρέρημι*, I pierce).—Absence of a natural opening or passage, whether congenital or caused by disease.

**ATROPHY, GENERAL**.—SYNON.: Marasmus.

DEFINITION.—Atrophy means, etymologically, simply want of nourishment (*ἀ*, priv.; and *τροφή*, nourishment), but the term is commonly applied to the condition resulting from want of nourishment, namely, wasting or diminution in bulk and substance, even though this may have been produced by some other cause, and even though the supply of nutritive material may have been abundant. *General atrophy* is used to denote wasting in which the whole body participates. All acute diseases, if severe, are accompanied by emaciation, for at such times nutrition is temporarily interfered with. The use of the word 'atrophy' is, however, confined, as a rule, to cases where the interference with nutrition has been gradual, and the loss of flesh consequently slow.

**ETIOLOGY.**—Atrophy is common enough at all periods of life. In infants and children it is due, in the majority of cases, to chronic functional derangements which interfere with the digestion and elaboration of food. Less frequently it is a consequence of organic disease. In adults general atrophy seldom results from any other cause than organic disease, and functional disorder as a cause of serious wasting is the exception. In old age atrophy is a common consequence of the degenerations of tissue which accompany the decline of life. The interference with nutrition may, however, be aggravated by the presence of disease.

In *infants under twelve months old* there are four principal causes to which persistent wasting can usually be referred, namely, unsuitable food; chronic vomiting (gastric catarrh); chronic diarrhoea (intestinal catarrh); and constitutional diseases, such as inherited syphilis or tuberculosis. Bad feeding, by setting up a chronic catarrhal condition of the stomach and bowels, is a frequent cause of both vomiting and diarrhoea, but it may produce atrophy without either of these symptoms. When an infant is fed, for instance, with large quantities of farinaceous matter—a form of food which is alike indigestible and innutritious—a very small part only can enter as nutriment into the system. The remainder passes down the alimentary canal, and is ejected at rare intervals in an offensive putty-like mass or in hard roundish lumps. The child, therefore, although overloaded with food, is really under-nourished, and loses flesh as long as such a diet is persisted in. If, as often happens, diarrhoea or vomiting be set up by the irritation to which the digestive organs are subjected, wasting is more rapid and the danger of the case is increased. Not only excess of farinaceous matter, but any form of bad feeding will produce this result. Wasting, indeed, will be found in every case where the food selected is unfitted for the child, and thus it is not infrequently seen in infants who are fed upon milk and water alone. The casein of cow's milk is difficult of digestion by many infants on account of its tendency to coagulate into a large firm clot like a lump of cheese. In this respect it differs from the curd of human milk, which forms light small flocculent coagula, and is digested without difficulty. Special preparation is therefore generally required to render cow's milk a suitable diet for a young child.

It is not only, however, unsuitable food which is a cause of atrophy in infants. Catarrh of the stomach and bowels may be present, although the feeding is in all respects satisfactory. Infants are excessively sensitive to chills, and catarrh of their delicate digestive organs is easily excited. Now, catarrh of a mucous membrane is always accompanied by an increased flow of mucus,

and this alkaline secretion in excess acts as a ferment, and sets up decomposition of food. A subacute gastric catarrh from this cause is not rarely seen in new-born infants, who thus are rendered for the time incapable of digesting even their mother's milk. In such cases the fault is usually attributed to the milk, which is said to be unsuited to the child; and the mother is compelled, much against her will, to wean her baby and feed it in a different way. So long as the catarrh continues, however, no food appears to agree, and the child often after a time dies exhausted.

Between the ages of *one and three years* atrophy is commonly associated with rickets. In these cases the wasting is noted chiefly about the chest and limbs, for the belly is large and swollen from flatulent accumulation. At this age children are still liable to waste from catarrh of the stomach and bowels; indeed, rickets is itself often complicated by such derangements. Sarcoma of the internal organs is also sometimes found at this time of life, and is attended with extreme emaciation.

*After the age of three years* caseous enlargements of the mesenteric glands, if extensive, may become a cause of wasting.

*After the fifth or sixth year* chronic pulmonary phthisis begins to appear. Cases of heart-disease as a result of acute rheumatism are also more frequently seen. Diabetes, too, is sometimes met with. All these diseases may produce much interference with nutrition.

From the time that the child begins to take other food than that furnished by his mother's breast, he is liable to worms in the alimentary canal. The presence of worms is frequently accompanied by loss of flesh, not, perhaps, so much on account of the parasites themselves, as on account of the derangement of the digestive organs which is associated with them. Emaciation due to this cause may sometimes be extreme.

*In the adult* atrophy is rarely the result of mere functional derangement, but is almost invariably a sign of serious organic disease. All chronic ailments are not, however, accompanied by marked wasting. Purely local diseases lead to little loss of flesh unless they affect some part of the digestive apparatus, or of the glandular system which is concerned in the elaboration of nutritive material; or otherwise directly influence the processes of nutrition. Thus, emaciation quickly results from gastric ulcer or chronic dysentery, but chronic pneumonic phthisis may produce little diminution in weight if there is no pyrexia, and if the case is not complicated by diarrhoea or profuse expectoration. The most marked atrophy is produced by the constitutional diseases, such as cancer, tubercle, and syphilis in the third stage; by those which set up a persistent drain upon the

system, such as severe albuminuria, chronic hæmorrhages, and long-continued suppurations; or by those which directly impede the passage of nutritive material into the blood; and in the latter class of diseases, influences which act directly upon the thoracic duct, such as obstruction to its passage from pressure by aneurysm and other tumours, must not be overlooked. There is a form of atrophy sometimes seen in hysterical females, depending upon disordered innervation, in which the most extreme emaciation may be reached. Such cases are marked by a dislike to food which may amount to absolute loathing. See NEURASTHENIA.

**ANATOMICAL CHARACTERS.**—The most marked post-mortem appearance in this condition is diminution or loss of fat, especially of the subcutaneous adipose tissue; and this is accompanied by wasting of the tissues and organs generally. The histological elements are reduced in size without undergoing, as a rule, actual numerical diminution. With the atrophy is often associated a certain amount of fatty degeneration.

**SYMPTOMS.**—The symptoms of general atrophy are loss of flesh, loss of colour, and loss of strength, combined with other special phenomena arising from the particular disorder to which the impairment of nutrition is due.

**TREATMENT.**—The treatment of general atrophy consists in removing, if possible, the impediment to efficient nutrition. In the case of a child the diet must be selected with care. Excess of farinaceous food is to be avoided, and cow's milk can be diluted, if necessary, by admixture with barley water. Warm woollen clothing should be insisted upon, and special attention should be paid to the warmth of the feet. Any gastric or intestinal derangement must be at once remedied, plenty of fresh air should be obtained, and perfect cleanliness strictly enjoined. In an adult the disease which is the cause of the malnutrition must be sought for and submitted to treatment. Efforts should be made, on the one hand, to arrest any drain upon the system; and, on the other hand, by a judicious arrangement of the dietary, and by attention to the eliminatory organs, to remove all obstacles to nourishment. Even in cases of organic and incurable disease much benefit may often be derived from due observance of physiological laws. EUSTACE SMITH.

**ATROPHY, LOCAL.**—This condition signifies atrophy of a *part* of the body, which may be apparently congenital, or may be produced by various causes acting during life. It will be convenient to consider local atrophy according to the several forms which are met with.

**Congenital Atrophy.**—Congenital atrophy is that condition in which some part of the body never reaches its full standard

of size. It is more correctly denominated *arrested growth* or *congenital smallness*. When the whole of one side of the body is thus affected, a marked and permanent disproportion between the two sides results. This *hemiatrophy* is, in theory, difficult to distinguish from hypertrophy of the opposite side, but mostly the paralytic or enfeebled state of the atrophic side shows it to be abnormal. The limbs are most strikingly implicated; while the corresponding side of the face and head is sometimes similarly, sometimes conversely, affected. In some cases atrophy of the opposite half of the cerebrum was found on post-mortem examination. The same condition may be partial—*hemiatrophia partialis*, and it then chiefly affects the face, or some part of the territory of the fifth cranial nerve. These conditions must be ascribed to some perversion of innervation occurring during development. Other congenital atrophies, *local* but not *hemi-atrophic*, are more probably referred to obstruction of blood-vessels during the same process. The defective development of the brain in cretinism has been attributed to the pressure of an enlarged thyroid upon the carotid arteries.

**Physiological Atrophies.**—These form a distinct class, where atrophy of a part of the body takes place in the ordinary course of development. Such are the wasting of the thymus gland in early life, of the mammae and sexual organs after a certain age. Most commonly the atrophy is here closely connected either with the involution or perhaps the development of some correlated organ; but it is not possible to say what the nature of this connexion is, whether one of nutrition or innervation.

**Acquired Atrophies.**—The conditions thus distinguished possess most interest for the practical physician. Wasting of any part of the body during life, when not physiological, usually depends either upon some interference with the blood-supply, or some disturbance of innervation; but to these must be added, in the case of organs which have an active and continuous function, disuse or over-stimulation. Deficient blood-supply, which causes atrophy, may be produced by the obstruction of a nutrient artery, especially if it be gradual, since sudden blocking will produce more complicated phenomena. Constant pressure is a cause of atrophy, because it interferes both with the blood-supply, and with the vital actions of the tissue-elements. Intermittent pressure, on the other hand, by causing hyperæmia, is more likely to lead to hypertrophy. Moreover, inadequate renewal of blood—that is, filling of the vessels, even to excess, with venous blood—or venous engorgement, though at first it may cause enlargement, mostly leads to atrophy in the end; as is seen in the granular induration of liver

and kidneys caused by disease of the heart obstructing the circulation. Many forms of atrophy in old age are clearly dependent upon senile obstruction of the arteries, for example, that of the skin, spleen, and kidneys. The instances of atrophy from disturbed innervation are less easy to discriminate, except where there is actual paralysis. In two distinct diseases, however,—progressive muscular atrophy and infantile or essential paralysis, loss of power in the muscles is followed by a remarkable wasting, far more rapid than that which results from disuse alone. Division of the nerve of a limb produces rapid wasting of the muscles no longer used, and this is accompanied in the end by some diminution in the size of the bones and accessory parts. Local atrophy of the skin is sometimes seen in regions limited by the distribution of a nerve, especially some branch of the fifth; and more extensive atrophy of one side of the face or head, equally marked out by nervous distribution, and resembling some cases of congenital atrophy, has also, though rarely, been observed. In such cases the writer has found anesthesia of parts corresponding to branches of the fifth nerve, but absence of muscular paralysis, such as would be due to affection of the seventh nerve.

Disuse produces atrophy only in organs whose functions are active and constant, such as nerves and muscle. Nervous tissue wastes constantly, and sometimes rapidly, when currents cease to traverse it. This is seen not only in the nerves of paralysed limbs, but even in the nerve-centres, where any interruption of the nervous channels, either above in the cerebrum, or below in the nerve-trunks, is followed by degeneration, ending in atrophy, of the whole nervous tract leading from the cerebral cortex to the peripheral termination—so-called *secondary degeneration* of the cord. In muscular tissue the wasting is almost as constant, but hysterical paralyses make an exception, the helpless limbs preserving their nutrition in a surprising manner. In organs whose functions are intermittent or periodic, disuse does not appear necessarily to produce atrophy, as is seen in the testicles, ovaries, and mammae.

That excessive stimulation or over-work may produce atrophy is seen in degenerative diseases of the nerve-centres arising from undue mental activity; and of the sexual organs from excessive indulgence. Over-work of muscles very rarely produces atrophy; but the writer has seen instances where special muscles were exposed to strain while the general nutrition was low. Atrophy of muscles is said to occur in soldiers when badly fed, after long exhausting marches.

**Unexplained Atrophies.**—Cases of local atrophy occur of which it is impossible to give any satisfactory explanation. Such are the conditions known as linear atrophy

of the skin; some remarkable cases of atrophy of bone, especially of the skull (*fragilitas ossium*), and of some parts of the cerebrum. Atrophy of the thyroid gland appears to be certainly the cause of the disease called myxoedema, but the cause of the original atrophy is unknown.

It is possible that deficiency of special kinds of food may lead to atrophy of special organs—thus deficiency of lime may make the bones soft, and deficiency of iron arrest the development of blood-corpuscles; but even these familiar instances must be accepted with a little reserve. In the same way it is still doubtful whether any special drugs, such as iodine, can produce atrophy of special glands.

**PATHOLOGY.**—Wasting may occur simply, or as a consequence of change of substance, or from the intrusion of some new material; in other words, there may be *simple* atrophy, atrophy from *degeneration*, or atrophy by *substitution*. The first is probably rare; generally some change of substance occurs. The most frequent degenerative process is fatty degeneration; the albuminous substance being replaced by fatty matter, which, if afterwards absorbed, leaves a void. Organs thus affected may be apparently enlarged, though the original substance is wasted. Atrophy from substitution is seen when the connective tissue of an organ, for instance, increases, compressing and destroying the other tissue-elements; and these not being renewed when the newly-formed connective tissue contracts, the whole organ is diminished in bulk. This is seen in all the changes called cirrhosis or fibroid degeneration, as in cirrhosis of the liver and kidneys.

**TREATMENT.**—No general rules can be laid down for treating all cases of local atrophy. Where the blood-supply is deficient, we have rarely any means of supplementing it; where innervation is at fault, it is seldom under our control. In general, harm rather than good results from any attempt to attract blood by artificial irritation. In the case, however, of atrophy from disuse of the nervo-muscular system, a line of treatment, and more especially of prophylaxis, is very clearly indicated; this is, to keep the muscles in exercise by artificial means, particularly by electricity, or by the processes of friction and kneading known as passive motion (*see* MASSAGE). In this way so much of the atrophy as is due simply to disuse may be checked for the future, and even the former loss reinstated. We shall, moreover, never do harm by attempting to supply some special elements of food which appear to be deficient, as iron for the blood and phosphorus for the bones or nervous system.

J. F. PAYNE.

**AUDITORY NERVE, Diseases of.**  
*See* EAR, Diseases of; and HEARING, Disorders of.

**AURA** (*aŭpa*, a breeze).—A peculiar sensation, subjective in origin, immediately preceding an epileptic or hysterical convulsion, and named respectively *aura epileptica* and *aura hysterica*. The word was originally adopted because the sensation is often described as that of the passage of cold air or light vapour from the trunk or extremities to the head; but it has been extended so as to include any phenomenon, whether sensory or motor, that ushers in a fit of epilepsy or of hysteria.

**AURAL DISEASES.**—See EAR, Diseases of.

**AUSCULTATION** (*ausculto*, I listen). A method of physical examination, which consists in listening over various parts of the body, either by the direct application of the ear (*immediate auscultation*), or by the aid of special instruments (*mediate auscultation*), for the purpose of studying certain sounds produced in health and disease. See PHYSICAL EXAMINATION.

**AUSCULTATORY PERCUSSION.** A method of physical examination, in which the sounds elicited by percussion are studied by means of auscultation. See PHYSICAL EXAMINATION.

**AUSTRALASIA.**—The portion of Polynesia lying between 10° and 50° S. latitude, and 110° E. and 170° W. longitude, which may be said to include Australia, Tasmania, New Zealand, the Fiji Islands, the New Hebrides, and some less important islands.

**Australia.**—The climate of the vast continent of Australia, which is partly temperate and partly tropical, depends, first, on its latitude, and, secondly, on its conformation, the mountain ranges being distributed along the coast lines, especially on the eastern shores. In the interior, which is comparatively flat, and believed to be for the most part a sandy desert, there is great heat and little rain. The hot winds from the interior are often sufficient, in the summer, to raise the thermometer to 127° Fah., and on the amount of protection from these enjoyed by the various towns depends their climate. There are also sea winds from the N. and N.E. The southerly winds, prevailing chiefly from November to February, blow from the Antarctic Circle, and are cold winds of great velocity, ending in heavy thunderstorms. In the tropical region the rainfall is from November to April; and in the temperate, which lies to the east and south, it prevails only in the winter season.

The following are among the principal towns or centres to which invalids proceed:—

*Adelaide*, the capital of South Australia, lat. 35° S., long. 135½° E. It suffers from great heat and drought. The mean temperature is 65°, the maximum 115°, and the

minimum, 34°; the range being 81°, and the mean daily range 20°. The humidity is 60 per cent., and the rainfall 21 inches. The soil is sandy.

*Brisbane*, the chief town of Queensland, lat. 27½° S., long. 153° E. The climate is almost tropical. The mean temperature is 70°, the maximum is 108°, the minimum, 34°; the range 74°, with a mean daily range of 21°. The rainfall is 51 inches, and the mean humidity 76 per cent. Queensland is for the most part elevated; and the climate of the Darling Downs, on an average 2,000 feet above sea-level, is considered very fine. The townships at Toowoomba and Warwick in this region may be recommended.

*Melbourne*, the capital of Victoria, lat. 38° S., long. 145° E. It has the reputation of being a healthy and agreeable residence; the climate being dry and temperate, and far cooler in summer than that of Sydney. Mean temperature 57°, maximum 111°, minimum 27°, showing a range of 84°; daily range 18°. Mean humidity 72 per cent. Rainfall, 26 inches.

*Perth*, in Western Australia, very healthy, but as yet little suited to the requirements of invalids. The temperature is 63° (mean), and the rainfall 30 inches in 110 days.

*Sydney*, the capital of New South Wales, lat. 34° S. and long. 151° E. The climate of New South Wales is clear and dry, the temperature depending more on the altitude than on the latitude. The plains in the interior, swept by hot winds, are very dry, while the coast districts have abundant rain. Mean temperature 62·5°, maximum 107°, minimum 36°; range 71°; mean daily range 14°. Humidity 72 per cent., and rainfall 50 inches. Paramatta, on Port Jackson, is drier and cooler than Sydney (Lindsay); and the Illawara district, including the stations of Eden and Twofold Bay, is suitable for invalids; but the finest climate in Australia for pulmonary cases is to be found in the Riverina, a district lying between Queensland, the Blue Mountains, and the central desert, consisting of rolling prairie and undulating downs, with a rainfall ranging from 5 to 24 inches, the average being 14 inches, and a clear bright atmosphere, most exhilarating in its effect on invalids, who at the settlements of Denilliquin, Menindee, and the various sheep-farms, pursue an open-air life by day, and often by night, with great advantage.

**Tasmania.**—Tasmania lies 150 miles south of Australia, between lat. 40° 40' and 43° 38', and is mountainous, with a deeply indented coast-line. The climate is more temperate and equable than that of the south coast of Australia. In winter the cold is sufficient to produce thin ice in the low lands, and snow showers in the higher ranges. The mean temperature of *Hobart Town* on the S.E. coast is 54°, the summer mean being 62° and the winter 47°. The

rainfall varies greatly, from 100 inches at Macquarie Harbour on the W. coast to 24 inches at Hobart Town, distributed over 145 days. The prevalent winds are from the N.E. and S.W. The climate is favourable to infant life, and the country is regarded as a sanatorium for invalids.

**New Zealand.**—New Zealand lies between 34° 50' and 47° 50' S. lat., and consists of a North and a South besides smaller Islands. The North Island is for the most part volcanic, and abounds in hot springs, which are extensively used, and active craters, which impart an important influence to its climate. The South Island contains a lofty range of snowclad mountains, whose lower slopes form on the eastern shore a series of terraces known as the Canterbury Plains, and other fertile regions.

The climate is mild and bracing, but decidedly of a windy character, and not suited for all invalids: at Auckland in 1876 no calm day was recorded, the prevalent winds being W.S.W. The mean temperature of the North Island is 58°, of the South 54°. The maximum varies from 87°, at Christ Church, to 75°, at Hokitika, and the minimum from 25° to 34° in the South Island. Cold is as a rule unknown in the North Island, while in the South there are a few snowy days each year on the coast. The rainfall varies from 32 inches in 135 days

at Christ Church, to 131 inches in 186 days at Hokitika.

**Fiji Islands.**—The Fiji Islands, partly of volcanic and partly of coralline origin, have a tropical climate, moderated by the trade winds, so that the mean temperature does not exceed 80°, the minimum being given as 65°. The rainfall is chiefly from October to April—the hot season, and varies from 124 to 215 inches in 170 days. See CLIMATE Treatment of Disease by.

C. THEODORE WILLIAMS.

**AUTOPHONIA** (*αὐτός*, himself; and *φωνή*, the voice).—A physical sign obtained by studying the modifications of the resonance of the observer's own voice during auscultation. See PHYSICAL EXAMINATION.

**AUTOPSY.** See NECROPSY.

**AX**, in Ariège in France.—Thermal waters. See MINERAL WATERS.

**AZORES; St. Michael.**—Warm, very moist, equable climate. Mean winter temperature 58° F. Prevailing winds N. and E. See CLIMATE, Treatment of Disease by.

**AZOTURIA.**—A condition of the urine in which there exists an absolute and relative excess of urea, without accompanying pyrexia. See URINE, Morbid Conditions of.

## B

**BACILLI** (*bacillum*, a little rod or staff).  
SYNON.: Fr. *Bacilles*; Ger. *Bacillen*.

The term *bacillus* was applied by Cohn to the longer rod-shaped bacteria, *bacterium* to the shorter forms; filaments being distinguished as 'leptothrix,' 'vibrio,' &c. But the great variety in length of the individual elements of the same organism, and the variations under different conditions and periods of life, rendered such a distinction impossible. Hence the term *bacillus* is now used for all the species of schizomycetes which commonly have the form of straight rods, although in some conditions of growth they may form filaments. By some mycologists the term is used in a more restricted sense; but it seems well at present to use it in this somewhat wide manner, until our knowledge allows of more scientific classification. See MICRO-ORGANISMS.

**BACTERIA** (*βακτήριον*, a rod).—SYNON.: Fr. *Bacteries*; Ger. *Bacterien*.

Originally applied to common rod-shaped organisms of the class *Schizomycetæ*, and especially to one supposed variety called *Bacterium termo*, this name was later extended to include the whole of the class of

schizomycetes or fission-fungi. Thus the term *bacteriology* has been practically applied to the whole science of the lower orders of chlorophyllless alga. But of late, owing partly to the confusion involved in the double use of the term for the rod-shaped organisms and for the entire class; partly to the fact that fungi of other orders than schizomycetes are related to parasitic disease; and perhaps still more to the dominant influence of Pasteur and the French school, the term *microbes* has largely replaced the names 'bacteria' and 'micro-organisms'; and for the particular species formerly named *bacterium x, y, &c.*, the more accurate terms *bacillus, spirillum, &c.*, are used. See MICRO-ORGANISMS.

**BADEN-BADEN**, in Germany.—Thermal saline waters. See MINERAL WATERS.

**BADEN**, in Austria.—Thermal sulphur waters. See MINERAL WATERS.

**BADEN**, in Switzerland.—Thermal sulphur waters. See MINERAL WATERS.

**BADENWEILER**, in Germany. Simple thermal waters. See MINERAL WATERS.

**BAGNÈRES - DE - BIGORRE**, in France.—Simple thermal and earthy waters. See MINERAL WATERS.

**BAGNÈRES - DE - LUCHON**, in France.—Thermal sulphur waters. See MINERAL WATERS.

**BALANITIS — BALANOPOSTHITIS** (*Bálanos*, an acorn; and *πόσθη*, the foreskin).—**SYNON.**: Bastard Clap; *Blennorrhagia Balani*; Inflammatory Phimosi.

**DEFINITION.**—Inflammation of the opposing surfaces of the glans and prepuce; sometimes acute—even gangrenous, and sometimes chronic. A purely local affection; frequently, but not necessarily, of venereal origin.

**ÆTIOLOGY.**—Balanitis is much less common than urethritis, being met with at the Lock Hospital in the proportion of one to twenty-four of the latter. It may be either primary; or consecutive to chancres, syphilitic eruptions, warts, accumulated smegma, variolous pustules, or gonorrhœa. When primary, the common predisposing cause is a long, tight foreskin. Fourmier attributes two-thirds of the cases of balanitis to a long prepuce with insufficient cleansing; about one-third to irritation by chancres and gonorrhœal pus; and a very few to other causes.

**SYMPTOMS.**—The symptoms of balanitis depend on the intensity and extent of the inflammation. In the simplest form there is heat and itching of the furrow, slight redness of that part, with a milky or yellowish secretion. When the inflammation is more severe and extended, swelling and pain are added, the other symptoms are more marked, and characteristic excoriations appear. These excoriations are irregular, shallow, never extending deeply, but often coalescing into large raw chafings. An abundant yellowish-green matter of offensive odour bathes the surface. When the urine trickles over these excoriations there is severe smarting pain. If still further irritated, the foreskin swells enormously, is divided at the free border by deep creases, and can no longer be turned back. Aching, smarting, great tenderness, and painful erection, often accompanied by constitutional disturbance and fever, are present.

**COMPLICATIONS.**—The cellular tissue and the lymphatic ducts of the foreskin and sheath, or the lymphatic glands, may inflame to suppuration, to ulceration, and, in persons enfeebled by any cause, even to gangrene. Sloughing begins on the inner surface of the foreskin at the upper part; seldom to much extent, though the whole prepuce, except the frænum, may be lost, and when cicatrisation sets in the organ appears circumcised. Paraphimosis is caused by impudent retraction of a swollen foreskin. Warts keep up chronic posthitis of the furrow. Adhesions, usually

at the corona and the furrow, may attach the prepuce completely to the glans. Thickening and phimosis are not uncommon after repeated attacks.

**COURSE.**—The duration of balanitis depends on the anatomical condition of the parts. When remedies can be easily applied, it is not more than three or four days. With phimosis, the course is severe, and the duration is indefinite; even when limited to the furrow, posthitis is often obstinate.

**DIAGNOSIS.**—This is easy when the parts can be exposed. *Herpes* is distinguished from balanitis by small round ulcers, grouped on a red area, and limited to one or two points of the mucous surface, without general congestion. *Simple chancre* has well-defined undermined edges and a spongy surface. The *syphilitic sore* has the indurated base and enlarged lymphatic glands. When there is phimosis, the discharge may come from the urethra or from a chancre. If from the urethra, it can usually be seen escaping thence, and there is pain along the penis, with other signs of urethritis. An ulcer under the foreskin is betrayed by a hard and tender swelling felt through the skin, and after the lapse of a few days consecutive sores usually appear at the orifice of the prepuce.

**PROGNOSIS.**—The prognosis of balanitis in the simple form is always good. If the complaint is secondary or symptomatic, gangrene is not uncommon.

**TREATMENT.**—The chief indication is to keep the inflamed surfaces separate. After washing and thoroughly drying, the excoriations should be touched with a 10-grain solution of nitrate of silver, and a bit of dry lint laid on the glans before the foreskin is drawn forward. If there is phimosis, frequent injections of tepid water, and twice daily of a 5-grain solution of nitrate of silver, must be thrown to the farthest part of the foreskin with a long-nozzled syringe. Leeches to the groins, and morphine internally, or hypodermically, relieves pain. Acupuncture gives vent to simple œdema, but tend to accelerate gangrene with brawny tension and erysipelatous redness. Incisions, if needed, should be free; one on each side, carried quite back to the furrow. The upper half of the foreskin can then be easily turned back, and the subsequent deformity is less than if the foreskin were divided at the dorsum. In paraphimosis, before replacing the swollen foreskin, the tension should be relieved by acupuncture and astringent lotions, or by incisions if needed. Chancres and inflamed inguinal glands must be treated with the balanoposthitis according to their needs. When there is an ulcer, concealed by phimosis, and the syringing with warm water has cleared away the discharge, an injection of 20 drops of the following emulsion (iodoform, 1 part; powdered tragacanth,

1 part; glycerine, 3 parts; water, 10 parts) rapidly subdues irritation and ulceration. If the signs of syphilitic infection be present; *i.e.*, the persistence of defined induration when the general inflammatory swelling is subsiding, quill-like thickening of the dorsal lymphatic ducts of the penis, and multiple enlargement of the lymphatic glands in the groin, mercury should be administered without delay, in quantity sufficient to bring on and maintain slight sponginess of the gums.

BERKELEY HILL.

**BALDNESS.**—SYNON.: Alopecia; Fr. *Calvitie*; Ger. *Kahltheit*.

DESCRIPTION.—Baldness or loss of hair presents an extensive range of variation in degree, from moderate thinness of the hair, such as occurs in *deftuvium capillorum*, to complete baldness—*alopecia calva* or *calvities*, the latter not limited to the scalp alone, but involving eyebrows, eyelashes, beard, and every hair of the body. Instead, however, of being *general*, baldness may be *partial*, affecting more or less of the surface of the scalp for example, the summit and forehead in men, and the summit and occiput in women. One remarkable form of partial baldness has been denominated *alopecia areata*, or simply *area*, and as this was described by Celsus, it has likewise been called *area Celsi*. *Area* occurs suddenly, and is a mere falling-off of the hair over a space of circular figure; there may be one or more of such *areæ*, and sometimes *area* is only the beginning of *calvities*. *Area* is likewise occasionally met with in the whiskers and beard. See ALOPECIA AREATA.

ÆTIOLGY.—The causes of alopecia are exhausted nutritive power of the skin; nerve-paresis in the case of *area*; syphilis; and local injury. The alopecia of syphilis follows the plan of distribution of its exanthem. Partial alopecia may result from a blow; from the accidental tearing out of a lock of hair; from the sting of a bee; from nervous shock; or from other causes.

PATHOLOGY.—The pathology of alopecia is a loss of nutritive power of the skin, sometimes progressive, and consequent on advancing age, as in general baldness; and sometimes limited to a nerve-district of small extent, as in *area*. This fact is very evident in the latter form, inasmuch as, conjoined with the sudden dropping-out of the hair, the integument is pale and thin, poorly nourished, somewhat anæsthetic, and thinner in the centre than at the circumference; while the hairs which remain at the periphery are altered in structure, clubbed and broken off.

TREATMENT.—This consists in the restoration of nerve-power and nutritive power, and in local stimulation. The best applications for the latter purpose are the stimulating lini-

ments of the British Pharmacopœia; *e.g.* liniment of ammonia, compound camphor liniment, and the liniments of chloroform and mustard; or the acetum cantharidis properly diluted for general alopecia, or applied with a brush in its concentrated form for *area*. In the treatment of the latter, ammonia, turpentine, and the compound tincture of iodine are likewise useful; while for syphilitic alopecia the white precipitate ointment with camphor is the best local application, conjoined with anti-syphilitic constitutional treatment. See HAIR, Diseases of. ERASMUS WILSON.

**BALNEOLOGY** (*Βαλανείον*, a bath; and *λόγος*, a word).—A scientific exposition of all that relates to baths and bathing. See BATHS; and HYDROTHERAPEUTICS.

**BALNEOTHERAPEUTICS** (*Βαλανείον*, a bath; and *θεραπεία*, I heal).—That department of therapeutics which deals with the application of baths in the treatment of disease. See BATHS; and HYDROTHERAPEUTICS.

**BARBADOES LEG.**—A form of Elephantiasis. See ELEPHANTIASIS.

**BARBIERS.**—A synonym for Beriberi. See BERIBERI.

**BARÈGES, in France.**—Thermal sulphur waters. See MINERAL WATERS.

**BARTFELD, in Hungary.**—Alkaline saline chalybeate springs, with iodine. See MINERAL WATERS.

**BASEDOW'S DISEASE.**—A synonym for exophthalmic goitre. See EXOPHTHALMIC GOÏTRE.

**BATH, in Somersetshire.**—Simple thermal and earthy waters. See MINERAL WATERS.

**BATHS.**—Baths may be regarded as *simple* and *composite*, *medicated*, or *artificial*. They may be used in the form of liquid, vapour, or air. We shall consider them under these heads in the following description.

A. SIMPLE BATHS.—I. Simple Liquid Baths.

1. *The Cold Bath.*—By a cold bath is meant the immersion of the body in water below the temperature of 70° Fh. Anything below 50° is considered a very cold bath. The first effect of the bath is a sensation of cold amounting almost to shivering, with slight gasping for breath. If the bath is continued for more than two or three minutes, the temperature of the skin is diminished; and if it is protracted, the blood and the subjacent tissues lose a little heat, but this does not generally occur till after quitting the bath. If the cold is intense and prolonged, there is a certain degree of numbness of the skin; while the pulse becomes small, and

may fall from ten to twenty beats in the minute. After a short time (the colder the water the shorter), reaction takes place, bringing redness to the skin and increase of temperature, with a certain amount of excitement; but if the bath be continued, the depression returns. The immediate action of the cold bath is to cause the capillaries to contract and repel the blood from the surface, while by its operation on the peripheral extremities of the nerves, it acts upon the central nervous system. In its more remote effects, the cold bath accelerates the transmutation of tissues, augmenting the excretion of carbonic acid and of urea from the system, and, as a consequence, increasing the appetite.

The body is usually immersed at once in cold water, but the shock of this may be diminished by first using tepid water, and then gradually adding cold to it.

The effect of a cold bath depends much on its duration. Brief immersion, that is for three or four minutes, makes both the depressing and the exciting action less; a longer duration, say of ten to fifteen minutes, increases both actions; but if the bath be very protracted, the continued abstraction of heat produces depression only. The effects of a cold bath are less intense if the bather is able to keep himself in motion, and especially if he swims.

2. *The Warm Bath.*—A warm bath of 96° to 104° Fh. produces no shock to the system; it causes a moderately increased flow of the circulating fluids to the surface, augmenting the frequency of the pulse; and scarcely affects the respiration. There is not the depression or the excitement of a cold bath. It rather retards the transmutation of tissues. With a hot or very hot bath—from 104° to 114°, the central nervous and circulatory systems are more affected. The frequency of the pulse increases greatly. The respiration becomes anxious and quickened. The skin is in a hyperæmic condition, and a free perspiration breaks out.

3. *The Tepid Bath.*—Tepid baths of the temperature of 85° to 95° Fh., are intermediate between cold and warm. Their effects seem to be confined to the peripheral extremities of the nerves, and they do not excite the nervous centres or the circulatory system. Neither the pulse nor the excretions and secretions are affected. As no heat is confined in the system or taken from it, there is no reaction, and the body temperature is unaltered.

It need scarcely be said that drying and rubbing after a bath materially assist its action on the skin; or that, according to circumstances, it may be convenient to order a whole bath, a hip-bath, or a slipper-bath. The foot-bath is a very useful and convenient one, especially when some stimulant substance is added to the simple water. Wet packing and the various pro-

cesses of hydropathy, and those powerful agents hot and cold affusion, whether as shower-baths or as douches, are described in the article on HYDROTHERAPEUTICS.

The *duration* of a bath must depend on a variety of circumstances—for instance, on the age and constitution of the patient, on the nature of his malady, and on the temperature of the bath. It may vary from a few minutes to many hours. A very hot or a very cold bath can be supported for a much shorter time than a tepid one.

**ACTION AND USES.**—Cold baths are indicated for the strong, for youth, and for manhood; warm baths for the delicate, for women, for early childhood, and for old age. Tepid baths are suitable for almost all constitutions, sexes, and ages. Cold baths may, in a general way, be considered tonic and bracing; they are useful when judiciously employed in many nervous affections, as in chorea and hysteria, and they are the best of all for general hygienic purposes. Of late years they have been specially employed in the treatment of fever (*see* TEMPERATURE). The great value of warm baths, besides their hygienic employment, as better detergents than cold ones, is in soothing and reducing excitement; in relieving spasms, such as colic and retention of urine; in the convulsions of children, combined with the affusion of cold water on the head; in cases of gout and rheumatism; and generally when action on the skin is desired. Where prolonged immersion is wanted, tepid baths are indicated, as in calming many chronic nervous disturbances, and in many cutaneous affections.

As to contra-indications, all baths, and especially prolonged and even tepid baths, are not suited for the asthenic. Both hot and cold baths are to be avoided where there is a weak, fatty heart, or any tendency to apoplexy. No one should ever enter a cold bath when exhausted, and such baths are also contra-indicated when there is a tendency to congestion of internal organs. Under such circumstances a warm bath is usually both safer and more refreshing. The too long and too frequent use of hot baths is debilitating.

**II. The Simple Vapour Bath.**—A vapour bath is one in which the skin is exposed to the action of hot water presented in the form of vapour. The vapour bath may be taken in a box with the head included or not; or in the more common form of the Turkish or Russian baths, where a large room is filled with vapour, and where therefore the vapour is inhaled; or by vapour obtained from a small and suitably constructed apparatus, which vapour may be diffused over the whole body or directed to a particular part. A very simple apparatus for the vapour bath may be prepared by placing under a chair a shallow earthenware or metallic pan,

containing boiling water to the depth of three or four inches, and from which abundant vapour can be obtained by placing in it one or two red-hot bricks. The patient sitting on the chair, surrounded by blankets and other suitable covering, will receive the full benefit of a vapour bath. Vapour baths produce profuse perspiration, and act in cleansing the skin much as hot-water baths do, only more powerfully. Vapour being a slow conductor, does not act so fast on the body as water. Vapour baths can be borne hotter than warm-water baths, but their use cannot be continued so long, as vapour interferes with radiation of heat from the body. In such baths a heat of more than  $122^{\circ}$  Fh. is not borne comfortably. The vapour bath, though falling considerably short in temperature of the air bath, raises the heat of the blood somewhat more. The great virtue of these baths lies in their sweat-producing properties. The average loss of perspiration by the use of a Russian bath has been set down at from  $\frac{2}{3}$  lb. to 3 lbs. In the Russian bath a slight degree of stimulation of the skin is caused by switching it with twigs of birch, and the alternation of depression and excitement of the cold bath is obtained by placing the patient, when in a state of profuse perspiration, under a douche of cold water.

**III. The Simple Hot-Air Bath.**—There are two forms in which the hot-air bath is administered: according as the patient does or does not breathe the heated air. The action of the former closely resembles that of a vapour bath, but differs from it in not impeding the respiration, as the latter does by depositing moisture in the bronchial tubes. The lungs, instead of requiring to heat up the inspired air, are subjected to a temperature above their own. Hot-air baths favour the highest degree of perspiration, while the moisture of vapour baths somewhat retards it. If they are very hot, they raise the temperature of the body by several degrees.

As the arrangements for vapour and hot-air baths are practically the same (except that in the latter it is attempted to exclude all vapour from the *calidarium* or *sudatorium*, the hottest room), the following description of an ordinary hot-air bath, the arrangements of which are closely copied from the Romans, will answer for both.

The patient, after unclothing, first goes into the *tepidarium*, which has a temperature of  $113^{\circ}$  to  $117^{\circ}$  Fh., in which he remains until the perspiration bursts forth, which happens in from twenty-five to forty minutes. He next proceeds to the hottest room or *calidarium* (in which the air is heated by hot-air pipes which are inserted in the walls), of a temperature of  $133^{\circ}$  to  $140^{\circ}$  Fh., and remains there until the perspiration runs down his skin, in twelve to eighteen minutes. An attendant then rubs off the perspiration with a woollen glove, and kneads all the muscles for four or

five minutes. The patient next betakes himself to the *lavacrum*, where he has water poured over him of the temperature of  $81^{\circ}$  to  $86^{\circ}$  Fh.; next, the whole body is soaped over, the suds are rubbed off, and the patient goes to the *frigidarium*, where he lays himself on a couch and waits till his skin is completely dry. This may occupy twenty-five to thirty minutes, when the patient dresses and leaves the bath greatly refreshed.

Such is a brief account of these baths, the revived use of which is at present so general. The arrangements vary in detail. For ordinary purposes it is easy to furnish either vapour or hot-air baths. A great variety of apparatus have been invented for this purpose, which resolve themselves into this, that the patient should lie in bed or on a seat, and have the bedclothes or other covering secured from contact with him by the employment of a framework or cradle. Beneath this hot air or vapour is introduced, either directly or indirectly, from a suitable apparatus.

*The Sand Bath.*—We may here mention baths of sand, which are a very old remedy. Of late years establishments for supplying them have sprung up in various towns. They are a convenient way of applying dry heat either locally or generally, and are employed in chronic rheumatism. Bags filled with heated sand (or salt) are useful in hospital and in domestic practice.

**USES.**—Both hot-air and vapour baths are indicated when increased action of the skin is desired. They are used most for the cure of catarrhs, of neuralgic and rheumatic pains, and sciatica. They have also been much employed for reducing obesity. They are useful for general hygienic purposes, but are apt to be given too indiscriminately. Hot-air and vapour baths are often locally applied with great advantage to a hand, or leg, or arm, in rheumatism or thickened joints.

**B. COMPOSITE, MEDICATED, OR ARTIFICIAL BATHS.**—A great variety of substances have been used in baths at different periods. We must confine ourselves to such as are at present in use and appear to be of real value, omitting even some that are employed, such as baths of iodine, of iodide of potassium, of iron, of fermented grapes, and of whey.

### I. Composite Liquid Baths.

**1. The Sea-Water Bath.**—The average amount of salts in sea-water may be set down at 3 per cent.; this may therefore be considered a suitable strength for ordinary salt baths. The quantity commonly used in London hospitals is about 9 lbs. of salt to 30 gallons of water. Some use bay salt, others Tidman's. Owing to the high price of sea-salt in inland continental places, various natural salts, some of them containing a comparatively small amount of chloride of sodium, have been suggested as substitutes; and also, for economy's sake, 22 to 25 gallons have

been set down as a minimum amount of water for the bath of an adult. The value of these substitutes can only be determined by observing the degree in which they stimulate the skin. Apparently it does not matter much what particular salt is employed to produce the stimulation. A salt bath can of course be increased to any strength by the addition of salt, or of the mother lye as it is termed.

The chief uses of salt-water baths are as tonic remedies, especially for the young, when there is any tendency to scrofula or chlorosis; also in convalescence from many diseases.

2. *Alkaline Baths*.—Alkaline baths may be made by adding 6 ounces of crystallised carbonate of sodium, or 3 ounces of carbonate of potassium, to 25 or 30 gallons of water. Alkaline baths are of use in a great variety of cutaneous affections.

3. *The Corrosive Sublimate Bath*.—Baths of corrosive sublimate are occasionally employed. They are commonly made by adding 3 drachms of corrosive sublimate and 1 drachm of hydrochloric acid to 30 gallons of water. They are employed in some skin-affections, and in secondary syphilis.

4. *Sulphuret of Potassium Bath*.—Baths of sulphuret of potassium are made by dissolving from 4 to 8 ounces of that salt in 25 to 30 gallons of water. A little diluted sulphuric acid is sometimes added. These baths have long been extensively employed in the treatment of cases of skin-disease in which the sulphur they contain is indicated.

5. *The Nitro-Muriatic Acid Bath*.—The nitric or rather nitro-muriatic acid bath is made by adding nitro-muriatic acid to water. The ordinary proportion is one ounce of acid to one gallon of water. The discolouring action on clothing makes a full bath of this kind inconvenient for domestic use, and it is best to take it in a bathing establishment. For the ordinary purposes of a foot-bath at home the old directions of Dr. Helenus Scott, who introduced the use of the acid, are sufficient. The vessel must of course be of wood or earthenware. Dr. Scott added four to six ounces of the acid to three gallons of water. This made a rather strong foot-bath. The patient was to keep his feet immersed for thirty minutes; and the bath was to be repeated every other day for two or three weeks. The axillæ, the groin, and the region of the liver were to be sponged with the acid solution. The bath causes slight tingling of the skin and a taste in the mouth, and is believed occasionally to produce salivation. This bath has been used very extensively in India and in England in liver affections. There is difference of opinion as to its value; many have great confidence in it.

6. *The Bran Bath*.—The bran bath is made by boiling four pounds of bran in one

gallon of water, straining, and adding the liquor to a quantity of water sufficient for a bath. Such a bath is useful in allaying irritability of the skin, and also in diminishing the stimulating effect of other baths.

7. *The Fucus Bath*.—This is made by adding a decoction of sea-weed, or the sea-weed chopped up, to an ordinary bath; it will become more or less gelatinous if enough be added. Such baths go popularly by the name of *Ozone Baths*; and they contain a certain amount of chloride of sodium and a minute proportion of iodine. They are useful in the same cases as sea-baths.

8. *The Mustard Bath*.—An extremely useful stimulating bath is the well-known mustard bath, which is made by adding a handful or two of mustard to the ordinary hot-bath. The pediluvium is its most useful form.

9. *Pine Baths*.—Baths of the balsam of pine-leaves may be prepared extempore by making decoctions of the fresh leaflets at certain seasons; but the usual way is to add about one pound of the extract, which is prepared from the leaves, and is everywhere for sale—at least in Germany. The extract dissolves in the bath, which is then ready for use; but of late it has been usual to add a small amount of an essence which is also prepared from the leaflets. It floats to the surface of the water, and attaches itself to the person on leaving the bath, and its aroma is grateful. Of course the quantity of the extract to be employed depends on its strength. These favourite baths are now more and more largely employed at all bathing establishments in every corner of Europe. They are slightly stimulant, and are much used in hysterical, rheumatic, and gouty affections, and also as an adjunct to the internal use of mineral waters.

10. *Baths of Conium, Lavender, &c.*—Aromatic or sedative baths are prepared by adding a decoction of lavender, hyssop, or conium to an ordinary bath.

It is scarcely necessary to add that, as a rule, all composite liquid baths should be of a temperature a little above the tepid; and that their strength, and the time that the patient is to remain in them, must be determined by the special circumstances of the case.

II. *Composite Vapour Baths*.—Vapour baths impregnated with *fir balsam* are popular, and are considered to be more powerful in their operation than pine baths. The vapour which rises in making the decoction of pine leaves is conveyed to a box in which the patient is enclosed.

Aromatic vapour baths may be given by making the steam of hot water pass through bunches of fresh aromatics (*conium, lavender, &c.*) before reaching the box in which the patient is placed. Such baths may be useful in hysteria.

### III. Composite Air Baths.

1. *Sulphurous Acid Bath*.—A valuable mode of applying sulphur in the form of a bath is by using its fumes—in other words, sulphurous anhydride. The patient is seated on a cane-bottomed chair, and his body is encircled with a cradle, over which oil-cloth is thrown, the head remaining uncovered. Sulphur is placed on a metallic plate, to the lower surface of which the flame of a lamp is applied, when sulphurous acid is disengaged. This bath is less used in cutaneous affections than formerly.

2. *The Mercurial and Calomel Baths*.—Very similar is the mode of applying the fumes of mercury. Under the chair are placed a copper bath containing water, and a metallic plate on which are put from 60 to 180 grains of the bisulphuret or of the grey or red oxide of mercury. Spirit-lamps are lighted under the bath and under the plate. The patient thus experiences the effects both of aqueous and of mercurial vapour. At the end of five or ten minutes perspiration commences, which becomes excessive in ten minutes or a quarter of an hour. The lamps are then to be extinguished, and when the patient becomes moderately cool he is to be rubbed dry. He should then drink some warm liquid and remain quiet for a time. This has often been a favourite mode of treating secondary syphilis with some practitioners. Calomel, in quantities of from 20 to 30 grains, is administered in a similar manner, under the name of the *calomel bath*. It may be given locally by a suitable apparatus.

IV. *Electrical Baths*.—Electricity is an extremely useful aid in the thermal treatment of paralysis; and Electro-Galvanic Baths are often connected with other bathing establishments, it being a very popular practice to galvanise a patient in water-baths between the poles of a battery. See ELECTRICITY.

JOHN MACPHERSON.

**BATHS, Natural.**—See MINERAL WATERS.

**BATTAGLIA, in the province of Venice in Italy.**—Thermal muriated saline waters, with sulphate of lime. See MINERAL WATERS.

**BEAUFORT WEST, in Cape Colony.**—See AFRICA, SOUTH.

**BED-SORE.**—See ULCER; and ULCERATION.

**BELL-SOUND.**—A peculiar physical sign associated with pneumothorax. See PHYSICAL EXAMINATION.

**BELL'S PARALYSIS** (named after Sir Charles Bell).—A synonym for paralysis of the facial nerve. See FACIAL PARALYSIS.

**BERIBERI.**—SYNON.: Barbiere; *Kakke*; and numerous other local names.

**DEFINITION.**—A disease characterised by anæmia, anasarca, degeneration of muscular

tissues, effusion into the serous cavities; debility; numbness, pain, and paralysis of the extremities, especially the lower; præcordial anxiety, pain, and dyspœnia; scanty and high-coloured urine; and in some cases drowsiness or sleepiness.

Beriberi occurs in a chronic and an acute form; in the latter often proving rapidly fatal from exhaustion, syncope, or the formation of cardiac or pulmonary coagula. There are two forms, the wet (*beriberia hydrops*), in which there is a hydræmic condition of the blood and distension of the areolar tissues generally with serum, giving the body a bloated appearance; and the dry (*beriberia atrophica*), in which there is a notable deficiency of fluid in the vessels and in the areolar tissues, and early atrophy of the muscles.

**ETYMOLOGY.**—The etymology of the word Beriberi is obscure. *Bhër-bhëri*, a Hindi word, signifies a sore, a swelling. Some persons believe that it is derived from a Cingalese word *beri*, meaning weakness; *beriberi* signifying great weakness.

**GEOGRAPHICAL DISTRIBUTION.**—Beriberi prevails endemically in Ceylon; and in India, on the Malabar Coast, and in the Northern Circars, between 13° and 20° N. latitude, extending inland from forty to sixty miles. It is known in other parts of India, probably occasionally all over the peninsula; in Burmah, and the Malayan peninsula; amongst the crews of ships trading to ports in the Persian Gulf, Red Sea, coast of Africa, Bay of Bengal, Singapore, China, Siam, and the islands of the Indian Archipelago, and in the Australian seas. On the West Coast and other parts of Africa beriberi also occurs, and is known as the *sleeping sickness*. In Europe *pernicious anæmia* resembles the same disease. Beriberi is also met with in South America, and probably wherever certain conditions of food, water, soil, climate, and mode of life coexist.

**ÆTIOLOGY.**—All observations tend to show that beriberi occurs where causes of debility have for some time operated, especially in the climates and localities previously mentioned, such as certain conditions of soil, air, and water; exposure to great alternations of temperature, especially when accompanied by wet, fatigue, mental and physical depression; food deficient in quantity and quality or variety; previous exhausting diseases; malaria, and other undefined atmospheric and telluric influences—all, in fact, that tends to depress the vital energies, impoverish the blood, and starve the nerve-centres. As regards a specific origin of the disease, Ogata and de Lacerda claim to have found bacilli in the blood in five out of seven cases of the disease, and to have cultivated them. They are said to be similar to, but somewhat smaller than those of, anthrax; and inoculation of mice, rats, and monkeys is said to

have been followed by symptoms characteristic of beriberi. It would be premature, in the present state of the investigation, to regard their causal relations as established. The symptoms, it is said, seldom begin to appear within ten months or a year after first exposure to the exciting causes. Beriberi has been ascribed by Ranking to disease of the kidney, but there is no evidence to prove that it is due to this cause, or indeed to structural changes of any of the viscera. Morehead refers it rather to a scorbutic origin, and in some respects it does resemble scurvy; it may, probably, also, be a consequence of the cachexia that so often results from long residence in a malarious climate, especially when that has been accompanied by exposure, privation, and excessive exhaustion of the vital powers. In such, the most complete anæmia, with debility, may occur independently of the existence of organic visceral disease, though naturally the symptoms will be intensified where such disease is present. An important contribution to our knowledge of beriberi has recently (January 1890) been made by Surgeon Thomas of the Indian Medical Service. He says that, in a very large number of cases he met with, the proximate cause was the presence of ankylostoma duodenale in the intestinal canal, and that the severity of the disease was in proportion to the number of parasites present. He refers to the discovery by Ogata, of Tokio, of a specific bacterium, and to the statement of Drs. Pekelharing and Winkler of Utrecht, that the cause of beriberi is a micrococcus, so that it is an infectious disease.

**ANATOMICAL CHARACTERS.**—Serous fluid is effused generally—in the areolar tissue, in the lungs, brain, heart, and abdominal viscera. The cavities are, like the tissues, soaked with watery effusion. The tissues are soft and degenerate. Muscular fibre is fatty, especially that of the heart, which is often enlarged and dilated. The kidneys are enlarged, anæmic, and softened.

**PATHOLOGY.**—The discovery, by the late Dr. T. Lewis, in India, of the embryo of a nematode worm in the blood of persons suffering from chyluria (*see* CHYLURIA), lymphorrhœa, and elephantiasis, of which diseases it appears to be, to some extent, the cause, suggests the inquiry whether a similar hematooxon may not also be in some way concerned in inducing beriberi. Fonsagrives and Leroy de Mericourt describe beriberi as general dropsy with a rapid course, weakness and loss of sensibility in the lower limbs, but no albuminuria. Dropsy commences as anasarca, and extends to the serous cavities. Though hepatic, splenic, or renal complications may exist, and intensify the severity and hasten the progress of the general symptoms, they are not essential concomitants of the disease, but appear to originate in a spanæmic state

of the blood, and to be kept up by its progressive imperfect elaboration. The resulting partial starvation of the cerebro-spinal nerve-centres, and the serous effusion into and amongst them, sufficiently account for the paralysis which, in severe cases, characterises this disease.<sup>1</sup>

**SYMPTOMS.**—Beriberi presents itself under a *chronic* and an *acute* aspect, rarely, it is said, occurring in either form until after an exposure of some months to the exciting causes. The general symptoms may be said to be those of anæmia and anasarca. Œdema pervades the limbs and body generally, accompanied with numbness, pain, heaviness, and loss of power, amounting in some cases to paralysis. Along with these symptoms there occur præcordial anxiety, dyspnœa, irregularity and palpitation of the heart, pain at the ensiform cartilage, anæmic murmurs, debility, and a small quick pulse, which at the outset may be rather hard and full, accompanied by dryness and heat of skin. The appetite is at first not impaired.

<sup>1</sup> Since the present article was written, certain very recent advances in neuro-pathology have made it possible to speak more decidedly on the relation of the nervous system to beriberi. The elaborate researches of Baelz and Scheube in Japan, and of Pekelharing and Winkler in Java, go far to elucidate this matter. In the light of these researches it may be said that, whatever the exciting and predisposing causes may be, the brunt of the disease falls in the first instance, and mainly, on the peripheral nerve-fibres. In beriberi the peripheral nerves are invariably found in a state of well-marked degeneration; and the farther from the cord the more pronounced the degeneration. That the neuritis is of a peripheral character is proved by the absence of degenerative or other changes in the brain, cord, and anterior nerve-roots; and also by the arrest, for the most part, of the degenerative changes at the intervertebral ganglia, which shows that the degeneration is an ascending not a descending one. Pekelharing and Winkler have also shown that the electrical signs of nerve-degeneration can be elicited in cases of impending beriberi during the premonitory stage. Thus beriberi may be defined as a specific endemo-epidemic, multiple peripheral neuritis; and classed with the neurites associated with lead, alcohol, diphtheria, &c., which it so closely resembles. In this way the pathognomonic combination of symptoms may receive a satisfactory explanation: implication of the spinal nerves causes the paresis and anaesthesia; implication of the vaso-motor nerves causes the œdema; and involvement of the vagus, the cardiac plexus, and the phrenic accounts for the laryngeal symptoms, the palpitations, and the breathlessness.—EDITOR.

Later there is coldness of the extremities; torpor of the bowels; scanty, high-coloured urine, of sp. gr. 1020 to 1040, but no albuminuria as a rule. According to Horton and others, excessive drowsiness and stupor attend some stages of certain cases of the disease; also pale, flabby tongue, and blanched mucous membranes; occasionally hæmorrhage from the stomach and bowels; with petechial eruptions; an anxious look; a puffy, swollen, and sometimes livid face; and a peculiar tottering gait. Death results rapidly in some of the acute cases, with symptoms of effusion into the thoracic and abdominal cavities, or within the skull, by exhaustion, syncope, or the formation of coagula, either in the systemic or in the pulmonic circulation. Beriberi frequently assumes a slight and modified form, indicated by anæmia, numbness, and a certain amount of pain in the limbs; an anxious expression; disordered bowels; scanty urine; cold skin; a low, feeble, and irregular pulse; præcordial pain or uneasiness, with palpitation; nervous depression; an unsteady, almost tottering gait; and a puffy face and neck. Dr. Paul says: 'I have met with a numerous class of cases that are not so serious (as the acute) or so often fatal, where the chief symptom complained of was burning of the feet.' Malcolmson describes this remarkable condition in connexion with beriberi, to which, he says, it is allied—it is found to affect the soles of the feet and calves of the legs, the back, and occasionally the muscles of the legs. It occurs in recent and slight examples of beriberi, and was originally observed in the troops after the first Burmese war—sometimes in men who had not had beriberi. On the whole, Malcolmson thinks it is neither rheumatism nor beriberi, and may accompany or follow other diseases, as an indication of nervous debility.

In the *acute* forms of beriberi the symptoms are very severe and often rapid; and the mortality would indicate it to be second only to cholera in fatality. The chief symptoms are:—Rapid general anæmia and dropsy of the cavities; scanty, almost suppressed urine; constipation; weak, irregular pulse; intense præcordial pain; hurried, irregular, and painful breathing; occasional vomiting—sometimes of blood; swelling of the limbs, with numbness, pain, and paralysis, accompanied by a feeble, tottering gait; and all the symptoms of pleuritic and pericardial effusion; failing heart; and death either from syncope, or perhaps almost suddenly from embolism—in the most acute cases within a few days. In this acute form the affection is very fatal, but in the milder and more chronic form recovery is frequent. Acute symptoms often supervene in those who have suffered from the milder disease, or in those who are exhausted and anæmic from other causes.

Thomas prefers, to the usual classification

of beriberi into the wet, dry, and the mixed, the following: *acute*, *sub-acute*, and *chronic*.

The symptoms of the *acute* form are pyrexia, rapid anæmia, and anasarca; dyspnoea; dulness on percussion; cough and expectoration; scanty urine; effusion into cavities, pleural and pericardial; peripheral paralysis well-marked, and great nervous depression.

The *sub-acute* form is characterised by epigastric pain, loss of power in lower extremities, wasting of muscles, loss of irritability, loss of patellar tendon reflex; the face is puffy and swollen; the lower limbs are œdematous; there is tremor of lips and hands; decubitus on back and right side; dyspnoea, vomiting, and jaundice are present; hæmic murmurs are heard over the heart; the apex-beat is indistinct, without signs of hypertrophy or dilatation. Mental debility is marked.

In *chronic* beriberi there is no rise of temperature; the face is puffy, swollen, and livid; there is tenderness of the adductors of the thigh and the gastrocnemius; anæmia, palpitation, and anorexia; the pulse is quick and small; aortic murmurs may be heard; œdema is present in the lower limbs with induration, pain, and numbness. The gait is tottering; the heart is dilated or hypertrophied.

TREATMENT.—Attention to diet, suitable clothing, and protection against vicissitudes of temperature, wet, and cold, are the best *preventive* measures against beriberi. Bad hygiene and exhausting habits of life tend to promote the development of the constitutional condition in which the disease commences.

When beriberi is established, these precautions are still necessary, and attention must be paid to the symptoms as they occur. Diuretics and diaphoretics relieve the œdema and dropsical effusions. Tonics and stimulants give vigour to the weakened muscular fibre; while appropriate remedies and diet may improve the condition of the blood.

Acetate of potassium, digitalis, and squill, and occasionally calomel, are said to favour the removal of the fluid. It is needless to say that the physiological action of mercury is to be avoided. Salines, hot-air baths, diaphoretics, and turpentine may at various stages be found useful. The object being to remove the fluid and strengthen the muscular fibre, quinine, iron, and other tonics are an important element in the treatment. No remedies, however, will be of much avail unless the patient be placed in favourable hygienic conditions. Malcolmson speaks highly of two remedies—*trëak farook*, and *oleum nigrum*, which are considered to be very effective in the treatment of the disease, especially in relieving the dyspnoea and œdema, which proved very fatal until these drugs were introduced. The composition of the *trëak* is apparently generally unknown—it seems to be diuretic and stimulating, and probably

not aperient unless combined with rhubarb, in doses of four to fifteen grains. The *oleum nigrum* is a stimulant and diaphoretic, given thrice daily, and has been found by Indian physicians to be very beneficial in some cases of beriberi. Sir William Aitken says that turpentine is a useful remedy. Ergotin, iron, and belladonna with zinc in the form of a pill, accompanied with sea-bathing, were useful in this disease as seen at Bahia. *Nux vomica* has often been found serviceable in the later stages of certain cases, as might be expected; and opium may be needed to allay pain and irritability. Hepatic and splenic complications need their appropriate remedies. Obviously the chief indications are to regulate the functions of the abdominal viscera; to promote removal of the œdema; to increase the action of the skin; and to give tone and vigour to the muscular fibre. By such measures can we alone hope to deal successfully with this profound form of cachexia.

Thomas believes that the first indication of treatment is the expulsion of the parasites, for which purpose he recommends frequently repeated small doses of calomel, with the administration of thymol to destroy the entozoa; the use of purgatives, also, such as jalap, elaterium; subsequently tonics—iron, quinine, and strychnine. For internal effusion of serum paracentesis may be performed. In some cases pericardial effusion has been beneficially treated by removal of the fluid by puncture with a small trocar and cannula.

Change of climate is of great importance, as well as careful attention to diet and other measures for maintaining or restoring the general health. JOSEPH FAYRER.

**BETHESDA SPRINGS, in Waukesha County, Wisconsin, U.S.A.**—Calcareous waters. See MINERAL WATERS.

**BEX, in Switzerland.**—Strong muriated saline waters, with some iodide and bromide of magnesium. See MINERAL WATERS.

**BIARRITZ, in France, on the Bay of Biscay.**—A fashionable seaside resort. The climate is considered to be bracing. See CLIMATE, Treatment of Disease by.

**BILE, Disorders of.**—Disorders of the bile held a large place in the medicine of antiquity, with the exception of the theories of Van Helmont and Paracelsus; the latter looking upon the bile as the balsam of life, and therefore incapable of begetting disease; the former regarding it as a mere excrementitious fluid, and therefore equally incapable of begetting disease. Disorders of the bile have, nevertheless, held their own quite into our time. It is common enough to hear persons speak of a 'bilious attack,' or 'being troubled with the bile,' expressions the survivals of the humoral pathology. The liver was formerly credited with most of the

dyspeptic disorders of the stomach. But, as Sir Thomas Watson says, it is an organ often blamed most gratuitously and unjustly, but no educated or scientific physician would now think of attributing a gastric catarrh, or constipation, to an 'attack of the bile,' or to a 'sluggish liver.'<sup>1</sup>

Physicians have arranged disorders affecting the bile under three heads—(1) diminished secretion of bile; (2) increased secretion of bile; and (3) secretion of morbid or altered bile. This division may very well be accepted as a convenient basis for the further discussion of biliary disorders, but it is nothing more than an hypothesis. It is likely enough that the bile, in certain diseases, changes its character as regards both its amount and constituent parts; but it cannot be denied that the means by which physicians are able to ascertain these changes can scarcely be said to exist in ordinary cases. A common saying is that the patient must be making plenty of bile because the stools are high in colour. No reasoning can be more fallacious. The colour of the stools may be high if the fæces be quickly swept through the intestinal canal, because there has been no time for the bile-pigment to be absorbed into the blood. Or the fæces may be pale if they lie long in the bowel and the colouring matter is absorbed. So that the colour of the stools is no sure sign of the poverty or abundance of the secretion of bile. In cases of pale-coloured fæces purgatives often do good, not because they have any special tendency to increase the flow of bile, but because they hurry the fæces out of the intestine, and thus give no time for the absorption of the bile, which, if it lay long in the bowel, would be absorbed, carried to the liver, and again excreted into the gall-ducts—the vicious circle of Schiff.

Nor is the analysis of the bile found after death in the gall-bladder of much value. Frerichs announced the presence of albumen in the bile in cases of congestion of the liver; but it is now generally thought that this appearance is due solely to a post-mortem transudation. Ritter also has described a colourless bile in which all the constituents of bile are present except the pigments. Most of his analyses were made on bile taken from the gall-bladder after death; but if the cystic duct be obstructed for any time, it is well known that the bile contained in the gall-bladder may become colourless without any real secretion of colourless bile having taken place in the first instance. It is impossible to be certain that the changes, which are found in the bile taken from the gall-bladder after death, have taken place during life.

The only source of what may be called knowledge of the disorders of the bile is

<sup>1</sup> The opinion of this eminent physician is nevertheless open to question.—EDITOR.

observation of men or animals in whom biliary fistulæ have been formed either by disease or by art. Unfortunately, the majority of such observations have been physiological or pharmacological; and but few are recorded of the changes which the bile undergoes in disease. Altogether contradictory experiments are recorded of the influence of the nerves and of the diabetic puncture of the fourth ventricle upon the secretion of bile. It is an admirable field for further research, but it will be seen that our present knowledge very closely approaches to complete ignorance.

It is disputed still whether the presence of bile in the stomach puts an end to the process of digestion. By many it is thought that the bile-acids throw down the albumen of the food, and with the albumen the pepsin. It is well known that in some disorders there is an inverted action of the duodenum, and bile is poured into the stomach, as in long-continued vomiting, for instance; thus the dispute has a practical bearing. The best treatment of this state would seem to be by saline purgatives.

Bile may also be taken up into the blood, and when this occurs, jaundice results. See CHOLEMIA; and JAUNDICE.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

**BILHARZIA.**—The name given (by the late Dr. Cobbold) to a genus of flukes, discovered by Dr. Bilharz, of Cairo. See ENTOZOA.

**BILIARY CALCULUS.**—See GALL-STONES.

**BILIARY FISTULA.**—There are two kinds of biliary fistula:—one, in which a communication exists between the gall-bladder and the surface of the body; the other, in which there is a communication between the gall-bladder and other internal organs. Neither kind is common, but the first is less rare than the other.

In the first variety a tumour forms, sometimes in the place of the gall-bladder, at other times near the umbilicus, in the linea alba or to the left of this line, or in the groin. The tumour, if opened spontaneously or by the surgeon, discharges a quantity of pus, bile, and gall-stones. If the cystic duct be obliterated, no bile need escape. A suppuration of the gall-bladder, caused by the presence of gall-stones, is the common cause of these fistulæ. The prognosis is good. The diagnosis, before the tumour opens, is very difficult. In a case which came under the notice of one of the writers, it was mistaken for an abscess of the liver.

In the second kind of biliary fistula, the gall-bladder may communicate with the duodenum or colon; with an abscess of the liver; with the portal or other abdominal vein, though it must be owned that cases of this kind seem somewhat dubious; or with

the urinary bladder, or at least with some part of the urinary tract. Gall-stones are in nearly every case the cause of the fistulous opening.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

**BILIER, in Bohemia.**—Cold acidulated alkaline waters. See MINERAL WATERS.

**BILIOUS.**—This term is used with much vagueness, and in popular language is often employed very incorrectly, though the general notion is to associate it with conditions in which an excessive formation of bile is supposed to occur. The chief uses of the word are as follows:—In the first place it is employed to designate a peculiar temperament—the *bilious temperament*. Again, individuals are often said to be *bilious* when they present a sallow or more or less yellowish tint of skin, but especially if they are distinctly jaundiced. *Bilious vomiting* and *diarrhœa* signify respectively the discharge of a quantity of bile, mixed with vomited matters or with loose stools. Certain febrile diseases, attended with yellowness of the skin, are sometimes designated *bilious fevers*; and under like circumstances pneumonia has been described as *bilious pneumonia*. Lastly, one of the most frequent applications of the term is to certain so-called *bilious attacks* or *biliousness*, which, however, are commonly merely attacks of acute dyspepsia or of migraine. The most prominent symptoms of a supposed *bilious attack* are anorexia, furred tongue, a bitter taste, sickness, constipation, and headache, with a feeling of marked depression and general malaise. Such attacks are most effectually prevented by careful regulation of diet, and the avoidance of exposure to cold, fatigue, and undue mental exertion or anxiety; when they come on, abstinence from food is desirable, with rest in the recumbent posture, and perfect quiet. Cholagogue aperients and saline effervescents may be given, alcoholic stimulants being avoided as far as possible.

FREDERICK T. ROBERTS.

**BIRTH PALSIES.**—See PARALYSIS.

**BITTER ALMONDS, Poisoning by.**—See PRUSSIC ACID, Poisoning by.

**BLACK VOMIT.**—Vomited matters may be more or less black in different diseases, but the peculiar *black vomit* is that which occurs in yellow fever (see YELLOW FEVER). The rejected matters are acid in reaction, and a sediment is deposited of coagulated albumen and disintegrated blood-corpuscles. Ammonia is also present. The black colour of the vomit has been attributed by some writers to altered bile, but there can be no doubt that it is due to its admixture with blood which has undergone certain changes

**BLADDER, Diseases of.**—SYNON.: Fr. *Maladies de la Vessie*; Ger. *Blasenkrankheiten*.

The bladder may be the seat of the following morbid conditions:—Inflammation, acute or chronic; Abscess; Neuralgia; Atrophy or Hypertrophy; Mechanical Distension, with chronic engorgement and retention of urine, commonly, but erroneously, termed 'Paralysis'; Sacculation; Displacements, such as hernia in the male, or, very rarely, inversion and protrusion in the female; Tumours or Growths of various kinds, from simple Mucous Polypi to Epithelioma, Sarcoma, and Carcinoma; Tubercular Disease; Ulceration, either simple or malignant; and Vesico-vaginal or Vesico-intestinal Fistula. The bladder may also be the subject of true paralysis, partial or complete, as the result of injury to the brain or spinal cord, or following disease of those organs.

Only a brief statement respecting the most important of this class of affections need be presented here, in order to facilitate an acquaintance with their diagnosis, since the treatment of almost all of them belongs to the province of the surgeon, and so far only as it consists of medicinal remedies will the subject be considered.

**1. Acute Inflammation—Acute Cystitis.**—The mucous lining of the bladder is the part affected by inflammation; and although, after long and severe attacks, some morbid action occurs by extension to the muscular coat, or even to the peritoneal covering, these structures are very rarely affected. An acute inflammation of a very severe kind occurs from injuries; from the presence of instruments, foreign bodies, or calculi; and from unrelieved retention of urine. A less severe, somewhat evanescent, but very painful form of cystitis arises from irritants taken internally, as cantharides. A still less severe but often troublesome form originates by extension from gonorrhœa.

In the *first* class of cases there are not only severe local symptoms referable to the bladder, but the general system may be gravely affected.

In the *second* class, of which cantharides-poisoning is the type, the phenomena of very frequent, painful, and spasmodic attempts to eject small quantities of urine, which is often bloody, occur within a very short time after absorption of the poison. A common blister is said to produce the affection in some persons. In two cases—the only two the writer has seen—it has followed the application of a blister to a surface already partially denuded of the scarf skin. In one of these a blister was applied to a knee which had been frequently painted with tincture of iodine, and was still slightly sore. In three hours after the application the patient was attacked with exceedingly painful efforts to micturate, which were at times intense. The attack lasted six

hours, gradually diminishing in force, and leaving no ill-effects behind. In less than twenty-four hours no trace of the symptoms remained.

In the *third* form of cystitis, which is the most common, and of which that arising by extension from gonorrhœa may be taken as the type, the usual symptoms are undue frequency of micturition; a necessity to perform the act immediately the want has declared itself, a condition conveniently expressed by the single word 'urgency'; a desire to pass more, accompanied by pain, when all the urine has been voided; and some dull aching over the pubes; together with a general febrile state of the system, often very slight, but corresponding for the most part with the degree of local inflammation. The urine itself is cloudy, and deposits some light mucus on standing, but it is not otherwise apparently altered. Under the microscope abundance of epithelium is visible, as well as some pus-cells; and if the affection is severe, a few blood-corpuscles are also present. It may be remarked here that the presence of a few pus-cells in the urine—a fact to which so many practitioners attribute considerable importance—by no means necessarily deserves to be so regarded. The very slightest attack either of this or of the preceding form of cystitis is certain to be attended by the formation of some quantity, however small, of fully-developed pus-cells.

Very rarely a *false membrane* may be produced on the surface of the mucous membrane of the bladder, and may be thrown off almost entire, leading to the belief that the inner coat has itself been removed or, as it has been termed, separated by 'exfoliation.' In women this membrane has been voided *per urethram* in a condition for examination; in men this cannot occur, because the urethra is too small to admit of it. Now and then examples of the former have been shown at the Pathological Society of London; and one of the latter, discovered on operation, may be seen in the museum of the Royal College of Surgeons, London. See CASTS.

**TREATMENT.**—In the first form of cystitis, the removal of the exciting cause, if possible, is the chief indication.

The treatment of the second form should consist of very hot bidets or hip-baths, the former being sometimes preferable as capable of being used at higher temperatures than the latter; together with large doses of the tincture of henbane, say a drachm, with 10 or 15 drops of liquor opii (Bat.) every four hours while pain is severe; 20 minims of liquor potassæ may be given either simultaneously or alternately, in water or in any bland diluent.

The treatment of a well-marked case of the third class consists in absolute rest in the recumbent posture, mild diet, abstinence from all alcoholic stimulants, gentle laxative action of the bowels, and the administration of small

doses of alkali. The writer prefers liquor potassæ, frequently repeated, to all others; and this may be combined with henbane, or, if micturition is very frequent and painful, with opium or morphine, or with belladonna in small doses. Hot hip-baths or bidets, followed by hot linseed-meal poultices or fomentations, give great relief. The patient may drink freely of decoction of triticum repens, linseed-tea, barley-water, or similar demulcents. Relief rapidly follows, but care is requisite to avoid relapse, which easily occurs if exercise be taken too soon, if injections for the gonorrhœa be resumed too readily, or if alcoholic stimulants be indulged in.

**2. Chronic Inflammation—Chronic Cystitis.**—Chronic inflammation of the bladder is separated from the acute form by very distinct characters. It is mostly the result of retained urine from stricture or enlarged prostate; but it may arise from the presence of calculi, or of growths in the bladder; from over-distension, or atony of its coats; from paralysis after injury or disease affecting a nervous centre; from disease of neighbouring organs; and sometimes from altered urine: it is also met with in certain affections of the kidney. Sometimes this condition is marked by the presence of a large quantity of viscid mucus, often called 'catarrh'; but more commonly this symptom is absent, and the urine contains merely ordinary mucus or muco-pus, rendering the secretion more or less cloudy and opaque. Perhaps there are some cases in which the inflammation is mainly due to the existence of gout.

**SYMPTOMS.**—The symptoms of chronic cystitis are increased frequency of micturition and pain, but the latter is by no means necessarily present. The urine is always cloudy, and contains some pus-cells. There is often, but not always, some supra-pubic uneasiness. The general health does not suffer unless the affection is prolonged or severe. If important causes, as the presence of stricture, calculus, &c., occasion the chronic cystitis, their specific symptoms will predominate. It is not common to find chronic cystitis as an idiopathic disease, although undoubtedly it occasionally is so; so that the writer has always regarded it as a useful maxim, 'When chronic cystitis is declared to be idiopathic, we may be sure that we have only not yet discovered the real cause.' As a general rule we may suspect that there is inability to empty the bladder, or calculus, stricture, or organic disease of some kind in some part of the urinary tract, when the group of symptoms are present which we denote by the term 'chronic cystitis.' Respecting the well-known glairy mucus, which is deposited so abundantly from the urine in some cases, in elderly people almost invariably, it should be said that it appears only in those whose urine is abnormally re-

tained, through atony of the vesical walls, or in consequence of enlarged prostate, or as the result of sacculation of the bladder, and that medicine has little or no effect upon it.

**TREATMENT.**—The regular and habitual use of the catheter, and perhaps also injections into the bladder, form the essential mechanical treatment of chronic cystitis in the cases just mentioned. In the few cases in which chronic cystitis is present and no organic cause, such as those named above, can be discovered—and also as adjuncts to mechanical treatment when these causes do exist—certain medicinal agents are undoubtedly useful: these are buchu, triticum repens, uva ursi, alchemilla arvensis, pareira, and the alkalis—potash and soda. Buchu is more useful in subacute and recent chronic cystitis than in cases already of long duration. The patient should take not less than ten ounces of the infusion daily. After this, in similar cases, the decoction of triticum repens (made by boiling two to four ounces of the prepared underground stem in water to make a pint and a-half of decoction, of which six ounces are taken three or four times in the twenty-four hours) is highly useful. For more chronic cases, where the urine is alkaline and deposits much mucus, and perhaps the triple phosphates also, alchemilla, uva ursi, and pareira may be very valuable. The alchemilla is administered in infusion, one ounce of the herb in one pint of boiling water, of which the dose is four to six ounces three times daily; the others to be made according to the directions of the Pharmacopœia, but taken in quantities at least two or three times as great as those there suggested. These may be taken alone or combined with potash, which in moderate quantities diminishes the natural acidity of the urine before it enters the bladder; the mucous membrane of which, although accustomed to that condition in health, is perhaps sometimes, when inflamed, irritated by urine of even the ordinary acidity. Whether this be so or not, there is no doubt that alkalis do frequently tranquillise an irritable bladder. They are often given in the form of Vichy water, Vals water, or that of Evian—all strong solutions of soda; but on many grounds the salts of potash are preferable. On the other hand, the mineral acids have been largely administered in cases where the urine is alkaline; although there is no reason to believe that the acid has any direct action through the kidneys, or that it is eliminated by those organs. Alkalinity of the urine in chronic cystitis is almost always due to inability of the bladder to empty itself, and the remedy wanted is not medicine but a catheter, at all events to ascertain whether this be the cause or not. A very small quantity of urine retained in the bladder, say one or two ounces, after every act of micturition, suffices in some cases to maintain an alkaline and

otherwise unhealthy state of the secretion; while it is equally true that some patients may habitually, and during long periods of time do, fail to empty the bladder, always leaving behind from half a pint to a pint, without the urine losing its acidity. Of course other signs, and notably great frequency of micturition, are present when such is the case.

3. **Neuralgia.**—It is impossible to deny that the bladder may be subject, like other parts of the body, to symptoms which are described as neuralgia, although the occurrence is an extremely rare one. All the writer can say is, that he has occasionally met with cases in which he has not been able to account, by the existence of any lesion, for pain and frequency in micturition, or for difficulty in performing that act, and where these symptoms have been more or less periodic in their appearance. In such instances he has given quinine, and has occasionally found great relief to follow a few doses; more frequently this has not been the case. But now and then the value of the drug has been so marked as to corroborate a belief in the existence of vesical neuralgia. It must be repeated, however, that examples of such phenomena are extremely rare. The writer has also employed arsenic on the same ground. It is invariably necessary to investigate the general health, as well as the habits and diet of the patient. This, perhaps, may be the place for stating that in all chronic and slight deviations from natural and healthy function in the urinary organs, it is essentially necessary to inquire into the state of the digestive organs, and, if possible, to correct by diet, and by medicine when necessary, any imperfect action on their part. Constipation alone, when habitual, may produce considerable irritability of the bladder, a slight affection of the organ already existing; so also may the unnecessary use of purgatives. A gentle, easy, and daily action of the bowels, a healthy condition of the primary digestion, the absence of flatulence and distension after food, should be ensured as far as possible in all patients complaining of frequent, difficult, or uneasy micturition; and many such may be completely cured of so-called urinary affections by strict attention to these matters. The writer's strong convictions relative to this fact, grounded on innumerable experiences of its value as an aid in practice, led him, upwards of twenty years ago, to ascertain the great value, for such patients, of the natural aperient waters of Germany and Hungary, now so extensively used; and he now more than ever insists on the use of a mild and laxative regimen and careful diet in their management.

4. Where obstruction to the outflow of urine exists (stricture of the urethra, most forms of enlarged prostate, other tumours, &c.), the muscular walls of the bladder be-

come the seat of **Hypertrophy**, which is a condition of compensation, therefore, and not of disease in or by itself. But such changes in the interlacing muscular fibres existing, **Sacculation** readily occurs, by protrusion of the lining membrane between the bands so produced. On the other hand, most commonly when the prostate is much enlarged, the bladder becomes gradually distended, its coats become expanded, thinned, and weakened, and a certain degree of **Atrophy** takes place. The power of the organ to expel urine is lost or diminished; and micturition being a function of a simply mechanical nature, the circumstances of the case demand only a mechanical remedy, viz. the catheter. No medicine can restore power and exercise of function under these circumstances. But atrophy and loss of power may occur from complete or partial loss of nervous influence over the bladder, as in those who are the subjects of paralytic states commencing in the spinal cord or brain. When the paralytic state follows an accident causing injury to a nervous centre, the nature of the case is obvious enough. But sometimes the onset and progress of chronic disease in these organs are very slow and insidious; the urinary troubles, as manifested by slowness or difficulty in passing urine, or by urine clouded through inability of the bladder to empty itself, may be the earliest signs of the nervous lesion. On the other hand, impaired gait, and other evidences of central mischief, may be (and mostly are) earlier phenomena, the derangement in the urinary function appearing at a later stage. For such patients, the habitual use of the catheter is often necessary (always of course when unable to empty the bladder by the natural efforts); while such constitutional treatment as is indicated by the cerebral or cerebro-spinal lesion present will comprehend that which the bladder demands. It is therefore unnecessary to allude further to that subject in this article. The same remark also applies to those few examples of **Tubercular disease** of the urinary organs, which is always a local expression of a general constitutional state sufficiently considered under its proper head in this work. All other treatment of this malady is local and surgical.

5. **Tumours.**—Tumours of the bladder have received much attention during the last few years, and the classification here offered is founded to a great extent on the writer's own experience, which has been considerable. Besides examining in consultation not a few cases, as well as taking the supervision of others in which, for varying reasons, no surgical procedure was admissible, he has operated for the removal of vesical tumour on upwards of forty patients. From these, from a study of the numerous preparations in the London museums, and from the growing experience of others, the following

varieties may be enumerated and classified, commencing with the most simple.

(1) *Mucous Polypus*.—Mucous polypus is not to be confounded with prostatic out-growths of the same form, these being inadmissible in any scheme comprising vesical tumours. It resembles polypus of the nasal cavity, but is more compact and solid in structure. Hitherto this product has been found in young children only.

(2) *Papillomatous Tumours*.—These appear in two forms. The essential character of each is that it closely resembles the natural papillæ occurring in certain parts of the digestive canal; the structure consisting of a prominence of simple membrane supported by a little connective tissue, but containing a large arterial twig as a central axis, the outer surface of the membrane being closely covered by a layer of cylindrical epithelium, or less commonly by an ovate or rounded variety. Formerly these growths were termed 'villous,' to which it was not unusual to add the word 'cancer,' although the implied allegation was entirely without warrant. When slender, long, and floating in the bladder, a papillomatous growth is termed 'fimbriated'; when more fibrous tissue is present and the growth is more solid, 'fibro-papilloma' appears to be the more appropriate term. These growths form very slowly at first, but when developed give rise to repeated attacks of hæmorrhage, which in time becomes continuous and copious, thus terminating life. They are the most commonly met with of all vesical growths, and their symptoms have been often mistaken for those of renal disease. Microscopic examination of the urine will sooner or later (especially if the bladder is washed out with water and the *débris* scrutinised) detect specimens of the growth which are quite characteristic. Moreover, on careful inquiry it may be ascertained that the bleeding is found occasionally, sometimes frequently, to appear in a manner which can rarely or never be met with in renal hæmorrhages. The patient commences an act of micturition with clear or almost bloodless urine, and at the end of the act, bright florid blood is mixed with the stream or appears alone at the close. This fact determined, the bleeding is without doubt vesical, and if little pain is complained of, as a rule the bleeding is certainly not due to a sarcomatous or carcinomatous growth, and is therefore probably a sign of papilloma.

The writer knows no styptics that beneficially affect hæmorrhage from this source, although he has seen all the known remedies abundantly tried. Preparations of iron, especially the perchloride, have the best claim perhaps to be employed. Treatment by local injections is apt to provoke rather than check the flow of blood.

(3) *Simple Myomatous Tumours*.—These are not common, and are not accompanied

by any characteristic signs of their presence. Nothing further remains to be said of them here in relation to diagnosis.

(4) *Tumours made up of Fibrous Tissue*. This kind of vesical tumour consists chiefly of fibrous tissue, more or less associated with small nuclei interspersed; sometimes the nuclei are in large quantity, a fact which leads to a graver estimate of their character.

Both these and the preceding kind of tumour involve the deep structures of the organ, and cannot be removed entirely by operative procedures; on the other hand, the simpler forms of papilloma, especially when growing from a pedicle, may be completely removed, and often do not return.

(5) *Epithelioma*.—The nature and characters of epithelioma of the bladder are the same as those of this growth in other parts of the body. This and the two following will be considered together.

(6) *Scirrhus*.—'Cancer,' properly so called, not occurring until after the middle term of life has been reached, is occasionally met with in the bladder.

(7) *Round- and Spindle-celled Sarcomata*. These were formerly known as 'encephaloid' disease. They are most commonly met with in children; although occasionally in the adult also.

DIAGNOSIS.—The diagnosis of malignant disease of the bladder is not difficult. Examination of the rectum furnishes valuable information. Scirrhus growth is very hard, irregular in form, and thus unlike any simple prostatic enlargement occurring in elderly men. The sarcomata are full and rounded in form, more elastic to the touch than 'prostatic enlargement,' and far more rapid in their rate of increase. Epithelioma, slow and restricted in its growth, may exist some years before presenting a tumour sufficiently dense to be recognised by palpation or sounding. In malignant disease pain is soon a more or less constant symptom, and some hæmorrhage is liable to appear, increasing at a later date; contrasting with papilloma, which latter is rarely recognisable by the rectum, commences with hæmorrhage, and is rarely painful. The progress of epithelioma is slow, and without any very distinctive characters. In all these three forms of recurring growth, microscopic observation may corroborate a diagnosis based on the symptoms, but cannot by itself establish one.

TREATMENT.—In relation to surgical treatment of the malignant diseases of the bladder, nothing can be said in behalf of operative measures for their removal. It is always impossible to extirpate the disease, which largely involves the organ itself, and rapidly reappears after the attempt has been made, although large masses may have been taken away, and an apparently almost empty cavity has been left as the result of the procedure.

The palliative treatment for all patients

thus affected consists in the avoidance of habits, occupations, and any amount of exercise found, by the experience of each individual case, to produce serious increase of hæmorrhage. The maintenance of the digestive power, and healthy activity of the functions which support life, are as far as possible to be ensured. Lastly, all means should be adopted, especially towards the closing stage of a case which is certainly destined to be fatal, to alleviate suffering, which is usually severe and prolonged, unless dealt with unflinchingly, as it ought to be, in such painful circumstances. Ample use should be made of morphine, usually most effective by subcutaneous injection, so that, although unable in any way to check the progress of the malady, we may at least mitigate the most terrible accompaniment of its distressing and inevitable course.

With almost all affections of the bladder, simple or malignant, ulcerative or associated with fistula, cystitis to some extent and in some form co-exists. It is this which gives rise to the presence of an undue quantity of mucus in the urine; it is often the source of pus, sometimes of blood in small quantity. Thus, in all the above-mentioned diseases, some degree of cystitis appears sooner or later.

HENRY THOMPSON.

**BLADDER-WORMS.**—A general term embracing various parasitic entozoa, having the character of cysts or vesicles. *See* ENTOZOA.

**BLANKENBERGHE, in Belgium.**  
A sea-bathing resort. *See* CLIMATE, Treatment of Disease by.

**BLEB.**—A large vesicle or bulla, containing, for the most part, a serous fluid, as in pemphigus, erysipelas, or burns and scalds. *See* BLISTER.

**BLÉNORRHŒA, BLÉNORRHŒA-GIA** (*βλέννα*, phlegm; and *ῥέω*, I flow. *βλέννα*, phlegm; and *ῥήγνυμι*, I burst out).—These terms are most correctly used to express excessive flow of mucus from any mucous surface. By means of an affix, the locality or nature of the discharge is expressed: e.g. *blenorrhœa oculi, nasalis, urethralis*. More commonly, however, and less accurately, blenorrhœa is employed as synonymous with gonorrhœa in the male or female.

**BLEPHARITIS** (*βλέφαρον*, an eyelid).—Inflammation of the eyelids. *See* EYE AND ITS APPENDAGES, Diseases of.

**BLEPHAROSPASM** (*βλέφαρον*, an eyelid; and *σπασμα*, a spasm).—Spasmodic movement or contraction of the eyelids. *See* EYE AND ITS APPENDAGES, Diseases of.

**BLINDNESS.**—Loss of sight. *See* AMAUROSIS; and VISION, Disorders of.

**BLISTER.**—SYNON.: Bleb; Blain; Fr. *Bulla*; *Bulle*; Ger. *Blase*.

**DEFINITION.**—A vesicle of the skin, caused by the separation of the horny cuticle from the rete mucosum by the transudation of serous lymph beneath the former.

**ÆTIOLOGY.**—Blisters may be idiopathic, as in pemphigus; or symptomatic, as in erysipelas. They are met with under the influence of any cause which depresses the vitality of the integument, as in some forms of prurigo, in chilblain, and in carbuncle; in scalds and burns; and as an effect of powerful irritants, such as cantharides or the aniline salts.

**DESCRIPTION.**—A blister ranges in size from that of a pea to a turkey's egg; it is more or less convex according to the amount of exudation; and conforms in colour with that of its contents, being sometimes yellow or amber-coloured and transparent, like serum, sometimes opalescent from the presence of pus, and sometimes red or purple from admixture with blood. The fluid of a blister, generally limpid and free, is sometimes held in the meshes of a delicate network, resulting from the stretching of the connecting cells of the rete mucosum and horny epidermis. This is peculiarly the case in blisters developed under the influence of acute inflammation, and especially in dermatitis anilina. Blisters may be dispersed, or aggregated, or even single, as in pemphigus or pompholyx solitarius.

**TREATMENT.**—Blisters when idiopathic are essentially asthenic in their nature, and call for corroborant therapeutical treatment. Locally they should be punctured so as to admit of the gradual escape of their contents, and then dusted over with some absorbent powder, such as oxide of zinc, fuller's earth, or cinchona. ERASMUS WILSON.

**BLISTERING.**—A therapeutic measure, which consists in the artificial production of blisters on the skin. *See* COUNTER-IRRITATION.

**BLOEMFONTEIN, in the Orange Free State.**—*See* AFRICA, SOUTH.

**BLOOD, Abstraction of.**—SYNON.: Bleeding; Blood-letting; Fr. *La Saignée*; Ger. *Der Aderlass*.

**DEFINITION.**—The withdrawal of blood from the body, either (a) from the general circulation, by arteriotomy or phlebotomy; or (b) locally, by leeches, scarifications, or wet-cupping.

It is to the first two of these methods of abstracting blood that the term 'bleeding,' or 'blood-letting,' has by common usage been restricted.

The topical abstraction of blood by means of leeches, scarifications, and cupping, though often valuable, is of secondary importance.

The present article will, therefore, chiefly be devoted to *general bleeding*.

**General Bleeding.**—This art, practised for centuries more or less universally, has of late years in this country fallen into disfavour. Much discussion has been raised as to the grounds for so great a difference. It has been attributed—(1) to the type of disease having undergone a change; (2) to mere fashion or caprice; (3) to a better knowledge of the nature of disease, teaching us that its processes were of a lowering or depressing character, which were to be overcome by the more general use of drugs which control the circulation, as well as of stimulants and support. It is probable that several causes have contributed to the undoubted change which has taken place. The year 1830 and subsequent years were marked by the epidemic visitation of cholera and of influenza. These diseases were characterised by extreme depression.<sup>1</sup> If antiphlogistic measures were adopted, they proved failures, and taught the physician that blood-letting was not the universal panacea it was supposed to be. By degrees it ceased to be practised as it used to be. A new generation which knew not the past has sprung up; and, as in all reactionary movements, the practice has become at length as limited as it formerly was universal. It is almost certain that in either extreme there is an evil, and that we may have recourse in certain cases to abstraction of blood with some degree of that success which formerly led to its extensive use, if not its abuse. It will be well to consider the subject at some little length, and under the following heads:—

1. The effects of moderate losses of blood on the healthy economy.

2. The value of bleeding as a remedy in disease, together with the indications for its employment in various affections.

3. The method of performing the operations of opening an artery—arteriotomy; and opening a vein—phlebotomy.

1. **EFFECTS.**—We have first, then, to consider the effects of moderate losses of blood upon the healthy economy. Upon this point we have abundant evidence, for the custom of regularly bleeding healthy people had reached such a point during the earlier part of this century, that in country districts it became a habit for adults to be bled as regularly as they went to market. No better testimony regarding the effects of this practice could be adduced than that of Sir James Paget, who, when referring to these customary venesections, says: 'I can regard those as a series of venesections fairly performed for the determination of what is the influence

of the removal of blood up to the point of syncope upon a comparatively healthy person. I think I can say surely that not one of these persons suffered harm.' To this might be added other and abundant testimony to the harmlessness of moderate venesection on the healthy economy.

2. **INDICATIONS AND USES.**—Concluding, then, that the abstraction of a limited quantity of blood has no deleterious effect upon the healthy organism, we will next describe the general indications for the use of bleeding in disease, and briefly refer to the various affections in which it may most suitably be employed.

Broadly stated, it may be said that bleeding is indicated when there is evidence of marked over-distension either of the arterial or of the venous system. In either case the result will be cardiac distension—in the former case of the left, and in the latter of the right chambers of the heart. In such conditions general bleeding restores the lost equilibrium of the vascular system, and relieves the heart and the other parts concerned in the circulation of the blood.

The arterial system may be in a state of augmented tension from two causes: (1) contraction of the arteries (the smaller vessels) themselves, with a diminished amount of blood in the arterial system; and (2) engorgement or distension of the arteries from spasm of the arterioles: both may be regarded as vaso-constrictive neuroses. In the first case there may be engorgement of the venous system and embarrassment of the right heart, calling for abstraction of blood by *venesection*; or visceral fluxion, the skin being pale: in the second case, relative emptiness of the veins with overfulness of the larger arteries, calling for blood-letting by *arteriotomy*.

In the former condition there would be, in bedside language, a *small* hard or wiry pulse, and in the latter a *full* and hard or bounding pulse. In the former the surface of the body may present one of the two following conditions: either the skin is injected and perhaps dusky, and this appears to be the case ordinarily; or it is pale and cool, the blood having receded inwards, chiefly to the abdominal viscera. The second condition obtains and is well seen in cases of uræmic asthma, when the arterial system is turgid almost to bursting, while the veins are comparatively empty. 'Hardness' of the pulse is usually said to be an indication for bleeding, and in certain associations it is so; but it is necessary to discriminate carefully between the 'hardness' due to 'tension' of the sound artery arising from (a) excessive contraction (the small, hard, wiry pulse), and (b) overfulness (the full, bounding pulse) on the one hand; and that due to arterial degeneration with more or less hard deposit in the walls of the vessels, on the other. In doubtful cases inquiry should be made into

<sup>1</sup> This state of depression was especially remarkable during the influenza epidemic of 1890, even in cases in which the ordinary symptoms of the disease were not severe; and too was the persistency of its effects.—EDITOR.

the state of the brachial artery at the bend of the elbow. This can readily be done by flexing the limb, when, if calcareous degeneration have taken place, the vessel will be thrown into serpentine folds, visible, except in fat people, to the eye, and cord-like and rigid to the touch. The temporal artery is a less safe guide, but neither it nor an arcus senilis should be overlooked in this connexion. A visible and tortuous pulse in a young person may indicate aortic regurgitant disease: the age of the patient must therefore be taken into consideration.

Dilatation of the arterioles would permit of the rapid passage of arterial blood into the veins: under such circumstances, therefore, we should expect the blood issuing from a cut vein to present a more florid appearance than under ordinary conditions. Bleeding here should be undertaken with considerable circumspection, and not be pushed very far, for collapse out of proportion to the amount of blood abstracted might ensue. On the other hand, when there is spasm of the arterioles, and the abstraction of blood is deemed advisable, it would be well to resort to arteriotomy rather than venesection. The best guide here would be the sphygmograph, but, as this instrument may not be always available, the full, hard, bounding pulse must be relied on when found in association with corroborative symptoms.

Whatever leads to over-engorgement of either side of the heart may render bleeding necessary. If the left side of the heart be over-full arteriotomy is indicated; if the right, venesection. The object of the withdrawal of blood from the general circulation is the direct relief of the overburdened heart. Whether the right or left chambers be taxed, the immediate effect is the same; they are over-distended, and cannot exercise full power upon their contents. It is with the embarrassed heart as with other hollow muscular organs—the bladder and uterus. Over-distension paralyses them by removing the ‘point d’appui’ essential for the initiation of muscular contraction; the energy may be there, but it cannot be exercised. It is obvious that the amount of blood which it is necessary to withdraw, in order to free the embarrassed organ, must vary considerably in different cases; but it may be safely laid down as a rule that it need rarely exceed a few ounces. Excess in this respect is the evil which formerly existed.

On this subject we may refer to the eminently practical remarks of the late Sir Thomas Watson, who says: ‘I hold it, then, to be certain, that for some special morbid conditions, which inflammation may or may not accompany, general blood-letting, and especially venesection, is a potent and life-preserving remedy; that there are many exigencies for which it is not only safe to employ, but unsafe and unpardonable to withhold it.’

He also gives the following judicious advice:—

‘Always it is necessary to consider the age, the sex, the general temperament and condition of the sick person, when we are turning over in our minds the expediency of abstracting blood. The very young, the old, the feeble, the cachectic, do not bear well the loss of much blood. This consideration is not to deter you from bleeding such persons topically when they are attacked by dangerous inflammation, but it especially enforces, in regard to them, the golden rule that no more blood should be abstracted than seems absolutely requisite to control the disease.’

The following are some of the affections in which blood-letting would seem to be more or less indicated:—

*Pneumonia.*—Blood-letting in pneumonia, as in many other inflammations, is most useful in the early stages. It is indicated in healthy patients suffering from uncomplicated acute sthenic pneumonia, if they happen to be seen early enough. It relieves pain, abates fever, and if it does not arrest the disease, it certainly appears to lessen its duration. It may also be called for when there is severe pain and evidence of cardiac embarrassment. It did good, and will still do good, in cases of pneumonia attended by embarrassment of the circulation, and that in truth is the indication for bleeding in this disease.

*Apoplexy.*—The same may be said with reference to cerebral apoplexy. The old belief in the importance of ‘letting blood’ in cases of apoplexy was, if possible, stronger than in cases of pneumonia. But here again more accurate clinical and more extended pathological knowledge has taught us to look upon ‘apoplexy’ very differently to our forefathers. Recognising the escape of blood from the bursting of a brittle artery as a common cause of ‘apoplectic fits,’ we see the futility of venesection when the ‘stroke’ or ‘fit’ is due to a lesion of this kind. Nor will bleeding unstop an artery when it is plugged by an embolus, or carry nutriment to the region thus bereft of vital fluid. On the other hand we have learnt to recognise the value of bleeding in another class of cases of so-called ‘apoplexy’—those which are unaccompanied by effusion of blood or lesion of the nervous tissue, but depend on rapidly occurring compression of the nervous centres from sudden or unequal increase in the volume of any portion of the cranial contents; or in certain cases of eclampsia from the circulation of blood poisoned by uneliminated urinary excreta. In cases of this description, when the right heart and venous system are engorged, phlebotomy acts beneficially: the stertor ceases, the purple face resumes its natural hue, the clouded intelligence becomes clear, and the impending danger is for the time averted. This has not cured the patient, however; it has only ‘obviated the tendency

to death:’ it has saved the patient’s life, though he may ultimately die of the disease which afflicts him. Where we meet with evidence of cerebral congestion, accompanied by fulness of the veins, a dusky countenance, and a *slow* full pulse, bleeding may most usefully be resorted to. In a word, although bleeding will not remove the effused blood in cases of cerebral hæmorrhage, it may sometimes be usefully employed to prevent further escape, when the heart is acting too forcibly; but it is altogether forbidden when that organ is enfeebled. The pulse at both wrists should be attentively examined before bleeding, in cases of cerebral hæmorrhage, for as a rule it is larger on the paralysed than on the sound side. See BRAIN, Hyperæmia of.

*Eclampsia*.—Indiscriminate bleeding in eclamptic seizures would be a grievous error. It would not relieve, but rather would probably intensify, convulsions of reflex origin, as in certain cases of puerperal convulsions. On the other hand, cases of puerperal convulsions accompanied by great turgescence of the vascular system, whether venous (as is commonly the case) or arterial, would be immensely benefited by the withdrawal of blood from the general current, either by venesection or by arteriotomy, according to the indication. This treatment may serve to stop the convulsions, and though that may be far from curing the disease, it may, nevertheless, be of the utmost value, for in the first place the fits themselves may kill by their violence or frequent repetition; whilst, secondly, time may be gained for the employment of other measures calculated to relieve the oppressed system, as, for example, purging by hydragogue cathartics, vapour baths, cupping the loins, &c. This gain may be immense; for bleeding may avert impending dissolution. Moreover, permanent good may ensue, inasmuch as bleeding reduces temperature, and in the eclampsia of pregnancy the temperature is usually high. In this it contrasts with pure uræmic convulsions, in which there is lowering of temperature.

*Venous Engorgement*.—Engorgement of the venous system arising from chronic disease, such as pulmonary emphysema or heart-disease, does not call for bleeding, unless the condition be acutely intensified by some intercurrent mischief, such as acute bronchitis; for, as the derangement is slowly produced, the organs and structures involved learn to accommodate themselves more or less to the altered conditions. It is only when vascular engorgement suddenly occurs in apparently healthy subjects, or, as just stated, there is acute intensification of a chronic condition, that bleeding is required. Thus, in cases where mediastinal tumours impede the return of blood from the head and upper portion of the body, the condition is ordinarily of such comparatively slow production, that bleeding is seldom urgently

called for; and, moreover, it would be of but small service, for the obstruction is irremediable.

The lividity of the face which accompanies all fits should not be confounded with the duskiness due to engorgement of the venous system generally.

*Uræmia*.—In pure uræmia bleeding is useful; the kidneys being especially in default. For venesection answers a double purpose, by not only relieving the engorged right heart and venous system, but by removing from the body a certain amount of poisoned blood—blood that is charged with urinary excreta. Doubtless, it does good in both ways, but the former is, *quoad* the fits, the more important because more immediate mode of its action. The second effect, that of ridding the body of damaged blood, is obviously available for us when we have to deal with uræmia, occurring in the non-pregnant; and when coma is deepening, the heart labouring, and the vascular system turgid, no remedy is so swift and sure as the lancet.

*Plethora*.—Blood-letting may be called for in cases of general plethora, whether sthenic or asthenic. In the former condition the vascular system generally seems to be overfull, though the excess is most marked in the arterial system. Arteriotomy, however, is seldom called for, though it might at first sight seem indicated, since venesection usually answers every purpose.

In asthenic plethora, on the other hand, the venous system only is overfilled; the right side of the heart is distended and its action is laboured. Here venesection is sometimes called for, but it should be carefully employed. It is seldom necessary to withdraw more than from 6 to 10 ounces of blood, and often a smaller quantity will suffice.

*Peritonitis*.—The relief obtained by bleeding in acute peritonitis rendered venesection a famous remedy in this affection in former times. And there can be no doubt about its efficacy in relieving the pain of peritonitis, as of inflammation of other serous membranes. It may be used when the patient is young and strong, and in that stage of the disease which is accompanied by a small, hard, and wiry pulse—the pulse of a contracted artery, of augmented tension from contraction, not from overfulness. It is worse than useless in the later stages of the disease, when adynamia has set in. But valuable as bleeding may be in certain cases of peritonitis, it rarely happens that it is admissible, for in the great majority of cases inflammation of the peritoneum is secondary to other diseases, such as tuberculosis or disease of the kidneys. Where peritonitis arises after delivery it is commonly of septicæmic origin, and it is seldom indeed that bleeding is of any avail under such conditions. So that, practically, bleeding is not a remedy which we can often employ in peritonitis. A very high temperature cannot

alone be held to be indicative of its use, for it so happens that such pyrexia is, as a rule, present only in septicæmic cases. Some cases of peritonitis, even fatal ones, run their course without any marked elevation of temperature: or indeed without showing any definite symptoms. Probably venesection is most serviceable in traumatic peritonitis, or it may be, when the inflammation is localised, though in that case leeches are more suitable.

*Pleurisy.*—When pleurisy attacks a young and robust patient, and is accompanied by severe pain, great relief follows blood-letting. The blood should be taken from a vein, and *pleno vivo*. But when, as so often happens, pleurisy is, like peritonitis, secondary to damaged blood-conditions — *e.g.* Bright's disease — bleeding is often inadmissible. When, as in pleuro-pneumonic cases, pleurisy complicates pneumonia, bleeding may possibly be called for, if there be great pain and oppression of the chest; but it should be most circumspectly used. Local blood-letting is much to be preferred if bleeding be thought necessary; for general bleeding is usually incompatible with the strength of the patient.

*Uræmic Asthma.*—In the affection known as uræmic asthma, in which there is spasm of the systemic arterioles, with intense turgidity of the arterial system and engorgement of the left heart, bleeding, in the form of arteriotomy, appears to be clearly indicated, though probably the use of nitrite of amyl or of nitroglycerine may render it less necessary.

*Spasmodic Bronchial Asthma.*—Pure asthma is doubtless often a nervous malady, and bleeding is not a remedy which should ordinarily be employed for its relief. It is only admissible when spasm of the pulmonary vessels, or obstruction to the flow of blood through them from bronchiole-spasm, leads to rapid engorgement of the right heart and venous system. This is the exact converse of uræmic asthma; but the effect in both is impediment to the aëration of the blood. Here the abstraction of a few ounces of blood from a vein may give immense and very prompt relief.

*Emphysema.*—Bleeding may be used in the so-called 'asthmatic attacks' to which emphysematous people are chronically prone. Here venesection may be imperatively demanded when an acute attack of bronchitis has, by adding to the already difficult passage of blood through the lungs, excited rapid engorgement of the dilated heart and previously turgid venous system.

*Puerperal Diseases.*—Formerly bleeding was much employed in midwifery practice, and especially in inflammatory affections after delivery. We now recognise that for the most part post-partum affections arise from septic mischief, and are of an adynamic type. It is but seldom that we are called upon to bleed in these cases. Nevertheless,

now and again, venesection may be called for.

*Disorders of Menstruation.*—Bleeding is a most valuable remedy in certain menstrual disorders, and especially in the plethora of women at the change of life. Many women suffer distressingly from general vascular disturbance at this period. The flushings, headaches, giddiness, feeling of oppression, and other vaso-motor phenomena from which they suffer, in association with the cessation of the catamenial flow, are immensely relieved by an occasional venesection. No other measure will so quickly and so effectually relieve these symptoms. Some robust, plethoric young women with amenorrhœa require periodical bleeding. One of the writers has known the abstraction of a few ounces of blood speedily followed by the occurrence of the menses, in cases of what may be called congestive amenorrhœa of many months' or even years' duration. It may be well to state in this connexion that in certain pulmonary, intracranial, and other visceral lesions, danger may arise from the augmented arterial tension which for a few days precedes the flow, and that the abstraction of blood may avert hæmorrhage into the damaged organs.

*After Operations.*—Bleeding is less frequently called for in surgical than in medical practice. It is indicated mainly in the after-treatment of cases which present engorgement of the right heart and venous system, as in some cases of ovariectomy and other operations involving the abdominal or thoracic cavities. A turgid venous system, with a small hard pulse, and a labouring heart—a condition which is often associated with scantiness, amounting almost to suppression, of urine—indicates blood-letting after operations of the kind above referred to.

*Shock.*—In certain cases of shock the abstraction of a limited amount of blood may be required. This point will be found discussed elsewhere. See SHOCK.

*Fever.*—That bleeding will lower the temperature of fevered patients has been known from the earliest times. It is, however, not a remedy to be resorted to for that purpose alone, for, as the most pronounced hyperpyrexia occurs chiefly in association with conditions leading to great depression, blood-letting is under such circumstances as useless as it is dangerous.

*Insolation.*—There are few disorders in which blood-letting is more successful, when rightly employed, than in sunstroke. It is seldom necessary to take more than a few ounces of blood; and excess should be particularly guarded against, for fear of subsequent collapse. The insensible patient with turgid veins, a tight pulse, and a labouring heart, will gain immense and prompt relief from venesection. The extreme pulmonary

congestion and over-distension of the right heart so often found *post mortem* might probably be prevented by the timely abstraction of a little blood from the venous system. Bleeding is not to be thought of in the syncopal form.

3. **METHODS OF BLEEDING.**—The following are the methods of performing the operations of arteriotomy and phlebotomy.

*Arteriotomy.*—This operation is best performed on the temporal artery. The vessel should be partially cut through by a simple transverse incision, and when a sufficient quantity of blood has been obtained it may then be completely severed, so that, retraction of both ends taking place, the hæmorrhage may be arrested. A compress of dry lint should then be applied, and a tight roller-bandage applied over it.

*Venesection.*—The median basilic vein is the one usually selected for the operation on account of its being most readily found. The brachial artery lies immediately beneath it, and care must be taken to avoid wounding the latter vessel. The median cephalic vein is preferable, but is not so easily found. A vein on the dorsum of the foot or other part of the body may be chosen, but, as a rule, it is not desirable to open the jugular vein, especially on account of the danger of the entrance of air, and other risks.

The steps in the operation on the arm are as follows:—First, the limb is to be firmly bound above the elbow by a broad tape or fillet. This should be applied with sufficient tightness to compress, and prevent the return of blood by, the veins, but not so as to intercept the current in the artery, and to extinguish the pulse. An oblique slit is then to be made in the vessel by means of a small lancet, care being taken not to cut too deeply. The spiring blood should be caught in a vessel and measured.

When sufficient blood has been withdrawn, the operator should firmly place a thumb or a finger on the aperture, and then, on removing compression, place upon the wound a dossil of dry lint, antiseptic dressing, styptic-colloid, or such-like, over which a roller-bandage should be twined a few times like a figure of 8, the cross being over the wound.

**Local Bleeding.**—The object of local bleeding is the relief of congested vessels, and especially those of inflamed parts. Arteries convey more blood to, and veins convey more away from, inflamed parts; so that local bleeding may give great relief and initiate resolution, since absorption does not fully commence until inflammation has ceased.

**METHODS OF LOCAL BLEEDING.**—Blood may be abstracted topically by leeches, by scarifications, or by wet-cupping.

1. *Leeching.*—An average leech will abstract nearly half an ounce of blood. Leeches

are extremely useful in a great variety of affections, since a pretty definite amount of blood can be withdrawn from the affected or adjacent parts, or from more distant parts, through intimacy of the vascular connexion, as in diseases of the eye and ear, and in hepatic diseases, accompanied by obstruction to the flow of blood through the portal system, when the application of leeches to the anus is most valuable.

Care should be taken not to apply leeches to parts over which sufficient compression cannot be made so as to control the bleeding, should any difficulty arise in arresting it otherwise. Thus, leeches should not be applied over the trachea, especially in children, in whom the error of applying them over the episternal notch is sometimes made. For the same reason the fontanelles should be avoided.

The skin of the part where the leeches are to be applied should first be washed; and when they do not bite readily, the part may be wetted with a little milk or sugar and milk. A slight prick of a needle, sufficient to draw a speck of blood, will often cause them to bite when refractory.

Should the bleeding continue too long after the leeches fall off, pressure or styptics may be applied. If it is desired to encourage bleeding, fomentations of hot water or linseed poultices are serviceable.

In applying leeches to the cervix uteri, the precaution of closing the os by a plug of wool should not be neglected. When they are applied within the mouth, a leech-glass should be used.

2. *Scarifying.*—Scarifications consist in small cuts of a depth not exceeding the eighth of an inch, or less, into the tissue whence it is desired to take blood. This mode of topical bleeding is mainly applied to the cervix uteri, to the tongue in acute glossitis, and to the palpebral conjunctiva in certain kinds of conjunctivitis; in the last case only slight incisions are permissible. Deeper punctures are made by some practitioners into the tissue of the cervix uteri, but these are punctures and not mere scarifications.

3. *Wet-Cupping.*—Cupping and the use of the scarificator, constituting wet-cupping, is an important method of topical blood-abstraction; and as a considerable amount of blood can thus be withdrawn, the general circulation may be affected. It is ordinarily employed, however, for its local effects.

The method of its performance is as follows:—Cupping-glasses being first put on for a brief time, as for dry-cupping (*see* CUPPING—*Dry-Cupping*), the operator applies to the part selected a spring scarificator so adjusted as to cut only to the required depth—about an eighth of an inch or less. The cupping-glasses are then re-applied, and the desired number of ounces of blood abstracted. If the glasses be too tightly attached the blood will

not flow readily, and unnecessary pain may be caused. After their removal adhesive plaster, or dry lint and a bandage, should be applied to the part.

Cupping is extremely useful over the loins in renal ischæmia; on the temple or behind the ears in certain cerebral disorders; down the spinal column in inflammation of the spinal cord or meninges; and on the chest in certain pulmonary and cardiac affections.

ALFRED WILTSHIRE. RICHARD QUAIN.

**BLOOD-DISEASE.**—The term *blood-disease* was used by the humoral pathologists as synonymous with *dyscrasis* or *anomalous crasis of the blood*, and expressed the idea that the blood was the seat 'almost without exception' of all general diseases. And, further, since purely local disease was considered to be exceptional, the vast majority of diseases were referred to dyscrases, and were classed under the head of blood-diseases.

The condition of the blood was considered by the humoralists to depend upon the *crasis*, that is the mixture, of its constituents; and prominent among its constituents were reckoned the *blastemata*, or germinal substances of the different tissues, which exuded through the capillary walls in the process of nutrition. When the blood-crisis was disordered or diseased, a dyscrasis was said to exist; and dyscrases were held to be in the majority of cases primary, though it was allowed that local anomalies of nutrition might and did occasionally occur, and give rise to secondary dyscrases. A blood-disease or dyscrasis being established, all morbid changes throughout the body were believed to be but local manifestations of the same. For the purpose, therefore, of a rational classification of disease, a previous classification had to be made of the dyscrases. The principal blood-crises were said to be:—

1. The *fibrin-crisis*; including the *simple fibrin-crisis*, the *croupous crasis*, and the *tubercle-crisis* as varieties. The local expression of the fibrin crises was inflammation in some form.

2. The *venous crasis*, in which fibrin was deficient. This included a vast number of special crises, lying at the foundation of the most diverse diseases—*e.g.* plethora, heart-disease, the acute exanthemata, rickets, albuminous urine, cholera, acute tuberculosis, lardaceous disease, cancer, acute convulsive diseases, metallic poisoning, &c.

3. The *serous crasis*; associated with anæmia.

4. The *putrid or septic crasis*.

5. *Anomalous* crises; such as those of syphilis, gout, &c.

The theory of dyscrases may be said to have declined since the appearance of Virchow's *Cellular Pathology*. Virchow showed that the blood is, in every relation, not an independent but a dependent fluid, and that

the sources from which it is sustained and restored, and the exciting causes of the changes that it may suffer, lie without it, not within it. Substances may enter the blood and affect the corpuscles injuriously; the blood may act as a medium in conveying to the organs noxious substances that have reached it from various sources; or its elements may be imperfectly restored. But never is any 'dyscrasis' permanent, unless new influences arise and act upon the blood through some channel or through some organ.

At the present time, whilst it cannot be said that humoralism is professed by many pathologists, the notion of blood-disease, as generally entertained forty years ago, still clings to our nomenclature, and pervades some of our pathological doctrines. Diseases that affect the whole economy—syphilis, tuberculosis, gout, and cancer—are frequently described as 'constitutional,' or 'blood-diseases,' and this whether their general manifestations are secondary to local disease, as in syphilis and cancer, or are referable to inheritance. While the *morbid conditions* of the blood are real and numerous, 'blood diseases,' so called, are but abstractions, and, as such, a fruitful source of confusion and useless discussion. It is desirable that the term *blood-disease* should be abandoned, and that the expression *morbid conditions of the blood* should be applied to those pathological states of the vital fluid which can be distinctly demonstrated by physical, chemical, or histological examination.

J. MITCHELL BRUCE.

#### **BLOOD, Morbid Conditions of.**—

The characters, composition, and functions of the blood in health are sufficiently familiar, and are fully described in text-books. But certain facts connected with the physiology of this fluid have a special bearing upon its pathology, and must be briefly considered before its morbid states can be profitably discussed.

**A. PHYSIOLOGY OF THE BLOOD.**—The *red corpuscles* of the blood consist of two portions—a colourless, sponge-like matrix; and a coloured substance of complex composition, which occupies the interstices of the former and accurately fills them. The matrix is regarded as possessing chiefly physical properties; whilst its contents constitute the active part of the corpuscle, and consist of hæmoglobin. The source of the red corpuscles is of the greatest pathological importance. In the embryo the blood and blood-vessels are developed from the same elements, and thus the two structures in their physiological aspect are essentially inseparable. In fully-developed blood the source of the red corpuscle is obscure; but there can be no reasonable doubt that it originates in the spleen and the medulla of bones; and that light is of the greatest importance in the

formation of hæmoglobin. With respect to the properties and function of the red corpuscle, it is to be noted that the ultimate elements of hæmoglobin are carbon, nitrogen, hydrogen, oxygen, sulphur, and iron—the last closely associated with the colouring matter. Hæmoglobin is soluble in water, forming a lake-coloured liquid from which fine crystals may be obtained, and which may be variously decomposed, giving rise to other ‘blood crystals.’ Most important of all its properties, hæmoglobin combines with certain gases to form definite chemical compounds: with O to form oxyhæmoglobin; with CO to form carbonic-oxide-hæmoglobin; and with NO to form nitric-oxide-hæmoglobin. These compounds are more or less unstable, especially the oxyhæmoglobin, which is broken up even under very feeble influences to oxygen and reduced hæmoglobin. Alternate oxygenation of hæmoglobin and deoxygenation of oxyhæmoglobin are constantly going on within the red corpuscles of the circulating blood; and the two changes, occurring in the pulmonary and systemic capillaries respectively, constitute the first great function of the blood—its oxygenating or respiratory function. The volume of oxygen in arterial blood is 20 per cent.; in venous blood 8 to 12 per cent. It must be clearly understood that disorders connected with the red corpuscles or respiratory elements of the body, whether in amount, composition, or circulation, directly affect the oxidation-processes only. Besides its origin and its function, there is a third relation of the red corpuscle to the organism—namely, the disposal of its products. These are eliminated by the ordinary channels; the salts, which are chiefly salts of potassium, being excreted by the kidneys, and the coloured material furnishing the pigments of the bile and urine.

The *white or colourless corpuscles* of the blood, also called leucocytes, are chiefly derived from the corpuscles of the lymph, and the cells of the lymphatic glands and allied organs, which they closely resemble. By escaping through the walls of the blood-vessels, they become identical with the wandering-cells of tissues and with pus-corpuscles,—from which they are indistinguishable except by locality. Some authorities regard them as *phagocytes*, attacking, and if possible destroying, pathological organisms in the blood and tissues. They may also influence the plasma by continually ingesting and egesting certain of its constituents. It might, therefore, be expected that morbid states of the leucocytes would be associated with disorders of the lymphatic structures, connective tissues, blood-plasma, and blood-vessels; and this will presently be shown to be the case. The proportion of white corpuscles in the blood is subject to physiological increase, without becoming excessive—*e.g.* after meals, during periods of growth and development,

and in menstruation and pregnancy. This state is called *physiological leucocytosis* (Virchow), and signifies lymph-glandular excitement. The chemical products of the leucocytes contain a large proportion of potassium and phosphoric acid.

The *blood-plates*, *-platelets*, or *-tablets* (*hæmatoblastes* of Hayem), minute bodies to be seen in blood, both within the vessels and after it has been drawn, are of obscure nature, origin, and function. Possibly they play some part in coagulation.

*Plasma*.—The physiological relations of the plasma to the organism are extremely complex; and disturbance of these relations furnishes many of the symptoms of disorder of the blood. Its mature function is essentially one of nutrition: it supplies the tissues with oxidisable material for development, growth, support, secretion, and the liberation of force. The source of the plasma is equally extensive. It derives its principal constituents from the alimentary canal through the absorbent glands and liver; while other important albuminous substances are being constantly supplied from the tissues generally, through the lymphatic system. Lastly, the products of the plasma, such as carbonic acid, urea, and water, are discharged by the regular excretory channels. Thus the condition of the plasma is found to be most intimately associated with that of the organs and tissues generally, whether as regards its origin, its mature function, or its products; and it is affected by disorder or disease of every organ, whether alimentary, sanguificient, dynamic, or excretory, and of every tissue.

*Coagulation of the Blood: Fibrin*.—Under certain circumstances, especially after removal from the body, the blood coagulates, and a proteid substance, fibrin, separates more or less completely from the other constituents. This change is now believed to be due to the interaction of two fibrin-factors contained in the plasma, namely, a ferment—fibrin-ferment, and fibrinogen; and along with these there must be present some neutral salt. The amount of fibrin produced varies not only with the amount of these bodies, but with the amount of salts, with the degree of alkalinity and of heat, and with other influences; and these variations are subject to no law at present known. The rapidity of the process depends upon (1) the amount of ferment; (2) its increased activity by moderate elevation of temperature; and (3) agitation of the blood and the increased number of points of contact (so-called ‘catalytic’ action)—by the presence of red corpuscles, hæmoglobin, charcoal, &c. It thus appears that the expressions ‘amount of fibrin’ and ‘rapidity of coagulation,’ however important as facts, do not afford any definite indication of the state of the blood, as was formerly believed. Three essential factors, and a large number of

accidental influences, share in the process; they may do so in very various proportions and degrees; they do not vary together; the amount of fibrin is not in proportion to any one of them; and after coagulation is complete, portions of all the factors probably remain uncombined. The part played by the red corpuscles in coagulation is a double one—(1) the corpuscles, 'as points of contact,' greatly increase the rapidity of coagulation; and (2) they supply oxygen, which appears to be indispensable to the process. The leucocytes probably produce the ferment; but it is doubtful whether it exists in the intact blood-cells. The blood-plates may also share in the process.

**B. PATHOLOGY OF THE BLOOD.**—The morbid states of the blood will now be considered in the following order:—1. Changes in quantity, and the effects of such changes upon the composition of the vital fluid. 2. Morbid conditions of the red corpuscles. 3. Changes in colour. 4. Melanæmia. 5. Morbid states of the white corpuscles. 6. The pathology of the blood-plasma; and of the process of coagulation. 7. The presence of foreign materials in the blood, including poisons and infective substances. 8. Organisms.

**1. Changes in Quantity of the Blood.** Alterations in the total amount of blood in the body are perhaps never simple, but always associated with alterations in quality.

(a) *Polyæmia*, or *excess* of blood in the body generally, may be the result of excessive ingestion of the elements of blood; of the accumulation of the same by the suppression of habitual hæmorrhages or fluxes; of the loss or obsolescence of a part of the body, such as a limb or a lung; or of insufficient exercise. It cannot be said, however, that polyæmia has ever been demonstrated by exact investigation, inasmuch as the total amount of blood in the body is still uncertain, and the physiological limits in this respect are very wide. Polyæmia is believed to be present in *plethora*, along with relative excess of the solids, particularly the red corpuscles.

(b) *Oligæmia*, or *deficiency* of the total amount of blood, is, on the contrary, an exceedingly frequent change, and constitutes the simplest form of anæmia. It is, however, probably never pure, inasmuch as alterations in quality appear to be inseparably associated with it; and the terms *hydræmia* and *spanæmia* have accordingly been used as synonymous with the preceding. The manner in which diminution in quantity gives rise to alteration in quality must be considered here.

When hæmorrhage occurs to any amount, and the whole quantity of blood in the vessels is reduced, the pressure falls, and absorption of the parenchymatous plasma rapidly follows, by which, along with vasomotor stimulation, the physical relations are

restored. If the loss of blood has been moderate, the only change in its composition may be considered to be *oligocythæmia*, or diminution of the red corpuscles, which alone of all the constituents of the blood cannot be rapidly restored. If the hæmorrhage has been more serious, the fluid absorbed into the circulation from the tissues, from the suppressed secretions, and from the alimentary canal, consists of water in ever-increasing excess, which carries with it an amount of salts equal to one-ninth the loss in albuminous substances. The morbid state of the blood is now beyond oligocythæmia; there is deficiency of albuminous constituents, or *hypalbuminosis*, and the condition correctly called *oligæmia vera* or *anæmia* is the result. The total quantity of blood probably remains for some time below the normal. A similar impairment of the quantity, and therewith of the quality of the blood, may be slowly developed by repeated small hæmorrhages, or by any cause whatever that impoverishes the blood, whether of the nature of waste or of want. The condition which results closely resembles that just described in the acute form—*oligæmia* with oligocythæmia and hypalbuminosis; the same is also known clinically as anæmia. See ANÆMIA.

As a therapeutic measure, oligæmia may be desirable. It may be induced either (1) by direct abstraction of blood, or (2) by gradual impoverishment of the blood, and reduction of the intra-vascular pressure. See BLOOD, Abstraction of; and AORTIC ANEURYSM; *Treatment*.

**2. Morbid Conditions of the Red Corpuscles.**—The pathology of the red corpuscles is still imperfectly understood. The following comprise the most important changes connected with them so far as they are known.

(a) *Polycythæmia: plethora polycythæmica*.—Increase in number of the red corpuscles is never considerable, being generally transitory and within physiological limits—for example, in the newly-born, and after meals. It has already been mentioned as associated with polyæmia in plethora. In the algid stage of cholera the red corpuscles are relatively in excess.

(b) *Oligocythæmia*.—Diminution in number of the red corpuscles is, on the other hand, of very frequent occurrence and great pathological importance. Microscopically the number of red corpuscles in a given visible area of blood is diminished; and chemically the amount of hæmoglobin in a given volume of blood may fall from fifteen even as low as five per cent. (see HÆMACTOMETER). The principal circumstances under which oligocythæmia occurs are—(1) in anæmia, or diminution in the amount of blood as a whole, from any cause, whether rapid or protracted, especially as the result of fever, the red corpuscles suffering early, seriously, and persistently,

as compared with the other constituents; (2) in leukæmia—the development of the red corpuscles being interrupted; (3) in hypalbuminosis, where the red corpuscles, like other elements, suffer from want of albuminous material; (4) in chlorosis, from imperfect growth; and (5) in pernicious anæmia, from excessive destruction (hæmolysis).

(c) *Oligochromæmia*.—Deficiency of the red corpuscles in hæmoglobin has been described by this name. It is a morbid condition of much interest, inasmuch as it is one of the essential alterations of the blood in chlorosis. When the individual red corpuscle contains less hæmoglobin than normal, it is said to present a pale appearance to the eye. A more trustworthy method of determining the richness of the red corpuscles in hæmoglobin, is by means of the hæmoglobinometer (see HÆMOGLOBINOMETER). Or we may compare the amount of hæmoglobin in a given weight of blood with the number of red corpuscles in a given microscopical area. When the former is small in proportion to the latter, the defect must lie in the individual corpuscle; and this may be so great that the proportion of hæmoglobin falls, as in some cases of chlorosis, to twenty-five per cent. of the normal. See CHLOROSIS.

(d) *Aglobulism*.—The effects of the two conditions of blood just described, namely, oligocythæmia and oligochromæmia, may be discussed together under the head of aglobulism, or deficiency of the blood in hæmoglobin. Want of this, the oxygenating substance of the organism, gives rise to symptoms at once extremely various amongst themselves, and of serious import. Every vital process, whether developmental, plastic, secretory, dynamic, or nutritive, is absolutely dependent on a free and immediate supply of oxygen. All of these processes, therefore, will suffer in aglobulism. The respiratory and circulatory movements are accelerated. The complex processes of alimentation and secretion are performed imperfectly, and the results are dyspepsia, constipation, and disordered sanguification—which intensify the abnormal blood-state. Muscular contraction is feeble, and cannot be sustained. Psychological force is weak; and dulness, sleepiness, pains, and other symptoms indicate imperfect oxidation within the nervous system. Bodily growth and development—of the sexual organs, for example—remain incomplete, and puberty is deferred. Nutrition everywhere suffers, the materials being insufficiently oxidised; and substances ‘intermediate’ to albumin on the one hand, and carbonic acid, water, and urea on the other hand, are formed, especially oils. Thus the organs and the connective tissues become loaded with fat and enlarged, instead of suffering atrophy, as they do when the blood-plasma is deficient. Finally the excretions are disturbed, and the subject of aglobulism presents derangement of the

colouring matters of the bile and urine, which are derived from hæmoglobin. See ANÆMIA, PERNICIOUS.

*Histological changes*.—Alterations in the size, outline, and consistence of the red corpuscle have been frequently recorded, but such accounts are incomplete, and no entirely successful attempt has yet been made to connect any of these changes with morbid processes in the tissues. In severe fevers, such as typhus, and in some rapid malignant diseases, the red corpuscles appear peculiarly soft, their outline being less resistant and sharp, and the bodies running together into irregular heaps, instead of into *rouleaux* with well-defined lines of contact between the elements. In another class of cases the corpuscles appear small and crenated or like the ‘thorn-apple.’ *Macrocythæmia* and *microcythæmia* have also been described as temporary and variable conditions, in which the red corpuscles are abnormally large and abnormally small respectively. These changes, as well as alterations in form, the corpuscles being oval, tailed, pear-shaped, or irregular (*poikilocytes*), or provided with excess of hæmoglobin, occur in pernicious anæmia (see fig. 3, page 61). *Transitional cells* between the white and the red corpuscle are unusually numerous in some cases of leukæmia.

3. **Changes in colour**.—The colour of the blood is chiefly due to the red corpuscles, and alterations from the normal in this direction will be best considered in this place, although the white corpuscles and the plasma may also affect the colour, as will be presently shown.

(a) The chief determining cause of the colour of the blood is the chemical condition of the hæmoglobin. When this is united with oxygen, in the arteries, the blood is *scarlet*; as deoxidation advances, this colour passes into a *purple*, and finally becomes *black* or *venous*. The dark colour is directly due to absence of oxygen. The purest example of this change is seen in asphyxia, where oxygen is excluded from the blood; but it also occurs as the result of the action of certain injurious influences upon the corpuscle itself, such as extreme heat, or poisoning by phosphorus, prussic acid, and other toxic agents. If the change proceed no farther, the scarlet colour may still be restored by oxidation. This blackness of the blood is generally associated with imperfect coagulation or even a state of fluidity. See Fibrin, page 164.

(b) *Paleness* of the blood is observed in oligæmia and oligocythæmia, and is due to deficiency of the hæmoglobin.

(c) The blood may not only be pale, but present streaks somewhat resembling pus, even as it flows from the living vessels, as in leukæmia. The same blood will settle on standing into three layers—of plasma superiorly, loosely coagulated or not; of white corpuscles

in the middle—a puriform layer; and of red corpuscles at the bottom.

(d) The *milky* appearance of chylous blood will be presently described under the head of *Blood-Plasma*.

(e) *Lake-coloured blood*.—A remarkable change of the blood, in which it becomes lake-coloured or transparent, is frequently observed as a more advanced stage of that just described under (a); but it may occur under other circumstances than deoxidation, and is of grave significance, inasmuch as it indicates complete and hopeless destruction of the red corpuscles. Lake-coloured blood is no longer opaque, but transparent; the hæmoglobin has left the corpuscles and is dissolved in the plasma. The change can be effected experimentally by the addition to blood of water, chloroform, the bile-acids, or other solvents; and it is probable that some of the cases of rapid death after enormous draughts of water, and the destruction of red corpuscles which is believed to occur in jaundice, may be accounted for in this way. But the most important cause of 'solution' of the red corpuscles is complete deoxidation of the hæmoglobin, which is followed by its diffusion in the plasma. Thus drawn blood is rendered lake-coloured by the addition of sulphide of ammonium, phosphorus, phosphoric acid, or iron-filings; and the same effect is produced by the intravenous injection of salts of the bile-acids. This being so, it might be expected that blood would assume the lake appearance when exposed to the prolonged action of the causes that render it black; and recent observations seem to indicate that such is the case. In a number of diseases which are attended with an accumulation of oxidisable substances in the circulation, the blood has been described as 'fluid,' 'claret'- or 'cherry-coloured,' 'clear,' and 'staining the tissues,'—but apparently without more exact observations on the colour of the living plasma. Such morbid states are high fevers (hyperpyrexia), insolation, and poisoning by malaria, phosphorus, and perhaps other agents. The effect of some of these influences is obviously to produce an excessive amount of oxidisable material in the blood, while the others may lead to the same result by reducing the oxygenating capacity of the corpuscles. Persons dying under such circumstances present great lividity, from the black or venous condition of their blood; death occurs with symptoms indicative of want of oxygen, as if so much of the hæmoglobin had been diffused through the plasma; and *post mortem* the vessels are found stained with the solution, the tissues are soaked with fluid lake-coloured blood, and decomposition is early and rapid. The 'fluidity' of such blood, or absence of clot in it, will be presently accounted for under *Fibrin*. That a similar solution or destruction of the red corpuscle may occur in all cases of fever,

only in a much less degree, is supported by several facts: (1) the increased discharge of potassium-salts in fever; (2) a similar increase of the colouring matter of the urine; and (3) the anæmia that is found at the termination of the process.

(f) Other alterations in colour may occur in the blood. The blood is *cherry-red* after poisoning by carbonic oxide, and remains so after exposure. It is *chocolate-coloured* after poisoning by the nitrites, such as nitrite of amyl, giving the spectrum of methæmoglobin—a more stable compound than hæmoglobin; and other hues have been recorded.

4. **Melanæmia**.—In relation with the pigment-bearing element of the blood there may be mentioned a morbid condition which has been described under the name of melanæmia. In this disease the blood presents black and brown pigment-particles and flakes, free or contained in cells of various shapes. These bodies arise from the disintegration of the red corpuscles; and are partly absorbed by the leucocytes, partly deposited in the spleen, liver, brain, and bone-marrow. Melanæmia is especially associated with two other states, namely, malaria, and an enlarged deeply-pigmented condition of the spleen; and one view of its origin is that the red corpuscles are destroyed in the spleen by the fever, and that the pigment-particles thus produced find their way from the spleen into the blood. They are then deposited in the liver and other organs, and give rise to symptoms of visceral disturbance during life, and to the peculiar slaty or grey discolouration that is found *post mortem*. It has been said that in melanotic cancer pigmented cells have been found in the living blood.

5. **Morbid Conditions of the White Corpuscles**.—The white corpuscles of the blood may undergo certain morbid changes both in number and appearance.

(a) The most remarkable of these is *increase* in number, which may rise from the normal (1 to 350 red) until the white corpuscles become even the more numerous. This condition is known as *leucocythæmia* or *leukæmia* (*sec* LEUKÆMIA). Short of this, however, the proportion of white corpuscles in the blood may be appreciably increased, and to this minor condition the name of *leucocytosis* has been applied. Leucocytosis, according to Virchow, accompanies, almost unexceptionally, every case of lymphatic excitement, such as inflammation, and tubercular, scrofulous, or cancerous enlargement or swelling of the glands and allied structures—Peyer's glands, the solitary follicles, the spleen, and the tonsils. Leucocytosis is distinguished from leukæmia by its very moderate degree; by its evanescent course; by the absence of deficiency of the red corpuscles; and by the accompanying symptoms. Leucocytosis may be appreciated even by the naked eye in the

clot of drawn blood, by the presence of an irregular 'lymphatic layer'—*crusta lymphatica*, consisting of collections of white corpuscles between the red clot and the buffy coat, which so frequently occurs along with it.

(b) A *diminution* in the number of white corpuscles occurs in chlorosis; and, it is said, in malaria, especially during the paroxysm of fever.

(c) With regard to the *structure* of the individual white corpuscles, the proportion of uninuclear or young cells, and the multinuclear or aged cells, may be disturbed both in leucocytosis and leukæmia; while corpuscles may be found containing granules of various kinds, especially pigment-particles, bacteria, micrococci, and other structures.

**6. Morbid Conditions of the Blood-plasma.**—(1) **WATER.**—The limits of the physiological variations in the amount of water in the blood are very wide.

(a) *Diminution* of water in the blood is observed in various degrees. It is moderate and transitory as the result of stimulation of the kidneys, skin, or bowels, for the normal proportion is speedily restored by absorption. This condition is also found after severe purgation, sweating, diarrhœa, or dysentery; and its production is the rationale of several of the methods adopted for the relief of dropsy. If the drain of water continue, or if the supply fail, the anhydric condition of blood increases, so that the fluid appears black, thick, and tarry. Such is the state of the blood in the algid stage of cholera; the specific gravity of the blood rising from 1055° as high as 1080°, with a comparative excess of salts, albumen, and urea. The chief symptoms of great deficiency of water in the blood are intense thirst; a shrivelled, shrunken aspect of the body generally; coldness and lividity of the extremities; muscular pains; and suppression of the excretions—phenomena directly referable to loss of water, retardation of the circulation, and interference with the function of the red corpuscles.

(b) *Excess.*—*Hydræmia.*—Reference has been already made under the head of oligæmia to the anæmia or hydræmia that follows it. Excess of water in the blood (*plethora serosa s. aquosa*) is perhaps never absolute, and the change may therefore be regarded with equal accuracy and greater convenience as deficiency of solids.

(2) **ALBUMINOUS CONSTITUENTS.**—On reviewing what has already been said under the head of Coagulation and Fibrin, the reader will observe that 'amount of fibrin,' and other expressions connected with the albuminous constituents, must be regarded at present as comparatively meaningless, in the light of our knowledge of the process of coagulation. Inasmuch, therefore, as little value can now be attached to the analyses of

fibrin that have been made in different diseases, it follows that the estimates of the albuminous substances left after coagulation—that is, of the albumins of the *serum*, must also be rejected. But the total amount of albumins in the blood may be easily ascertained; and this is subject to extensive variations. The balance between the albuminous substances which enter the blood from the alimentary tract and the lymphatic system, on the one hand, and the products of their transformation by the tissues, on the other hand, is represented by the albumins of the blood. These will increase accordingly when the supply is excessive, or the consumption small; and will decrease under the reverse circumstances.

(a) *Plethora hyperalbuminosa* is the name given to *excess* of albumins in the plasma. The amount has been found notably increased when the activity of the tissues is abnormally heightened, as, for example, in inflammatory diseases (acute rheumatism, tonsillitis, pneumonia, and pleurisy), and fibrinogen, which is the product of this increased activity, is poured abundantly into the blood. The amount of albuminous fluid produced in an inflamed part, whether it appear as a catarrh, an infiltration, an exudation, or an effusion, is very great, and may be enormous; and, under favourable circumstances, this and much that cannot be so easily appreciated are carried into the blood, the lymphatic structures swelling *en route*. Albuminous plethora, as a result of diminished oxidation, probably does not exist; for the effect of an insufficient supply of oxygen to the albumins (from want of exercise or over-feeding) is not the accumulation of these in the blood, but the formation of 'lower' products, such as uric acid and its allies, and the deposit of fat. Relative hyperalbuminosis is a necessary but transient effect of cholera and other severe watery fluxes.

(b) *Oligæmia hypalbuminosa*, or *deficiency* of albumins in the blood, occurs under exactly opposite circumstances from the preceding,—whether the ingestion of albumins from the alimentary tract and the tissues be comparatively small, or the consumption excessive. Inanition, therefore, on the one hand, and its multitude of causes, are associated with such poverty of blood; and so, on the other hand, are loss of blood, profuse discharges of albuminous fluids, morbid growths, and other sources of waste, as well as excessive demands of growth and development. The albumins of the plasma may fall under these circumstances from 80 to 87 parts in 1,000. Such hypalbuminosis is, however, never simple: the blood cannot be deficient in albuminous substances and otherwise normal, for, as we have already shown, loss of albumen is always followed by absorption of water and salts from the tissues in definite proportions, and anæmia is the result. The

red corpuscles suffer at the same time, for their nutrition speedily fails in hypalbuminosis, and aglobulinis ensues. Hypalbuminosis is a serious disorder of the blood. The relations of these conditions to each other and to oligæmia are even more complicated clinically than they are pathologically; and in this relation the whole of them are most conveniently discussed under the comprehensive head of anæmia. See ANÆMIA.

(3) **Clot; Fibrin.**—However uncertain as a measure of any particular constituent in the blood, the amount of clot or fibrin demands a brief notice as a matter of fact.

(a) *Abundant* clot has been considered as indicating an excess of fibrin in the blood or *hyperinosis*, the proportion being stated to rise as high as 1·0 instead of 0·2 per cent. Acute rheumatism, erysipelas, croupous pneumonia, and pleurisy are the diseases in which hyperinosis is most marked; but it also occurs in pregnancy. The two principal conditions of its occurrence appear to be—(1) increased activity of the tissues, including inflammation; and (2) free and abundant communication of these tissues with the blood through the lymphatic system.

(b) *Deficiency, looseness, or absence of clot; 'Fluid blood.'*—A small loose clot is frequently observed, as for example in typhoid states, or in chronic wasting diseases attended with loss of blood; and has been described as indicating *hypinosis* or deficiency of fibrin. When the condition is extreme, the clot may be absolutely wanting, as in certain cases of anæmia. The blood then separates on standing into three layers—an upper, consisting of clear liquid; a middle, puriform, of white corpuscles; and a lower, red, of red corpuscles. In another and larger group of cases, non-coagulating or fluid blood is at the same time of an intensely dark or even lake colour, and is commonly described as 'black.' The circumstances under which this condition of blood occurs, and the cause of the remarkable colour, have been already noticed; and it remains to account only for the fluidity. The profound alteration of the red corpuscles, the want of oxygen, the interference with the production of the ferment, and the changes in the fibrinogen—one and all combine to prevent coagulation.

(c) *Buffy coat.*—Another phenomenon connected with coagulation, from which erroneous and even dangerous conclusions have been drawn, is the so-called *buffy coat*. The process of coagulation is generally sufficiently slow to allow of the gravitation of some of the red corpuscles from the surface of the blood; and the corresponding part of the clot is accordingly paler. When the pale layer is unusually large it is known as the buffy coat or *crusta phlogistica*: it may be seen in the blood in pregnancy, inflammatory fevers, hydræmia, and oligocythæmia. When

these cases are analysed, it is found that the conditions favourable to the formation of the buffy coat are probably all more or less connected with the red corpuscles, namely—(1) increased specific gravity of the red corpuscles, as in oligocythæmia and hydræmia—allowing more rapid sinking; (2) interference with the catalytic action of the hæmoglobin, which is so powerful in determining the rapidity of coagulation, as in fevers and oligocythæmia; and (3) want of oxygen, corresponding to the amount and condition of the hæmoglobin, as in the same diseases. One and all of these states render the process of coagulation slow compared with the descent of the red corpuscles; and the buffy coat is the result. It thus appears that the buffy coat is no indication whatever of excess of fibrin-generators, or of the opposite; and that it is found under the most diverse conditions of blood.

(4) **Salts.**—The amount of positive knowledge concerning morbid alterations of the salts of the blood is but small. It is to be observed that the salts of the plasma have chiefly sodium for their base in the form of chlorides, whilst potassium salts and phosphates mostly reside in the corpuscles.

(a) *Diminution.*—In febrile diseases there is an increased discharge of compounds of both bases, but at different periods; the potassium salts appearing in excess in the excretions until the crisis is past, and the sodium salts during defervescence. At both periods, it may be considered certain that the blood is the chief source of the salts excreted; and that it is accordingly deficient in these constituents.

(b) *Excess.*—On the other hand, the salts of the plasma are relatively in excess in hypalbuminosis, replacing, in the proportion already stated, the lost albumins. The effect on the salts of the blood of such drains as occur in cholera has been variously stated; some authorities declaring that it is an increase, others a diminution.

(c) *Reaction.*—The alkalinity of the blood is said to be diminished in gout, cholera, and osteomalacia.

(5) **Fats.**—The normal increase of fats in the plasma that occurs after meals may be exaggerated by a diet rich in oil, and, it is said, in the subjects of diabetes, in chronic drunkards, and in persons disposed to obesity—*lipæmia*. When this increase is so great that the serum presents a milky appearance the blood has been called *chylous*. A cream-like scum forms on the surface of the serum; and the milky appearance is found microscopically to be due to the presence of fine granules and oil-globules. A marked increase of fatty matters in the blood has been said to occur in some cases of chyluria. Fat may also appear in the blood as a foreign body, by the escape of marrow into the circulation in fracture of bones—and that

in such quantity as to cause fatal capillary embolism. *See* EMBOLISM.

(6) **CARBONIC ACID GAS**, which exists in arterial blood in the proportion of 39 per cent., and in venous blood of 52 to 58 per cent., by volume, may accumulate within the circulation either by increased formation or by retention. Although associated with asphyxia, this increase of carbonic acid gas is probably not the chief cause either of the symptoms of that condition, or of the dark colour of the blood that accompanies it. *See* ASPHYXIA.

(7) **OTHER CONSTITUENTS**.—Amongst the most important of the other constituents of the blood, the following are to be noticed:—

(a) *Urea*, which exists in normal blood to the amount of 1·8 parts in 10,000, may increase in uræmia by two or three times. There is still much uncertainty, however, on this subject (*see* URÆMIA). Disease of the urinary organs, which interferes with the elimination of urea and allied products from the blood, is the usual cause of uræmia; but excessive tissue-change, as in fever and inordinate muscular exercise, has also the same effect.

(b) *Uric acid*, found in normal blood in minute traces, is increased (as quadrurate of soda) in all cases of gout, and may amount, according to Sir Alfred Garrod, even to 0·175 parts in 10,000. Its presence is easily demonstrated by the thread-experiment (*see* GOUT). Uric acid is also increased in leucæmia and chlorosis—perhaps from the imperfect oxidation associated with the condition of the red corpuscles.

(c) *Leucin*, *tyrosin*, *hippuric acid*, *sarcin*, and other allied complex compounds, have been frequently found in the blood in small quantities, and the same may be said of *oxalic* and *lactic acids*, and of *acetone*.

(d) *Bile*.—Certain of the constituents of the bile may occur in the plasma. The most obvious of these is the *bile-pigment*—*bilirubin*—which either by direct formation in the blood from the hæmoglobin, or more frequently by absorption from the liver, accumulates within the circulation, and gives rise to the colour of the tissues in jaundice. The *bile-acids*—*glycocholic* and *taurocholic acids*—are also, under certain circumstances, absorbed into the blood, where they may be detected with difficulty. They have a destructive effect upon the red corpuscles, and act further as a powerful poison to the tissues, possibly causing the severe symptoms which may occur in hepatogenous jaundice. *Cholesterin* is credited by some pathologists with the cause of the same symptoms, and it has been found in the blood in increased proportion in some cases of severe jaundice. *See* CHOLÆMIA; and JAUNDICE.

(e) *Sugar*.—The sugar of the blood is increased in diabetes, in some cases reaching 0·3 to 0·5 per cent.—*melitæmia*; also after chloroform-inhalation, and the administration

of amyl nitrite, chloral hydrate, and certain other drugs.

7. **Extraneous Matters in the Blood**. Besides its normal constituents and their products, the blood may occasionally contain certain matters entirely foreign to it, such as the numerous poisons which act either directly upon the corpuscles, or specifically upon the organism. These, entering the circulation before they exert their specific effect, are in many instances readily discovered by analysis. The acid compounds of hydrogen with sulphur, phosphorus, arsenic, and antimony, respectively, act as blood-poisons by depriving the oxyhæmoglobin of its oxygen; while carbonic oxide and nitric oxide unite with the hæmoglobin, and expel the oxygen from the corpuscles. It is a matter of speculation whether other so-called poisons, the nature of which is still obscure, do not enter the blood and there exert their primary effect, such as the contagium of acute specific fevers and other infectious disorders. Similar infective matters, produced in the tissues of the body itself, are believed to be absorbed in septicæmia, pyæmia, and other allied diseases, and numerous observations support the further belief that the presence of micro-organisms is intimately associated therewith. A somewhat similar infection may occur in malignant disease, the juices being mixed with the blood-plasma, but in some instances the process may be different, namely, by means of cells. We cannot expect to detect these cells readily in the blood *in transitu*. The same remark applies to embola, of whatever nature, and to blood-crystals.

8. **Organisms**.—The blood may contain a variety of vegetable and animal organisms, either foreign or peculiar to itself; the latter being called *hematozoa*. *See* HEMATOZOA; CHYLURIA; and FILARIA SANGUINIS-HOMINIS; also MICRO-ORGANISMS; MALARIA; RELAPSING FEVER; SPIRILLUM; and ZYME.

J. MITCHELL BRUCE.

**BLOOD, Transfusion of**.—*See* TRANSFUSION.

**BLOOD-WORMS**.—This term is of general application. It refers to all kinds of entozoa living in the blood. *See* ENTOZOA.

**BLUE DISEASE**.—SYNON.: *Morbus Cæruleus*.—A condition in which the most prominent symptom is a peculiar discolouration of the skin and mucous membranes, due to the circulation of dark blood in the vessels. *See* CYANOSIS; and HEART, Malformations of.

**BOILS**.—SYNON.: Furuncles; Fr. *Furoncles*; Ger. *Furunkeln*.

**DEFINITION**.—Gangrenous inflammation of the skin, forming small painful swellings, and ending by expulsion of the necrosed centre or 'core.' The inflammation begins in the glandular structure, hence involving not only the skin, but also the cellular tissue immediately

beneath. The sebaceous glands are most commonly the seat of boils, but occasionally the Meibomian glands (*stye*), the ceruminous glands, and the sweat-glands of the armpit are affected; or, more rarely still, the glands of the lips, vulva, or anus.

*Ætiology.*—*General predisposing causes.* The general predisposing causes of boils are:—the male sex; youth and after middle life; a stout habit of body; seasons of spring and autumn; a diet too full of flesh, or one suddenly changed, such as that adopted during training for rowing, or in various forms of the so-called 'water cure.' To these must be added the vitiation of the blood during exhausting fevers and in certain cases of saccharine urine, or that induced by handling dissecting-room material; and by dirty occupations—for example, chimney-sweeping or rag-picking. Lastly, boils are sometimes epidemic.

*Local predisposing causes.*—The parts of the skin most exposed to dirt or chafing—the hands and face, the neck and back, the buttocks and knees—are favourite sites for boils; but they may form on any part except the palms and soles. Blisters, poultices, and stimulating liniments occasionally cause them.

*Determining causes.*—Following these predisposing causes is the specific contagion of a parasitic microbe, which, by developing in a gland-cell, or hair-bulb, causes limited necrosis (the slough) of the tissue in which it grows. This microbe is the staphylococcus pyogenes, which can always be detected in the discharge of a boil; and boils have been produced by inoculation of this micrococcus, artificially cultivated from the tissues of acute necrosis of bones to free it from other possible contagia.

*SYMPTOMS.*—Boils appear either singly, in succession, or several at once, forming then an eruption on the skin. The solitary boil begins with itching; soon a reddish pimple forms at the itchy point; sometimes the pimple is tipped with a minute vesicle, in the centre of which a hair may generally be detected. The pimple grows larger and harder, the red area increases and grows darker, and pain begins, stinging at first, then throbbing. In about five days the summit breaks, pus oozes forth, the pain abates, and the hardness diminishes. A day or two later the core, a shred of sphacelated cellular tissue, escapes. The boil then subsides and healing rapidly takes place; the scar is depressed, and for some time has a violet colour. Occasionally the inflammation affects chiefly the cellular tissue beneath the skin; the mass is then softer, more round and clearly circumscribed, and fluctuates like an abscess—this variety forms in the armpit. Rarely, the central slough extends rapidly beneath the surface, and communicates with the surface by several small apertures (resembling carbuncle). In other cases the

swelling is more diffuse; no core appears at the surface, but a hard, very painful, pimple is formed, which is long in subsiding (*blind boil*). The furuncular eruption, consisting of groups of small boils, forms successive crops, and thus the disease may continue a long time. Boils are generally limited to a small region, but this is not always the case; and the greater part, even the whole, of the body may be attacked.

The constitutional disturbance is usually *nil* or slight when the boil is due to local irritation, though it may suffice to render nervous, irritable persons unfit for work. When the boils are in exhausted persons, the general symptoms are severe and denote great depression. Prostration, agitation, stupor, low delirium, dry brown tongue, sordes, vomiting, and diarrhoea set in, and the case often ends fatally: or recovery is very slow, accompanied by much suppuration. Septic absorption and pyæmia very rarely take place.

*Carbuncular Boil of the Face.*—There is a rare and generally fatal form of boil, the determining cause of which is still uncertain. Doubtless it is due to inoculation with a specific contagium not yet determined. It is met with only on the head and neck, notably on the lip. Mild and trifling at first, like an ordinary boil, it rapidly extends by inflammation of the cellular tissue and veins or lymphatics, and causes poisoning of the blood. The earliest sign of this fatal change is the occurrence of violent and repeated shivers. The boil becomes a boggy swelling of blackish-violet colour; the surrounding tissues become hard and brawny; suppuration ceases; sloughing occurs; the complexion grows earthy; the features, if the boil is on the face, become everted; the skin round the eyes in some cases is puffed out, and the eyes themselves project from the sockets; anxiety and laboured, gasping breathing set in; and a violent constricting pain in the head, chest, or belly is frequently experienced. Delirium and coma usually supervene, but sometimes consciousness and terrible suffering remain to the last. The duration from the first shiver to the end is about four days, though death may supervene in half that time. The veins of the face first inflame, and the phlebitis extends by the veins of the orbit to the sinuses in the skull, to the diploë, &c.; hence abscesses form in the eyelid, the forehead, the meninges, or the brain, and occasionally in distant viscera.

*DIAGNOSIS.*—A boil is distinguished by the central cavity and slough—characters peculiar to it. The boil of the face accompanied with phlebitis has been confounded in this country with the 'malignant pustule' of Continental surgeons. The former is still a *boil* with a central core. The '*pustule maligne*' is said to have invariably a large vesicle surrounding a brownish eschar, with

a ring of smaller vesicles round the larger one,—a condition never met with in boils.

**PROGNOSIS.**—When due to local causes, the prognosis is good, unless the patient be exhausted by old age or fever; under such circumstances the extensive sloughing and suppuration often lead to a fatal issue.

**TREATMENT.**—*General.*—First remove predisposing causes, and invigorate the patient by change of air, outdoor exercise, vapour and Turkish baths. The sanitary state of the patient's dwelling must be tested, as sewer gas may cause boils. The diet should be moderate and mixed. Alcohol, unless the patient is greatly debilitated, should be given in very moderate quantity, and the form of fermented liquor most habitual to the patient is best; much alcohol taken before the core has loosened increases the pain and throbbing. Occasionally a saline purge should be given. Of empirical remedies, yeast (a tablespoonful thrice daily) is said to put an end to the repetition of boils. Quinine and perchloride of iron are also used. Quinine should be given to an adult in five-grain doses every six hours, till singing in the ears and headache begin; it should then be gradually lowered for three or four days to three or four grains per diem, and finally left off. In obstinate cases the waters of Vichy, Barèges, or Harrogate are believed to remove the disposition to boils. In diabetes omission of sugar-forming food and the free administration of alkalis and opium are the most effectual remedies. For the exhausting boil of the face, large doses of brandy methodically prescribed, whether with or without quinine, are required, and copious nourishment by beef-tea, with peptonised foods in frequent doses.

*Local.*—When signalled by itching, a boil may be stopped by plucking out the hair of the inflamed follicle, and in a long succession many boils may thus be prevented. When the areola has formed, if the pain be slight a drop of caustic solution applied to the centre will sometimes check the progress of the boil. A better plan at this stage is to cover the boil with a galbanum and opium plaster (Erasmus Wilson's) spread on leather. Under this treatment pain at once ceases, the inflammation gradually subsides, and the separation of the core proceeds painlessly; when the boil discharges, a hole should be cut in the centre of the plaster, for the escape of the products. When the pain is stinging, and the areola wide, with restlessness and headache, warm poultices are most soothing—those of starch cause pustulation less than linseed-meal poultices. Mixing lard with a linseed poultice, or sprinkling it with the dilute solution of acetate of lead, has a similar effect. Extract of belladonna and glycerine in equal parts should be freely spread over the boil before the poultices are applied. Sponging with hot water also greatly allevi-

ates the pain of boils, and may be continued for hours together. Poultices hasten the expulsion of the slough, but should be discontinued as soon as the hardness changes to doughiness, as they tend to excite fresh crops of boils in the skin near the first boil. If the slough is large, the surface may be dressed with lint spread with Peruvian balsam or dusted with iodoform, and the boil carefully protected by means of pads and compresses of carbolised tow. Poultices are now altogether abandoned by many surgeons; moist heat being applied by means of layers of boracic lint wrung out of hot water and covered with oiled silk.

Incisions are now much less employed than formerly. They increase rather than lessen the loss of tissue in ordinary boils, and do not shorten the duration of the inflammation. They give relief to pain, however, and check the spread of diffused boils. Incisions are therefore useful to relieve tension when severe. When made, incisions should be free, crucial, or even star-like, and carried beyond the boil. In the rapidly extending boil of the face simple incisions are of little avail; the free use of the actual cautery may be beneficial if employed at an early period; or if the boil be seen early, free excision of the boil and the healthy tissue around it may be made. Otherwise, quinine in large doses and local soothing applications give most relief. *See* PUSTULE, MALIGNANT.

BERKELEY HILL.

**BONE, Diseases of.**—**SYNON.** : Fr. *Maladies des Os*; Ger. *Knochenkrankheiten*.—Under this head are included:—Acute and Chronic Inflammations of bone and its membranes, with the consequences thereof, such as Necrosis, Caries, and Abscess; New Growths, which arise both within and upon the bone; Malformations; and certain disorders of nutrition, namely, Hypertrophy and Atrophy.

Viewed in another aspect, bone diseases may be divided into two principal groups, which somewhat overlap one another, namely, (a) those which are distinctly *inflammatory* in type; and (b) those of *diathetic* origin.

Bone-tissue should be regarded as being similar to other connective tissues; but some diseases affecting it are rendered obscure by its structure, while others are materially modified in their progress, by reason mostly of its meshes being filled with lime-salts.

**1. Inflammation.**—As a matter of clinical convenience, it is usual to consider separately inflammations of the periosteum, of the bone proper, and of the medulla; but it should not be forgotten that these structures are throughout continuous and interdependent, and that disease is rarely exclusively confined to any one of them: it may originate or be chiefly developed in one, but it cannot long exist without involving the others to a greater or less degree. Whilst inflammation of bone is

essentially the same as inflammation elsewhere, its characters are modified by the tissue in which it takes place, accordingly as it affects the periosteum, the compact or the cancellated tissue. The bones are most frequently affected by disease during the period of active growth, when the development is proceeding and the blood-supply abundant. The periosteum is especially vascular at this period; and as the bone derives its chief supply of blood through it, it powerfully reacts when inflamed upon the bone, and inflammatory changes taking place in the periosteum are very seldom limited to this membrane. Inflammation of the bone is very painful, from the unyielding nature of the tissue; and, when the inflammation has been sufficiently intense, the vitality of the bone is destroyed, and the dead portion is subsequently separated with some difficulty. So also in bone-disease dependent upon some general cause, such as syphilis, the characters of the disease may be modified or obscured by the mechanical conditions which obtain.

**A. Periostitis.**—*Inflammation of the investing membrane of bone.*—By periosteum is usually meant the thin fibrous envelope of the bone in which the vessels for the supply of blood subdivide. But between it and the bone is a layer of osteogenetic cells like the cambium-layer of a growing plant, and immediately external is a layer of cellular tissue, continuous with that of the adjacent parts; these are integral portions of the periosteum, and take an active share in all its diseased processes. Periostitis may be either *acute* or *chronic*.

(a) *Acute Periostitis; Osteoperiostitis; Acute Periosteal Abscess; Acute Necrosis.* This is a formidable, but fortunately comparatively rare, disease, at least in the adult. It attacks the long bones almost exclusively, usually those of delicate children or young adults, in whom active bone-growth is still going on, and the periosteum is highly vascular. Acute periostitis probably never occurs without coincident inflammation of bone, and it is by far the most common cause of necrosis. It will be best to regard it as an acute osteitis and periostitis combined, and to call it *osteoperiostitis*, just as we call inflammation of the bone and of the endosteum *osteomyelitis*. The extent to which the bone and the periosteum are in the first instance respectively involved is always difficult, and sometimes impossible, to determine—it may be inferred from the extent of the necrosis. There are two ways in which the disease may begin—either in the fibrous investing sheath of the bone and the cellular layers beneath and superficial to it, from whence it spreads inwards to the cortical bone-substance, or even to the medulla; or in the bone-tissue—the inflammation spreading outwards to the periosteum. It is impossible in the living subject to dis-

tinguish acute osteomyelitis and osteoperiostitis arising from idiopathic causes. The disease is usually attributed to an injury, often slight, or to exposure to extremes of cold or heat. Frequently no cause is assignable. The disease is in most cases associated with the development of a pathogenic micro-organism, the pus teeming with micrococci.

**PATHOLOGY.**—Rapid exudation takes place in the layers of the periosteum, and between it and the bone; also in the Haversian spaces and canals of the bone, often to such an extent as to obstruct the circulation, and probably to cause by pressure the severe pain complained of at the outset. The exudation beneath the fibrous layer of periosteum is copious, and soon becomes purulent; the periosteum is detached; the vascular supply of the bone is cut off; and necrosis results. Occasionally the disease originates at the epiphyseal junction, commonly between the diaphysis and the epiphyseal cartilage, less frequently in the layer of cartilage itself. The extent of the necrosis depends upon the extent to which the periosteum is engaged, while the thickness of the dead bone depends mainly on the depth to which the inflammation in the osseous tissue extends, sometimes the entire shaft perishing from one extremity to the other. Usually the diseased action is arrested at the epiphyseal junction; but in other cases the extremity of the bone and the adjacent joint become involved. Large accumulations of pus, swarming with micrococci, are often rapidly formed in these cases, the pus escaping through openings in the fibrous envelope into the circumjacent cellular tissue, and finding its way to the surface. The shafts of the tibia and femur are the parts most frequently affected; the disease occurs more rarely in the bones of the upper extremities and other parts of the skeleton.

**SYMPTOMS.**—One of the earliest symptoms of acute periostitis is sudden and severe pain in the affected bone, which is soon followed by rigor and intense fever. The temperature mounts quickly to 104° or 105° F. There is sweating, rapid feeble pulse, and delirium, aggravated at night. On the second or third day deep-seated swelling may be felt, somewhat obscurely at first; and the limb is swollen, hot, tense, and tender. An obscure sense of fluctuation may be distinguished at an early period; but when fluctuation becomes distinct very extensive damage will usually have taken place. After an interval varying from three to five days, the inflammation approaches the surface, the skin becomes oedematous, pits on pressure, and finally reddens and inflames. The length of interval depends on the thickness of muscles and soft parts covering the affected bone. Other things being alike in respect of pain and amount of fever, the longer the delay in the appearance of external swelling, the greater the probability that the bone is the first and chief

tissue engaged, the inflammation having reached the periosteum secondarily; while the early appearance of swelling and fluctuation externally suggests that the inflammation is chiefly periosteal. Blood-poisoning, either septicæmic or pyæmic, is a common consequence of acute inflammation of bone and periosteum.

**DIAGNOSIS.**—This disease may be obscure at the commencement, and its nature overlooked. It has often been mistaken for acute rheumatism, on account of the swollen joints; for phlegmonous erysipelas; for acute cellulitis, or for typhoid fever. The only malady with which acute periostitis need be confounded is an idiopathic inflammation of the deep-seated cellular tissue in a limb. This disease is rare. When we observe the chain of symptoms above described in a young person, we may safely assume the presence of an acute osteoperiostitis.

**COURSE AND TERMINATIONS.**—The disease almost invariably terminates in suppuration and necrosis; resolution happens rarely, but necrosis is not inevitable, even after suppuration. In a few cases, especially in young children, if the matter be speedily evacuated, the abscess collapses, the periosteum reunites with the bone, and no necrosis takes place. This result is unfortunately quite exceptional. As the separation of the periosteum cuts off the vascular supply from the bone, unless incisions be very speedily made to permit the escape of inflammatory products, more or less extensive necrosis is the inevitable result. If not evacuated, the pus confined beneath the periosteum will presently escape through one or more openings into the intermuscular spaces, and in this way reach the surface. In favourable cases, after evacuation of the matter, the acute symptoms subside; new bone is formed from the deeper layers of the periosteum; and the dead part, finally invested with a sheath of new bone, becomes a sequestrum.

**PROGNOSIS.**—This must be founded on the extent of the necrosis; whether blood-poisoning has taken place; and whether the adjacent joints are implicated in the disease. Cure cannot take place until the dead bone is cast off or removed, and this is often long delayed. The usefulness of a limb may be permanently impaired by the disease, or it may require amputation, or the patient may lose his life from the exhaustion of the discharge or some intercurrent malady. On the other hand, the use of the limb, and the health of the patient, may become completely re-established.

**TREATMENT.**—Early and energetic treatment is of the greatest importance, as it affords the best prospect of averting the disastrous consequences of acute periostitis, but in hospital practice the cases are rarely seen sufficiently early. In the first stage the limb should be elevated, and cold compresses applied. As soon as the nature of the affec-

tion is suspected free incisions down to the bone, so as to divide the periosteum, are indicated, even before there is clear evidence that pus is formed. They relieve pain and tension; and, by permitting the timely escape of pus as soon as it does form, the amount of periosteal separation, and consequently of necrosis, is limited. It is the more important to make an early incision, because evidence of fluctuation is at first by no means easy to make out; and an incision down to the bone should be made in the centre of the inflamed area in all cases of doubt. Antiseptic precautions should always be taken. Sometimes the abscess-cavity does not readily collapse, owing to its walls being stiff and infiltrated, and its contents may become septic, thus greatly increasing the patient's risks. If there be synovial effusion into a neighbouring joint, it should be kept at rest by means of a splint or a fixed bandage. As the acute symptoms subside, the abscess-cavity contracts, one or more sinuses remain, and the dead bone begins to separate. *See* Necrosis, page 174.

Where an epiphysis is engaged in the disease the case is more urgent; the fever runs higher, the suppuration is greater, and the degree of joint-implication more intense, proceeding in extreme cases to suppurative inflammation and destruction of the articulation. When the joint becomes tense and swollen, the fluid may be drawn off with a trocar and cannula, or an incision may be made into it. The affected limb should always be supported on a splint. Œdema of the limb often indicates a deep-seated phlebitis, the precursor of septic poisoning. Under these circumstances, amputation is often the only resource. It is imperative to amputate where there is extensive bone-destruction, and the symptoms indicate commencing pyæmia; or where, with the death of a large portion of the shaft, one or both of the neighbouring joints may have become gravely implicated, and great suffering and loss of strength forbid us to temporise. It is precisely in these cases, however, where the diaphysis has become necrosed up to the epiphyseal junction, that good results are attainable by the immediate extraction of the dead bone. The part of the shaft which joins the epiphysis becomes rapidly detached and loose, and may be readily separated and removed, while the portion beyond the limit of the necrosis in the other direction can be divided with a chain saw. It is difficult, however, in the early stage to diagnose the extent of the necrosis. Where the joints both above and below are involved, amputation is usually necessary.

A periostitis of a very acute form, almost invariably suppurating, and accompanied by necrosis, is very common in the fingers, where it chiefly affects the unguis phalanges. The pain is very great, but may be relieved

by an early and free incision down to the bone, which, nevertheless, does not usually avert either suppuration or necrosis.

(b) *Chronic periostitis*.—This disease is usually due to some diathetic cause, but may result from injury, or from some continuous pressure or source of irritation. It is most frequent on the superficial parts of the skeleton, as the tibia, clavicle, skull, and ribs, but may affect any bone; and it is often observed at the origin or insertion of muscles. When the disease arises from a general cause, such as syphilis, many parts of the skeleton are affected; when from a local one, usually only one.

**SYMPTOMS.**—Chronic periostitis often takes the form of what is called a *node*—a tender, more or less painful, rounded or oval swelling; at first tense and hard, afterwards softer or even fluctuating. The pain is more severe at the outset, from the tension of the parts involved, and is generally worse at night. Subsequently the swelling becomes indolent, and painless, unless it be pressed upon; and the effused matter may become organised first into fibrous tissue and then into bone. Nodes are due to a localised inflammation. The cambium-layer of the periosteum and its external layer proliferate and become filled with leucocytes, thus forming a well-marked projection on the bone, which may undergo resolution, suppurate, or ossify, according to circumstances.

**PROGNOSIS.**—In chronic periostitis this is usually favourable. Under the influence of early and suitable treatment, the inflammatory products are completely absorbed, and the bone resumes its natural shape. If the chronic inflammation of the periosteum be permitted to proceed unchecked, a deposit of new osseous lamellæ usually takes place on the surface of the affected bone, giving rise to permanent thickenings, or even to osteophytic growths. These are composed of light porous bone, with a rough surface. The skeleton of a syphilitic subject will often present numerous thickenings of this nature. On making a section of the bone, it is easy to see that the new bone is superimposed upon the old, and is formed by the periosteum.

**TREATMENT.**—When due to a local cause, the swelling will often spontaneously subside with removal of the cause and rest to the part; but in obstinate cases iodide of potassium internally, and iodine ointment or blistering externally, may be required. If the subject be unhealthy, or if the original injury be considerable, suppuration may take place, when the treatment will be that of an inflammatory abscess. *Syphilitic nodes*, which are a very common expression of chronic periostitis, yield rapidly to the influence of iodide of potassium; and this in some cases may usefully be combined with a mercurial course. Blistering or friction externally is hurtful in

such cases. Syphilitic nodes are not at first prone to suppuration, and even when they become soft and fluctuating, and the skin reddens over them, they should not be mistaken for abscesses, as they do not require incision, and will readily be absorbed under suitable treatment.

(c) *Periostitis after typhoid fever*.—A peculiar form of chronic periostitis is occasionally observed as a sequel of typhoid fever. It occurs during convalescence, and without general symptoms. It takes the form of hot, painful, and tender nodes, frequently symmetrical, and often placed on the tibia; the disease is also found on the ribs and other bones. It may be associated with necrosis, but if so the extent of the dead bone is small in proportion to the inflamed area of periosteum. The general health is not seriously affected by the periostitis, and the disease is amenable to treatment by iodide of potassium, combined with iodide of iron.

(B) **Osteitis**.—Osteitis is an inflammation chiefly affecting the bone-substance. It may be either *acute* or *chronic*.

(a) *Acute osteitis* is neither clinically nor pathologically to be distinguished from acute osteomyelitis or endosteitis. See (C) Osteomyelitis, p. 171.

(b) *Chronic osteitis* is a disease beginning in the bone, in which from first to last the chief changes occur, the periosteum being secondarily engaged. This affection may result from injury, or be excited by exposure to cold; but it often depends on constitutional predisposition, such as the syphilitic, the strumous, the gouty, or the rheumatic diathesis, the first being the most frequent cause. It may occur in any part of the skeleton; the chief changes, when produced by syphilis, occur in the shafts of the long bones. They consist mainly of hypertrophy, and the bone is ultimately increased in thickness, in length, and generally in density: its interior is often transformed into sclerosed bone-tissue, and the medullary cavity is obliterated. Another form, associated with the strumous diathesis, is generally seated in the joint-ends of the long bones, and in the spongy bones. It is prone to end in suppuration, accompanied by either caries or necrosis, or may terminate in the condition known as *osteoporosis* or *rarefying osteitis*. The gouty and rheumatic forms are associated with evidence of the presence of either of these diatheses.

**PATHOLOGY.**—Increased vascularity first takes place, the Haversian canals enlarge, the canaliculi disappear, the cancelli enlarge to contain the inflammatory products, and the earthy matter diminishes. Hence the inflamed bone softens, and, if macerated at this stage, will be found comparatively light and porous. When the inflammation affects the superficial laminae of the bone, the periosteum becomes thick and vascular; if the

deeper parts are involved, similar changes will occur in the endosteum. The porous condition of the bone may become permanent, when the condition is called *osteoporosis*, the result of so-called rarefying osteitis; or the granulations become transformed into new bone, and the cancellated structure is filled with osseous deposit, so that the whole of the inflamed area becomes very dense, and is then said to be sclerosed; or the inflammatory process may terminate in suppuration, followed by caries or necrosis; or an abscess may form in the interior of the bone, and be either diffused or circumscribed.

**SYMPTOMS.**—These are insidious, very obscure at the outset, and may be mistaken for those of chronic rheumatism, or mere periostitis. They consist chiefly in aching, gnawing pain in the affected bone, with characteristic remissions and nocturnal exacerbations. The bone is tender on pressure, and feels increased in bulk at first, from the infiltration of the immediately surrounding soft tissues; subsequently the bone itself enlarges. There is often increase of heat in the limb. The progress is chronic. If unchecked by treatment, chronic osteitis may give rise to considerable deformity.

**TREATMENT.**—Treatment should be directed to the cause of the disease. If this be syphilis, antisyphilitic treatment will be followed by good results; even in chronic bone-inflammation not dependent on syphilis, iodide of potassium is often of great service. Local counter-irritation may also be employed. Often the cause cannot be made out, and if iodide of potassium fail in producing an effect we must fall back on general treatment. In the early subacute stage, rest, with elevation of the affected part, is very desirable. Warm fomentations, followed by iced compresses, relieve the suffering. If there be much pain and tension, leeches should be applied. Puncturing the tissues down to the inflamed bone, with a tenotomy-knife or fine bistoury, relieves the tense periosteum, and allows extravasation beneath it to escape, so that the pain promptly abates.

(c) *Osteitis deformans.*—A peculiar form of chronic inflammation of bone has been described by Sir James Paget under this title, from the changes it produces, both in the form and density of the affected bones. It is a chronic osteitis of the most extensive kind. The compact tissue is chiefly affected, becoming thickened, soft, spongy, and vascular; the Haversian canals are enlarged, and contain granulation material. It begins after middle age, and may continue for an indefinite time without influence upon the general health—a feature which distinguishes it clinically from other bone-inflammations. It is usually symmetrical, and affects chiefly the long bones of the lower extremity and the skull. At first the bones enlarge and soften, from excessive

production of imperfectly developed granulation-tissue and increased blood-supply; and, yielding to the weight of the body, they become curved and misshapen. The femur, for example, shows a marked anterior curvature, and its neck becomes horizontal; the spine becomes curved; the skull is enormously thickened. The limbs, however, although deformed, remain strong and fitted to support the body. In its early period, and sometimes throughout its course, the disease is attended with pains in the affected bones, which vary much in severity, and are not especially nocturnal or periodic. It is not attended by fever, nor associated with any constitutional disease. It differs from the chronic osteitis dependent on simple inflammation of bone, or that produced by gout or syphilis, in affecting the whole length of the bone; whilst hyperostosis and osteoporosis dependent on these latter causes rarely affect the entire bone. No treatment appears to produce any effect upon this disease.

(C) *Osteomyelitis.*—This is an inflammation chiefly affecting the endosteum and interior of the bone. Like osteitis, it may be either *acute* or *chronic*.

(a) *Acute osteomyelitis* or *endosteitis* is a suppurative inflammation of the medulla and bone, which is very frequently associated with septic poisoning and necrosis. We must remember that the medulla of a long bone is very vascular; it consists of fat and numerous white corpuscles contained in a delicate connective tissue. Near the cortical part of the bone the connective tissue assumes a very vascular membranous form, which has been called the endosteum. The disease nearly always arises in connexion with bone-injury, and most frequently happens after amputation, or gunshot fracture, in which the cancellated structure is injured; a severe contusion of the bone, an injury to the periosteum, or exposure to sudden extremes of heat and cold, is capable, under some circumstances, of producing the disease.

**SYMPTOMS.**—The symptoms are obscure, more especially if there be no opportunity of inspecting the affected bone, as the changes in the bone are often masked by inflammation of the superficial parts. They usually make their appearance from five to ten days after the injury to the bone; in some cases much earlier. The pain may not be excessive; there is fever and probably rigor. If there be a wound, the secretion from it diminishes in quantity, and becomes less healthy; the medulla protrudes from the central cavity; the parts soon become surrounded by putrescent fluid; and the symptoms resemble those of more or less intense septicæmia. The periosteum generally, but not always, separates from the bone. In young persons the disease is sometimes arrested at the epiphysis; but in the adult the whole length of the bone is liable to be

affected. The risk of septic poisoning is infinitely greater in osteomyelitis than in osteoperiostitis. Thrombosis of the bone-veins is especially prone to happen, and by the breaking down of the clot septic emboli are carried into the circulation, and deposited in the liver, lungs, and elsewhere. In this disease, too, fatty embolism often takes place—a condition associated with a very acute and fatal form of blood-poisoning. The prognosis is generally bad. It may be impossible at first to distinguish acute osteomyelitis from acute osteoperiostitis, arising from non-traumatic causes. In military hospitals, in war time, acute osteomyelitis is often epidemic, and probably of septic origin.

**TREATMENT.**—Where the symptoms lead us to suspect osteomyelitis, although the medullary cavity of the bone may not have been exposed, it will be desirable to trephine the bone, and if suppuration in its interior be discovered, it becomes necessary to amputate as soon as the nature of the disease is recognised, and this affords the best chance of saving the patient's life. The chief difficulty consists in arriving at a correct diagnosis, and deciding when it becomes necessary to interfere. This may best be done by observing the general progress of the case; and locally by the introduction of a probe into the medullary cavity when this is exposed. Should it reach healthy bleeding medulla near the surface we may temporise, if the constitutional symptoms admit of this; but it is rare for the disease once commenced to be limited—it has an extreme tendency to become diffused. Experience shows that nothing short of amputation at the next joint, or even above it, is sufficient to arrest the consequences of the malady; and to be successful this must be done before the systemic poisoning has become marked. Amputation in the continuity of the affected bone is worse than useless.

(b) *Chronic osteomyelitis* is an obscure affection, not to be distinguished, either clinically or pathologically, from chronic osteitis. It may terminate in sclerosis, or in the formation of an abscess. See *Chronic Osteitis*, p. 170.

2. **Abscess.**—This is a term usually applied to a limited suppuration in the bone, unattended by necrosis. Young adults are most prone to the disease, or boys about the age of puberty; it is very rare in women. It is the result of a chronic inflammation of bone, which may be associated with some injury. This affection is most frequently met with in the upper and lower extremities of the tibia, just external to the epiphysal cartilage, less frequently in the ends of the femur, only occasionally in other bones, and very seldom in the compact tissue anywhere.

**SYMPTOMS.**—A circumscribed, slightly elevated, very tender and painful swelling may be discovered on the surface of the bone.

This is due to a local periostitis with new bone-deposit. In old-standing cases the bone is often half an inch or an inch longer than its fellow, by reason of the chronic hyperæmia and consequent increase of activity of growth at the epiphysis. The skin and superficial parts are unchanged at first, or there may be but trifling subcutaneous œdema. There is often slight local increase of temperature. The pain, aggravated on deep pressure at the central point, is often intolerable. It is intermittent at first, but generally worse at night. After a time it becomes continuous, and deprives the patient of all rest, owing to its severity. The abscess may persist with little change for months or years. The symptoms generally resemble those of osteitis, from which at the outset it is difficult to distinguish this affection. When the abscess tends to reach the periosteal surface, the soft parts become engaged, and there will be slight redness and œdema of the skin. More rarely the pus makes its way into the adjacent articulation, in which it sets up destructive inflammation; but usually the joints are free from implication. The subjects of the disorder have often suffered from antecedent bone-disease. Evidence of this should be looked for, as giving a clue to the diagnosis.

**TREATMENT.**—Spontaneous cure cannot occur: even if the abscess discharge itself, a permanent fistula will usually remain. It is necessary to lay the abscess-cavity freely open. A crucial incision must be made through the soft parts, down to the bone, at the most tender and prominent point, and a disc of bone removed by the bone-trephine—an instrument without a shoulder, about half-an-inch in diameter. The sudden loss of resistance indicates the piercing of the abscess-cavity. The layer of granulation-tissue lining its interior should not be interfered with, but the cavity simply washed out. The pus is often foul, and greenish in colour. The wound should be dressed antiseptically; granulations presently fill it, which are subsequently transformed into a fibrous cicatrix. Immediate and permanent relief follows the operation. If the abscess is missed, the trephine may be re-applied, or drill punctures made in the most likely directions in the adjacent bone, in order to discover the pus. Sometimes an error of diagnosis is committed, and the symptoms are found to arise from chronic osteitis, without suppuration. The operation, nevertheless, affords relief in these cases also. Where there is doubt, a preliminary course of iodide of potassium will often resolve it.

3. **Caries.**—Caries is a form of chronic inflammation resulting in osteoporosis and suppuration, with gradual disintegration of bone, which has been likened to the process of ulceration in the soft tissues. It is generally found in the spongy bones, in any part of the

skeleton, the vertebræ and tarsus being the parts most commonly affected. There are two forms of the disease: the one, *simple caries*, resembling an indolent ulcer of the soft parts, is most common in the flat or short bones, but is sometimes met with in the compact tissue of long bones; the other, *fungating caries*, is often met with in the articular ends of the long bones, and usually terminates in joint-disorganisation. It has been called *subarticular caries*; and is part of the disorder known as *tumor albus*, a form of tuberculous disease (see JOINTS, Diseases of). The non-articular form of simple caries often originates in a localised periostitis, and is frequently due either to syphilis or struma—the latter is most frequent in young persons, the former in adults.

**PATHOLOGY.**—In caries the bone gradually disintegrates as the result of a chronic inflammation of its cancellated tissue. The trabeculæ become invaded with leucocytes; the Haversian canals, lacunæ, and canaliculi enlarge from the same cause; and granulations form, which prove, on an exposed surface, the source of purulent discharge, just in a granulating surface of the soft parts. The process is, however, interfered with and delayed by the act of getting rid of the osseous structure, between the trabeculæ of which the cells remain shut up until the bone-tissue finally breaks down, and comes away in the discharges, being often distinguished in the form of gritty particles. Until this process is completed the dead bone is soaked in pus, which often becomes putrid, and until it is got rid of a healthy granulation-surface is impossible. Caries is frequent in scrofulous subjects, and is then due to a deposit of tubercle in the cancellated tissue. Syphilis is a frequent cause of caries, the bone being infiltrated by gummatous material. Fungating caries occurs in the epiphyses of the long bones; the fungating granulation-tissue perforates the cartilages and invades the synovial membrane. The cancelli are rapidly filled up, and the granulations, on reaching the joint-surface, form fungating projections. Exposed to pressure, the softened bone-tissue yields and wastes away. In some joints, especially the shoulder, this may take place without the occurrence of suppuration—a condition known as ‘dry caries.’ More frequently purulent disintegration of the granulation-tissue occurs, and the disease presents the form of ordinary suppurating caries, or ulceration of bone. In other cases the inflammatory products undergo fatty degeneration and caseate, and portions of the bone necrose—‘necrotic caries,’ which occurs in connexion with both syphilis and tubercle. When recovery takes place, the granulation-tissue is in part absorbed, and partly transformed into fibrous material and new bone. A carious bone is soft and porous; the cancelli are thinned or destroyed, the

spaces enlarged, and portions of necrosed bone are often found in the midst—a condition also known as necrotic caries. On section, a pink, gelatinous granulation-material is seen, often caseating towards the centre. In dry caries the granulation-tissue is pink and gelatinous throughout. An abscess-cavity may form in the interior, with portions of necrosed cancellous bone in its midst. The cause of this chronic inflammation of bone is usually the deposit of tubercle in the cancellous tissue. Nodules of tubercle are to be found in the granulation-tissue which has invaded the bone, as well as in the surrounding soft parts, containing in their centre a giant-cell with branching processes, surrounded by lymphoid corpuscles. The spinal bones are most frequently affected; next, the expanded extremities of the long bones in the lower limb and the bones of the foot; lastly, those of the upper extremity.

**SYMPTOMS.**—Caries is very chronic in its progress, and often causes extensive damage to the bone, involving the destruction of a joint, or loss of a limb. It is almost always associated with an impaired condition of general health. The adjacent soft parts are involved in the inflammation; abscesses form in them, generally connected with the diseased bone; these burst or are opened; and sinuses lined with gelatiniform granulations, and discharging a thin pus, persist for an indefinite time. On examination with the probe the surface of the bone is felt bare, rough, and much softened; and outside the area of carious bone periosteal deposits of newly-formed osseous tissue are often found. The diagnosis and prognosis depend upon the age, constitutional condition, and clinical history of the patient, as much as on the local signs.

**TREATMENT.**—This must be directed to relieve the constitutional taint, as well as the local disease. Merely to excise or destroy the diseased portion of bone is not always sufficient to cure the patient. Local means prove efficient only when the general condition has been sufficiently ameliorated, especially in the unhealthy chronic inflammation of bone frequently called strumous. Fresh air, good food, and tonics are, therefore, of great importance. If syphilis be present, an anti-syphilitic treatment must be pursued. The principle by which the local means act is to facilitate the formation of a healthy granulating surface—to transform, in fact, an indolent into a healing ulcer. The disintegration of the dead and diseased trabeculæ must be assisted. For this purpose the application of strong sulphuric acid diluted with two or three parts of water, or some other mineral acid, often proves useful. Partial gouging out of the diseased bone seldom succeeds, because of the injury done by the instrument to the adjacent bone, weakened as it is by inflammatory changes, and therefore prone to set up fresh disease. The complete

*evidement* of the bone, leaving nothing but its thin outer shell, is more successful; but when the disease begins to invade adjacent joints, as in the tarsus, excision of the entire bone is best; or when several bones are involved, amputation of the whole part involved becomes necessary. In children operations of this kind are not so often required; general treatment frequently proves sufficient. In the early stages the actual cautery, applied over the most painful spot, is a valuable counter-irritant. When it is undesirable to make early incisions into strumous abscesses in connexion with diseased bone, it is right to empty them by a small trocar, so as to preserve the diseased area as long as possible from atmospheric influence, and meanwhile to improve the general health. When they require to be opened, this should be done under antiseptic precautions, and the whole of the diseased tissues must be scraped away with the sharp spoon. Iodoform dressing is the best. When the carious action is arrested, the cavity fills with healthy granulations, the sinuses close, the parts cicatrise, and the gap in the bone-tissue is filled by fibrous or sometimes by osseous material.

4. **Necrosis.**—The complete arrest of nutrition in a portion of bone from any cause is followed by the death or necrosis of the bone, and by a series of inflammatory changes in the adjacent parts, which result in the complete separation of the dead from the living tissue. It is a common result of the acute inflammation of bone which is invariably connected with some septic or infective cause.

**ÆTIOLOGY.**—Necrosis is most frequently the result of acute bone-inflammation or severe injury, as after amputation, compound fracture, or contusion. It is especially prone to happen in the compact tissue, but it also occurs in the spongy structure, as the joint-ends of the long bones, or the tarsus and carpus, where it is usually associated with more chronic forms of inflammation, and is more limited, as in necrotic caries. The peculiar nature of the blood-supply to bone, and the facility with which it may be interfered with or arrested under the pressure of inflammatory exudation, go far to explain the frequency of necrosis as a result of bone-inflammation. Acute suppurative osteo-periostitis or osteomyelitis rarely terminates without necrosis. Whether the dead bone will be in the superficial or the deep lamellæ, or include the whole thickness, depends on the seat of the inflammation, and on the extent to which the periosteum and endosteum are respectively implicated. The long-continued action of crude phosphorus, as observed in match-makers, and also of mercury, may induce necrosis. Syphilis is a frequent cause of necrosis, through its tendency to produce chronic osteo-periostitis, the sclerosed bone thus originated being after-

wards prone to necrose. It is not an uncommon sequel during convalescence from some eruptive and continued fevers. After scarlatina, osteo-periostitis, followed by necrosis, is by no means rare, although affections of the joints are more common. It is probable that many cases of necrosis occurring in childhood are connected with an antecedent attack of scarlet fever. The nasal bones may necrose as the result of severe coryza, the vertebræ after pharyngitis, or the petrous portion of the temporal bone as a consequence of otitis. Arterial thrombosis and embolism are occasional sequelæ of typhus, and may produce a local gangrene, not only of the soft parts, but of bone. This is, however, more frequent in connexion with typhoid fever. In endocarditis the nutrient artery of a bone has been observed to be obliterated by an embolus, thus producing necrosis.

**PATHOLOGY.**—After the death of a portion of bone, the living tissue, in immediate contact with the dead, becomes inflamed. The Haversian canals and canaliculi become distended with migratory cells; the leucocytes multiply, and by degrees consume the hard substance of the bone; the trabeculæ are absorbed or eaten away; loops of capillaries form from the pre-existing vessels: a granulating surface, in fact, is formed in the layer of living bone, surrounding or in contact with the dead, in a manner precisely similar to what takes place in the soft parts when a slough is being detached. The periosteum separates from the bone, and becomes thick and vascular, while the osseous surface beneath is smooth and white, like macerated bone. In cases of syphilitic necrosis, as well as in that resulting from phosphorus, the surface is rough from antecedent periosteal deposit, and in the former the interior may also be sclerosed. How the osseous trabeculæ are dissolved or disintegrated at the surface of separation, so as to loosen the dead bone, is not certain. Probably the granulation-tissue that forms from the living bone possesses ameboid properties, and thus disposes of the calcareous particles. The pus that is formed has a mechanical influence, while according to one theory lactic acid is produced, which transforms the insoluble into soluble salts of lime. While this loosening process is going on, new bone, formed chiefly from the periosteum, which becomes thick and vascular, is being deposited, constantly becoming thicker, and with one or more openings in it for the escape of pus, called *cloaca*, so that eventually the dead portion becomes completely invaginated, and is named, from its position, a *sequestrum*. This sequestration of the dead bone is not invariable. For instance, in the spongy bones, the bones of the skull, and the upper jaw, or where from any cause the periosteum has been destroyed, no sheath of new bone will be formed. Necrosis very

rarely takes place without suppuration ; when this does happen the nature of the case is very obscure. It has been aptly called 'quiet' necrosis. Occasionally nearly the whole shaft of a long bone has been found necrosed, and after an interval of months, or even years, no suppuration may have taken place. Such forms of necrosis are usually central and limited in extent. They may very closely simulate malignant disease, and often cannot be relieved or even recognised save after amputation. A chronic osteitis, followed by hypertrophy and sclerosis of the bone, is the most common antecedent condition of this form of necrosis.

**TREATMENT.**—The changes already described, which separate the dead bone from the living, do not cause its expulsion from the body. On the contrary, they shut it up, like a kernel within its shell, and nothing so imperatively demands surgical interference as the presence of necrosed bone. It acts as a foreign body, is a constant source of risk to the patient, and should be removed as soon as practicable. Its continued presence excites the periosteum to further formation of bone, so that the invaginating sheath becomes of great thickness in old-standing cases. The period at which an operation is usually undertaken is when the sequestrum has become loose, and the time required for this purpose varies with the extent of the necrosis. In the actively growing bones of the young, especially when the sequestrum involves the epiphysial junction, the process of separation is accomplished more quickly than in the adult. Roughly estimated, a period of from three to six months might be named as that within which loosening of the sequestrum usually occurs. Beyond the latter term an effort to extract the dead bone should not be delayed, even if it cannot be felt to be loose. Among other risks involved by delay may be that of amyloid degeneration of the viscera, principally the liver, kidneys, and spleen, which are subject to this change as the consequence of long-continued discharge from bone-disease. In order to remove a sequestrum, a director should first be introduced through a cloaca as a guide, and the soft parts sufficiently divided. An adequately large opening must now be made in the encasing sheath of new bone with the chisel, trephine, small saw, or cutting-forceps, and the dead bone extracted, either in one or several pieces, as may be the more convenient. The operation may prove difficult on account of great thickness of the soft parts or of the sequestral envelope, or because the sequestrum itself is extensive. After the removal of the dead bone the cavity fills with granulations, which subsequently ossify, and the soft parts cicatrise. When the loss of bone-substance is very large the reparative process has been much facilitated by the implantation of small pieces of freshly

killed rabbit's bone. Finally the sequestral envelope of new bone is partly absorbed, partly consolidated, just as the redundant callus is after fracture, and the bone tends more or less to resume its normal size and shape.

**5. Tubercle.**—An examination of some cases of chronic bone-disease in scrofulous subjects seems to prove their connexion with the formation of tubercle in the bone. The cancellated tissue in the joint-ends of the long bones, and cancellated bone generally, are chiefly affected. Miliary tubercles, precisely similar to those found in other parts, are formed in the soft structure of the medulla. These may become confluent, and tend to caseate or undergo a fatty degeneration. The caseous mass only differs from a similarly produced mass elsewhere by reason of the necrosed bony framework which retains it. When macerated, the bone shows a rounded cavity, from which the tubercle has been discharged. The external appearances are those of fungating caries ; but microscopical examination discloses multitudes of round cells like lymph-corpuscles, with protoplasmic matter, filling up the interspaces, and enclosing one or two giant-cells. The cells are found surrounding the soft, grey, non-vascular patches, which are often seen on section of an inflamed cancellated bone in strumous individuals, and this central part may be the subject of calcareous, fatty, or suppurative changes. The bone when so affected undergoes osteoporotic changes, and is never sclerosed ; hence these are not simply cases of chronic inflammation.

**TREATMENT.**—In cases of this kind general tonic treatment becomes of the greatest importance. Rest must be given to the affected part, and exercise to the body generally, combined with fresh air both day and night, and simple nourishing food. When the bone is extensively diseased, it must either be excised, or the part amputated. The presence of the tuberculous diathesis does not forbid an operation, the local source of irritation and drain upon the system being thus removed, and a healthy traumatic surface substituted for one infiltrated with inflammatory products. The removal of the local disorder often proves a comfort to the patient ; increases his chance of regaining health and strength ; and diminishes the liability to dissemination.

**6. New Growths.**—The bones are liable to be the seat of most of the tumours found elsewhere in the body. The most important, and the most frequently occurring, are the sarcomata.

(a) **Myeloid Sarcoma.**—Some forms are peculiar to bone, as, for instance, the myeloid sarcoma, so called from the many large nucleated corpuscles contained in it, analogous to those found in foetal marrow ; it is of endosteal origin, causing an expansion of the

bone in which it grows. It is most common in the inferior maxillary bone, and near the epiphysal ends of the long bones. It is generally observed in young persons; requires removal; and extirpation, if complete, is not, as a rule, followed by a return of the disease.

(b) **Periosteal or Fasciculated Sarcoma.**—This tumour, springing from the periosteum of a long bone, such as the femur, is not uncommon. The shaft of the bone may be seen on section passing through the centre of the tumour. Numerous bands of fibrous tissue, often ossified, radiate from the periosteum through the growth, like an outspread fan. The cells are spindle-shaped or round in form, of various sizes and in different proportions. This form of sarcoma is malignant. The best treatment is amputation of the limb at the joint above, which does not, however, ensure against recurrence of the disease.

**Carcinoma.**—This does not occur as a primary growth in bones.

(c) **Exostosis.**—This is a bony outgrowth developed on any part of the skeleton. It is difficult to distinguish cartilaginous from osseous outgrowths. The two structures are often mixed, and a tumour originally cartilaginous is often transformed into bone. Cartilaginous outgrowths, called **Ecchondroses**, are met with on the costal cartilages of old persons, also on the intervertebral discs, near the synchondroses, and arise also from the articular cartilages in osteoarthritis. Cartilaginous tumours, growing either from the periosteum or the medulla, have their favourite seat upon the phalanges; they are usually multiple, and from the deformity and inconvenience they produce often demand either enucleation of the tumour, or, in extreme cases, amputation of the finger. Chondromata also arise from the scapula and the shafts of the long bones, when they often become mixed with sarcomatous tissue. The more special forms of exostosis are of two kinds, the *spongy* and the *ivory-like*. *Spongy exostosis* is often developed near the articular ends of the long bones, where it forms a nodulated outgrowth of cancellated bone of variable size, encrusted with a thin layer of cartilage, by means of which it grows, and having generally a bursa superimposed. This kind of exostosis is often met with near an epiphysal cartilage, and ceases to grow when the bone is fully developed. This fact, as well as the proximity of the neighbouring joint, renders surgical interference often unnecessary, and it may be hazardous. Another form of spongy exostosis, sometimes called *osteophyte*, depends on a local excessive periosteal growth of bone. At first this outgrowth is porous, and but slightly connected with the bone on which it is developed. Afterwards it may become dense and hard from interstitial deposit, or it may always remain spongy. Such exostoses often depend

on some local exciting cause, such as a blow; or they may be found at the insertion or origin of a muscle, as in the so-called rider's bone, at the origin of the adductor longus muscle, or the exostosis frequently found at the insertion of the adductor magnus, or the 'exercise bone' of the German soldier. They may be regarded as morbid exaggerations of the normal tuberosities of the skeleton. *Ivory exostosis*, so called from its dense, eburnated character, is more rare. It is found on the flat bones, and especially the cranium. It varies much in size, and may be pedunculated or sessile. Hereditary influence appears to exist in some cases, in others a predisposition to chronic periostitis, but there may be no apparent cause. The development is slow and painless.

**TREATMENT.**—Interference is seldom required in the spongy exostoses, except on account of pain or loss of function. When pedunculated, they can be broken off or divided subcutaneously; and although they may reunite, it will probably be in a more convenient and painless relation to adjacent parts. Otherwise they should be excised.

Except on account of deformity, or of pressing on important structures, an ivory exostosis should not be meddled with. It can often, however, when necessary, be enucleated; or where only a partial removal is possible, the low vitality of the tumour often leads to necrosis and subsequent exfoliation of the remainder. Spontaneous necrosis also occasionally occurs.

(d) **Osteo-aneurysm.**—Certain sarcomata and myeloid tumours, when very vascular, pulsate, and have been mistaken for aneurysm. There are, however, undoubted cases of aneurysmal tumours dilating the bone, which have been cured by ligature of the main vessel of the limb. When the tumour is small it may be excised, or the actual cautery applied. Sometimes amputation is required. The causes and pathology of the disease are obscure. It is probably in some cases of a nævoid nature.

(e) **Bone-cysts.**—These are tumours distending and thinning the bone, and filled with serum or bloody fluid. In some rare cases they contain hydatids. The origin of bone-cysts is obscure; some originate in the dentigerous cavities of the maxilla, in which bone-cysts are most frequent, but they are sometimes found elsewhere. A very slow, painless increase in size takes place. The bone becomes gradually very thin, and often affords on pressure a peculiar and characteristic parchment-like crackling. In obscure cases an exploratory puncture should be made.

**TREATMENT.**—This consists in freely laying open the cyst-cavity, and providing for subsequent drainage. The cavity gradually contracts and becomes obliterated.

(f) **Hydatids.**—The formation of echinococcus-cysts is exceedingly rare in bone,

compared with other parts of the body. The causes are unknown, and the symptoms very obscure, resembling those of an ordinary cyst. A cavity is formed, usually in the spongy extremities of the long bones, to contain the mother cyst. But it is also found in the medullary canal. The affection is grave. Serious inflammation often follows interference with these entozoa. It is sometimes difficult to remove the whole disease; and unless this be effectually done, a relapse will occur; while in such parts as the pelvis art is unavailing. The cavity should, if possible, be freely laid open, and all the cysts carefully removed or destroyed. The actual cautery may be sometimes employed with advantage, or the surface of the adjacent bone removed, as it may be invaded by the cysts. An exploratory puncture can alone resolve the diagnosis, by finding the hooks of the acephalocyst in the fluid.

**g. Actinomycosis.**—This disease arises from the transmission to man of a vegetable parasite formed in the calf (*see* ACTINOMYCOSIS). Its development in bone mainly occurs in the jaws (generally the lower), in which it causes great swelling. The jaw is distended by a mass of material chiefly inflammatory—consisting of a fibrous basis which is riddled with tracts occupied by the fungus. When suppuration occurs, the abscess-cavity presents the appearance of meshwork. The mode of infection may be either through the respiratory or digestive tracts, or by the socket of a diseased tooth.

**7. Malformations.**—These consist in any departure from the normal type of the skeleton, by reason of excess, deficiency, or irregularity, either congenital or acquired. It is not necessary to more than allude to the facts that the skeleton is often defective in parts; that senile changes occur, especially in certain bones; and that supplementary bones and processes are met with. Various deformities occur in bones from fractures, both intra-uterine and subsequent to birth, and from curvatures due to rickets or softening. Treatment of curvature consists in gradual straightening by splints or other apparatus, or immediate straightening under chloroform, methods which, in the soft growing bones of the young, prove successful in abating many deformities. The curvatures of adult bones do not yield in this way. When there is loss or impairment of function from deformity, the bone may be safely divided subcutaneously with the chisel or saw, and the limb straightened—often with admirable results. Forcible fracture is a clumsy and somewhat dangerous method, as the force employed cannot be regulated.

**8. Hypertrophy.**—Hypertrophy means an excessive growth of bone-tissue, increasing the bulk of the bone, the added bone completely reproducing the normal structure. Apart from inflammation hypertrophy is rare;

but in museums specimens of excessive growth are met with, especially of the bones of the face and skull. The causes of this rare condition are unknown, and no treatment appears available. *See* ACROMEGALY.

**9. Atrophy.**—This disease consists in a diminution of the size or compactness of a bone. It may be the result of inflammatory changes, of senile degeneration, of disuse of a limb, of continuous pressure, or of an injury—such as a fracture—followed by non-union. The bone-tissue gradually wastes away, the cortical portion often becoming a thin parchment-like layer of bone, filled with soft medulla. This has been called *excentric atrophy*. The external appearance and size of the bone remain unchanged. *Concentric atrophy*, where the size of the bone diminishes in all its dimensions, may occur in bones which have been disused for lengthened periods, as from paralysis of a limb, disease of a joint, the bone in a stump, or un-united fracture. *Spontaneous fractures*, or fractures due to trifling causes, are very common under these circumstances. The term *fragilitas ossium* has been applied to this condition of bone-tissue, which also frequently occurs in cases of cancerous cachexia.

**10. Softening.**—This change occurs in rickets and mollities ossium. *See* RICKETS; and MOLLITIES OSSIIUM.

WILLIAM MAC CORMAC.

**BORBORYGMI** (*βοβορύζω*, I grumble).—Rumbling sounds produced in the abdomen by the movements of gas within the bowels or stomach. *See* FLATULENCE.

**BORDIGHERA**, in Italy, on the Riviera.—A suitable winter residence for patients suffering from some forms of chest-disease. The climate is warm and dry. *See* CLIMATE, Treatment of Disease by.

**BORMIO**, in Italy.—Thermal waters. *See* MINERAL WATERS.

**BOTHRIOCEPHALUS** (*βόθριον*, a pit; and *κεφαλή*, the head).—A genus of cestode entozoa, characterised by the possession of two pits or depressions, one on either side of the head, in place of the four sucking discs usually present in tape-worms. *See* ENTOZOA.

**BOTS.**—A term employed to designate the larvæ of certain dipterous insects called gadflies. They infest man more rarely than animals. *See* ENTOZOA.

**BOULIMIA.**—*See* BULIMIA.

**BOURBONNE - LES - BAINS**, in France.—Common salt waters. *See* MINERAL WATERS.

**BOURBOULE, LA**, in France.—Thermal alkaline and arsenical waters. *See* MINERAL WATERS.

**BOURNEMOUTH, in Hampshire.**—Regarded as a suitable winter residence for patients suffering from certain forms of chest-disease. The climate is mild and slightly humid. See CLIMATE, Treatment of Disease by.

**BOWELS, Diseases of.**—See INTES-TINES, Diseases of.

**BRAIDISM.**—SYNON.: Hypnotism.—Braidism is the name which, after its inventor, James Braid, has been applied to a therapeutic method destined to utilise the undoubted powers of mind over body for the cure of various diseases. In essence it consists of a species of Mesmerism, the patient being reduced to a partial or complete trance-like condition, by being made to look fixedly for a few seconds at a bright object held by the operator at 'about eight to fifteen inches above the eyes, at such a distance above the forehead as may be necessary to produce the greatest possible strain upon the eyes and eyelids, and enable the patient to maintain a steady fixed stare at the object.' The patient must be made to understand that he is to keep his eyes steadily fixed on this object, and his mind riveted upon the image of it. After so short a time as ten or fifteen seconds some patients may be intensely affected; and if so, it will be found, on gently elevating the arms and legs, that the patient has a disposition to retain them in the situation in which they have been placed. 'If this is not the case,' Mr. Braid writes, 'in a soft tone of voice desire him to retain the limbs in the extended position, and thus the pulse will speedily become greatly accelerated, and his limbs in process of time will become quite rigid and involuntarily fixed.' By slightly prolonging this process a condition of profound 'nervous sleep' may be induced, in which operations may be performed as easily and in as painless a manner as if the patient had been under the influence of chloroform. All this has been abundantly proved by Esdaile and others, who performed numerous operations upon Hindoos, with absence of all pain, whilst they were in the hypnotic state. In his attempts to cure morbid conditions, however, Braid only rarely proceeded so far as to induce actual unconsciousness. Whilst in a semi-cataleptic condition the patient's attention is strongly directed to the morbid part, and some very marvellous instances of relief are recorded by him, said to have been effected under the influence of this faculty only, without the aid of imagination, since some of the patients operated upon were quite incredulous as to any good being likely to result. In a recent work on 'The Influence of the Mind upon the Body,' Dr. Daniel H. Tuke remarks: 'Braidism possesses this great advantage, that while the Imagination, Faith or

Expectation of the patient may be beneficially appealed to, this is not essential; the mere concentration of the attention having a remarkable influence, when skilfully directed, in exciting the action of some parts, and lowering that of others. The short period of time required, also, compares favourably with that consumed in some other forms of mental therapeutics. . . . The great principle which appears to be involved in all is the remarkable influence which the mind exerts upon any organ or tissue to which the Attention is directed, to the exclusion of other ideas, the mind gradually passing into a state in which, at the desire of the operator, portions of the nervous system can be exalted in a remarkable degree, and others proportionately depressed; and thus the vascularity, innervation, and function of an organ or tissue can be regulated and modified according to the locality and nature of the disorder.' Braidism is a method very difficult of adoption in ordinary practice, and which, however legitimate may be its foundations, would, unless the greatest care and vigilance were exercised, be apt to descend perilously near to the level of quackery. If, however, only a small part of the results attributed to Braidism would follow the systematic adoption of this method for the alleviation of many diseases, it is one which should commend itself to the earnest attention of inquirers, who may be able to place the practice upon a broader and firmer foundation than that on which it rests. But unfortunately the method has of late fallen somewhat into disrepute. This has been due to two causes. First, because of the facility with which it may be practised by non-medical persons, and the difficulty often experienced, even by medical men, in rousing persons from the state thus induced. And, secondly, because, even when practised by medical men, disagreeable consequences are apt to follow with some patients: fits may be induced, or the patient's moral and emotional control may be for a long time greatly impaired. In consequence of such results, a Government order has been issued in France, forbidding surgeons in the army and navy to have recourse to this method of treatment. Unquestionably, however, the labours of many French physicians have done much of late years to render definite and precise our knowledge of the various phases of hypnotism, of the modes in which they are inter-related, as well as concerning the best means of inducing or terminating this or that particular phasis. A revival of the practice has recently taken place in this country—sometimes in the highly objectionable form of public exhibitions. See MESMERISM.<sup>1</sup> H. CHARLTON BASTIAN.

<sup>1</sup> See also *Mesmerism, Spiritualism, &c., Historically and Scientifically Considered*. By W. B. Carpenter, C.B., M.D., LL.D., F.R.S. 1877.

**BRAIN, Diseases of.—General Observations.**—The range of unnatural phenomena which manifest themselves as the result of disturbed actions of the brain, whether from functional perturbations or structural disease, is wide and varied. This result is due: (1) to the fact that the brain, though spoken of as a single organ, is really a congeries of many distinct but functionally related parts; (2) to the fact that this congeries of parts is continuous with the spinal cord and intimately related to a scattered network of ganglia—entering into the formation of the nervous system of organic life; and (3) to the fact that these several centres within and without the cranium are brought into connexion, through the intervention of nerves, with all other structures in the body, whether entering into the composition of the organs of relation, or into that of the visceral system.

The action of particular parts of the brain may be stimulated, depressed, or suppressed, and either of such altered modes of activity may entail a stimulation, depression, or suppression in the functions of one, two, or more distant parts of the nervous system. The first class of effects are spoken of as *direct*, and the second as *indirect* symptoms. It is often extremely difficult, if not impossible, for us to say which of the symptoms presented by a patient suffering from organic disease of the brain should be ranged under the one head and which under the other. Our ability to make such distinctions is at present hindered by our still incomplete knowledge concerning the anatomical details of the brain, the proper functions of its several parts, and the precise modes in which they co-operate with each other.

The effects of a shock, whether produced by injury or disease, falling on such an extensive assemblage of sensitive and mutually related organs are, as may be well imagined, subject to much variation; and as a matter of fact it happens that in different cases of cerebral hæmorrhage, the symptoms produced are dependent upon three factors, viz., the *situation*, the *extent*, and the *suddenness* of the lesion. Except in so far as the nature of the lesion tends to entail variations in one or other of the above-mentioned respects, it is not of much significance from a clinical point of view (*i.e.* it does not lead to much difference in the sets of symptoms produced) whether we have to do with a case of hæmorrhage into, or with a case of softening of the brain. Thus the 'locality' and extent of the lesion, in the case of a local disease of the brain, has always to be inquired into as a problem altogether apart from that of the nature of the pathological change in the part affected. In other words, the problem of diagnosis in brain-disease is twofold: it must have reference to the region affected

(regional diagnosis) and to the pathological cause (pathological diagnosis). The causes interfering with the progress of our knowledge in the former direction are both numerous and baffling, so that, until the last few years, comparatively little progress had been made.

**SYMPTOMS.**—The most frequent effects or symptoms of functional or structural brain disease may be thus classified:—

**1. Perverted Sensation and Perception.**—The special senses of smell, sight, hearing, touch (fifth nerve), or taste may be interfered with by diseases of their respective nerves or primary ganglia within the cranium. Owing to the decussation of the optic nerves, disease of the optic tract gives rise (most frequently) to an affection of the sight of the opposite eye. The sense of taste is subserved by two nerves proceeding to the same nucleus. Thus, the glosso-pharyngeal has to do with this special sensation in the back part of the tongue, the palate, and fauces; whilst the taste-nerves for the front part of the tongue, though they pass from these parts with the lingual branch of the fifth, leave it by the chorda tympani and then proceed to the brain as the nerve of Wrisberg, which terminates in the nucleus of the glosso-pharyngeal. Disease of the intracranial portion of the fifth nerve thus does not affect the sense of taste, though it impairs the common sensibility of the tongue.

Disease of the primary ganglia of these nerves, whether they are separate (first and fifth) or lodged in the brain-substance at its point of connexion with the nerve, will produce decided impairment of the several special senses. But disease of portions of the brain above these regions on one side only, even though very extensive, often exists without seeming to disturb the exercise of the special senses on either side. This, doubtless, depends in part upon the comparatively wide distribution of the several sets of sensory fibres within the hemispheres, and in part upon the liability of unilateral sensory defects to escape observation. There is, however, one limited region in each cerebral hemisphere where the several sensory paths are brought into close relations with one another, before they diverge to different parts of the cerebral cortex. This region is the posterior third of the hinder segment of the internal capsule, and it is now well known that lesions which involve it give rise to a complete *hemianæsthesia* of the opposite side of the body, in which the special senses as well as the common sensibility of the one half of the body are affected. Sometimes this result seems to be due to functional disorders, and at other times it is a consequence of structural disease. Hemianæsthesia varies widely in its degree of completeness, and also in its duration or persistence, and that is the case where it is of

functional origin as much as where it is due to actual organic disease.

There may, however, be *illusions, hallucinations, or delusions*, in connexion with one or other of the special senses, in many functional and structural diseases of the brain, where the morbid condition is situated in parts higher up than the primary ganglia, or where there is a functional exaltation of the ganglia themselves. This latter functional exaltation seems sometimes to be favoured by morbid states of some of the viscera—especially of the stomach, or of the uterus and ovaries.

Still, modern clinical as well as experimental evidence has done something towards revealing the paths and special cortical termini connected with different modes of sensibility. The experimental observations of Ferrier point to the convolutions about the tip of the temporal lobe as the seats of the senses of smell and taste. The same observer locates the cortical terminus of the sense of hearing in the upper temporal convolution. The more recent experiments of Schäfer and Sanger-Brown seem, however, to negative this conclusion, and to leave its site altogether uncertain.

In regard to the visual sense, both experiment and clinical observation now point to the occipital lobe (and more especially to its inner aspect) as the region most concerned with this endowment. But the relation is a peculiar one. Extensive disease of the inner part of the right occipital lobe, for instance, would not give rise to more or less complete blindness of the opposite eye only—it would produce rather a left-sided hemianopsia. Similarly, the right halves of the visual fields are represented in the left occipital lobe, so that destruction of it would give rise to right-sided hemianopsia.

The cortical centre for touch and common sensibility is now, since the experiments of Horsley and Schäfer, believed to be the hippocampal convolution and the gyrus fornicatus, that is the region of the brain named by Broca the 'faliform lobe.' Under the term 'common sensibility' are included different modes of sensibility; thus it comprises, in addition to touch, the appreciation of differences of pressure and of temperature, and of painful impressions. Ferrier and some others would also include the impressions of the 'muscular sense' under this head of 'common sensibility' and would refer them to the same cortical terminus. This, however, is a position which the writer contests. He believes, with Hitzig and others, that Ferrier's so-called 'motor centres' are in reality muscular-sense centres, and that no real motor centres exist in the cerebral cortex (*Brain*, April, 1887). Others again, like Seguin, without questioning Ferrier's general doctrine as to the existence of motor centres in the cerebral cortex, think there is evidence to

show that 'muscular sense' impressions are registered in the cortex apart from those of common sensibility, and that their special terminus is to be found in the inferior parietal lobule. This subject, therefore, although very important, is at present in a very unsettled condition. See KINÆSTHESIS.

Besides diminutions of sensibility, we often have to do with perversions of sensibility—that is, with disagreeable sensations of numbness, tingling, or actual neuralgic pains in parts. The seat of the two former may be widely distributed, though neuralgia from intracranial disease is principally limited to some part of the territory of the fifth nerve.

**2. Perverted Emotion and Ideation.** These manifestations vary, from the mere increased tendency to emotional displays seen in hysterical persons, or in persons suffering from hemiplegia associated with emotional weakness, to those more complex aberrations met with in the various forms of delirium and insanity. See INSANITY.

**3. Perversions of Consciousness.**—Under this head may be included the comparatively rare states known as somnambulism, ecstacy, catalepsy, and lethargy; as well as the common conditions of drowsiness, stupor, and coma. The former may be said in almost all cases to be associated with functional rather than with structural disease of the brain; at least, this is most in accordance with our present knowledge. Drowsiness, stupor, and coma are, however, among the commonest results of organic disease of the brain (see CONSCIOUSNESS, Disorders of), though they are also common conditions in blood-poisoning—whether arising from fevers, uræmia, or alcohol, poisonous doses of opium, or other narcotic or narcotico-irritant drugs.

**4. Perversions of Motility.**—These manifest themselves in many forms, which, however distinct they may appear to be, are, nevertheless, closely linked to each other.

*Tremors* may be general or local, and in the latter case they may be most marked in the tongue and facial muscles—principally those about the corners of the mouth or the orbicularis palpebrarum. General tremors may arise from debility, over-exertion, 'nervousness,' old age; or they may be due to alcoholic or mercurial poisoning, or to degenerative disease about the pons and medulla, and other regions of the brain.

*Twitchings* may be characteristic of a highly nervous habit of body, and are especially frequent in some epileptics in the interval between their fits, either in some of the facial muscles or in those of the neck or limbs. They may also occur in acute febrile affections, in which the functions of the cerebrum are involved, as shown by co-existing delirium, &c., and also in the course of many organic diseases of the brain. In chorea the irregular movements of different parts of the body are often of this nature;

they may affect both sides of the body, or only one (hemichorea).

*Spasms* of a continuous or 'tonic' character are encountered in various diseases of the nervous system, such as laryngismus stridulus, trismus, hydrophobia, tetanus, hysteria, and some forms of hemiplegia and paraplegia. Such tonic spasms produce a muscular rigidity which has to be distinguished from that due to chronic changes in joints and tendons, such as are apt to occur in limbs long paralysed.

*Clonic Spasms* or *Convulsions* may be either unilateral or general, and may be induced by the most varied causes. When well-marked they are mostly attended by loss of consciousness, as in epilepsy and the majority of epileptiform attacks. They are often limited in their distribution, rather than general, whether the convulsions be of unilateral or bilateral type.

*Co-ordinated Spasms*, or movements of a struggling type, are met with in many epileptiform and hysterical paroxysms. Spasms of this type may be also limited to particular groups of muscles, as in the conjugated deviation of the eyes and neck occurring in hemiplegia, in wry-neck, in writer's cramp, and other allied affections.

*Paralysis* may be local and limited in seat to some of the ocular muscles, the muscles of mastication, the facial muscles, those of the tongue, or to parts supplied by the spinal accessory and pneumogastric nerves, in those cases in which there is merely an implication of the intracranial portion of one or more of the motor-cranial nerves about the base of the brain. In other cases it may take an incomplete or a complete *hemiplegic* type, when the lesions are limited to one half of the brain, and involve directly or indirectly certain regions thereof. Beginning at the surface, these regions include the so-called 'excito-motor' area, comprising the convolutions bordering the fissure of Rolando and the contiguous inner surface of the hemisphere, from which a fan-like distribution of white fibres converges to what is known as the 'internal capsule,' whence these efferent fibres are continued through the peduncle and pons to the bulb. Lesions at the part of the surface mentioned, or directly or indirectly involving any part of the tract of fibres issuing therefrom, give rise to a more or less complete hemiplegic form of paralysis. Whether these portions of the cortex contain motor centres, or centres which register 'muscular sense' impressions, is a matter of interpretation which is at present involved in doubt. What seems certain is that these are the last portions of the cortex called into activity in the execution of movements, and that from them issue the fibres which convey motor incitations to centres throughout the bulb and spinal cord (see KINÆSTHESIS). In other

cases, where a large lesion exists in the pons Varolii, or where the functions of both cerebral hemispheres, or their peduncles, are gravely interfered with, the paralysis may be general, involving both sides of the body. In some of these cases, and especially with right-sided paralysis, various difficulties exist in giving expression to thoughts by means of speech or writing (see APHASIA). Deficient action of the will (induced by lowered activity in one or other of the cortical sensory centres, though without obvious structural change) may cause a temporary paralysis in hysteria and allied states.

*Defective Co-ordination* of muscular acts is met with, as in stammering and in some hemiplegic defects of speech; also in the body generally in some cases of cerebellar disease, producing a peculiar and unsteady gait (titubation) closely resembling that which may be met with in alcoholic intoxication. Similar motor disturbances may be induced by vertigo of well-marked degree. Vomiting, again, is a reflex motor act due to impaired co-ordination, which occurs in many forms of brain-disease. More rarely the sphincter ani and the sphincter vesicæ become relaxed, or the bladder may be paralysed. But incontinence of feces or of urine, or inability to void the urine, is comparatively rarely met with as a result of brain-disease, except in the comatose state, in patients who are more or less demented, or in those in whom lesions exist in both hemispheres of the brain.

##### 5. Nutritive or Trophic Changes.—

With lesions in the 'motor tract' of the brain, in or below the Rolandic area, a band of degeneration occupies a part of the internal capsule, crus cerebri, pons, and medulla on the same side, and (below the decussation of the pyramids) the opposite lateral column of the spinal cord. This is one of the most important of the trophic changes occasioned by brain-disease, because the degeneration in the lateral column of the cord is apt to spread to the contiguous grey matter, and thus give rise to some secondary trophic changes not infrequently met with in paralysed limbs.

Trophic changes in other organs (occasioned by some severe lesions in the brain) appear as low inflammations and congestions—for example, of the lungs, or as hæmorrhages into these organs; also as hæmorrhages beneath the pleura or endocardium, or even into the substance of the suprarenal capsules or kidneys.

Again, we may have acute sloughing of the integument in the gluteal region on the paralysed side, dropsy of paralysed limbs, inflammations of joints and of the main nerves of paralysed limbs, and, though more rarely, marked atrophy of paralysed muscles. Retardation or arrest of growth is also apt to occur in paralysed limbs, when we have to do with infants or young children suffering from severe organic brain-disease.

Blanching of the hair, or altered pigmentation of the skin, also occurs not infrequently in connexion with brain-disease or violent mental emotions; whilst in the insane the nutrition of bones, and of the pinna of the ear, is apt to be interfered with.

**6. Perverted Visceral Actions.**—Exalted activity of the uterus, bladder, intestine, stomach, or heart may be occasioned by functional brain-disturbance more especially; whilst related brain-conditions may give rise to depressed or exalted activity of the liver or kidneys. With other functionally disturbed or emotional brain-states there may be a lowered functional activity of the salivary glands, of the heart, of the respiratory organs, of the organs of deglutition, of the organs of digestion, or of the sexual organs. These are only to be taken as mere indications of the kinds of modification that may be produced in visceral activity by brain-disease. Much doubtless remains to be learned in this direction.

It seems fitting here also to mention those contractions and dilatations of vessels which are apt to take place in different parts of the surface of the body, or in internal organs, from stimulation or the reverse of vaso-motor nerves, occasioned either by direct or indirect influence exerted upon the principal vaso-motor centres in the region of the medulla. These contractions or dilatations produce correlated alterations in the temperature, sensibility, and functional activity of the parts or organs affected. The temperature of paralysed parts, as well as the general body temperature, is subject to great variations in the apoplectic state, and these are now beginning to be studied more attentively. They are capable of yielding diagnostic indications of great value.

**GENERAL REMARKS.**—Some general remarks on the subject of structural and functional diseases of the nervous system, showing how intimately these two classes of disease are related to one another, will be found in the article **NERVOUS SYSTEM**. Most of what is said there is applicable to diseases of the brain in particular; here, however, it is necessary to call attention to certain points specially related to brain-disease.

When paralysis occurs from brain-disease affecting one cerebral hemisphere, in the great majority of cases it is situated on the opposite side of the body, owing to the fact that the fibres conveying the volitional impulses to the muscles decussate in the medulla oblongata. It is true that many cases are on record in which the paralysis either has, or has been said to have been present on the same side as the brain-lesion. A certain number of these cases are probably due to errors either in the clinical or in the post-mortem records of the case. Others, however, may be due to the fact that in the individuals in question the customary decus-

sation, above referred to, did not exist. The characters of the various forms of paralysis due to brain-disease are briefly set forth in the article on **PARALYSIS**.

Lesions of the left hemisphere, much more frequently than those of the right, are associated with aphasic defects of speech; whilst, according to Brown-Séguard, lesions of the right hemisphere are more frequently and rapidly fatal than otherwise similar lesions of the left hemisphere. They are also said to be more apt to be associated with acute sloughing of the skin on the paralysed side. Convulsions at the onset, and subsequent tonic spasms of the paralysed limbs, are also said by the same authority to be more frequently associated with left- than with right-sided paralysis. These statements, however, stand in need of confirmation.

Congenital atrophy of one hemisphere, or an atrophy or severe lesion occurring in early infancy, is mostly associated with an arrest of growth and development in the limbs on the opposite or paralysed side of the body, or with a more or less spastic condition of the limbs ('birth palsies').

Very little is positively known concerning the diseases of the cerebellum. Of its functional affections we may be said to know absolutely nothing. That is, of the various functional diseases of the nervous system with whose clinical characters we are familiar, we are unable to name even one which we can positively say is a functional disease of the cerebellum. Whatever the precise mode of activity of the cerebellum may be, there is a general consensus of opinion that it is principally, if not exclusively, concerned with motility, and that it has more especially to do with the higher co-ordination of muscular acts. Atrophy of one hemisphere of the cerebellum is followed by atrophy of the opposite half of the cerebellum, so that there is a strong presumption that the functional relationship of either half of the cerebellum is with muscles on the same side of the body. Clinically we know that disease of the cerebellum is not infrequently associated with more or less marked paralysis on the opposite side of the body; but this effect is now generally attributed to the pressure which structural diseases of the cerebellum are apt to occasion on the pons and medulla of the same side.

**ÆTIOLOGY.**—The principal modes of causation of diseases of the brain, functional as well as structural, may be thus summarised:—

1. *Defective nutrition* operates by modifying the proper constitution of nerve-tissues as well as the constitution of the blood, and thereby interfering with the normal functional relations of the several parts of the brain. Anæmia, chlorosis, syphilis, ague, and all lowered states of health, howsoever induced, and whether acquired or inherited, become predisposing or actual causes of brain-disease.

To these states, favourable to the manifestation of brain-disease, should be added the various acute specific diseases, uræmia, metallic poisoning, poisoning by the narcotic and narcotico-irritant poisons generally, and also by the occasional qualities of certain articles of food, such as mackerel, mussels, mushrooms, &c.

2. *Emotional shocks* cause cerebral disorder, especially in children; whilst prolonged overwork, particularly when combined with worry and anxiety, with sexual excesses, or with protracted lactation, is apt to induce it in those who are older. Religious excitement, again, in combination with lowered states of health, not infrequently leads to insanity.

3. *Physiological crises*, such as the period of the first dentition, the period of puberty, pregnancy, and the climacteric period, all favour the manifestations of various nervous diseases.

4. *Visceral diseases or surface-irritations* (especially in children or in persons having a very sensitive and mobile nervous system) may give rise to varied nervous diseases. Thus we may have convulsions or delirium in children from the presence of worms or other irritants in the intestines; or convulsions in adults during the passage of a renal calculus. Again, we may have the phenomena classed as hysteria, or we may have nymphomania, in consequence of certain states of the sexual organs. Cases of paralysis are said also to have a reflex origin occasionally, though this must be a very rare event. With much greater frequency we find surface-irritations of various kinds leading, as in Dr. Brown-Séquard's guinea-pigs, to epileptiform attacks.

5. *Structural lesions* of the brain itself give rise to a very large proportion of its diseases. The various kinds of change will be found enumerated under another heading (*see* NERVOUS SYSTEM, Diseases of). Hæmorrhage, softening, and tumours are the most common, and, therefore, the most important of these morbid conditions.

6. Brain-disease may be determined by the action of *heat (insolatio)*, especially when combined with fatigue and deficient aëration of blood. A somewhat similar brain-affection, however, is occasionally developed in the course of rheumatic fever or in that of one of the specific fevers, especially typhoid or typhus, when the temperature of the body rapidly rises to a lethal extent (109°–111° F.).

7. *Concussions* (whether from blows or falls) may give rise to brain-disease, even where no traumatic injuries or lacerations of the brain seem to have been produced.

TREATMENT.—The treatment of brain-disease will be discussed under the articles NERVOUS SYSTEM, Diseases of; PARALYSIS; and CONVULSIONS; and in those on the several special diseases which will now be described in alphabetical order.

H. CHARLTON BASTIAN.

**BRAIN, Abscess of.**—This term is applicable whenever a circumscribed collection of pus is formed in any part of the cerebral mass.

ÆTIOLOGY AND PATHOLOGY.—Amongst the most frequent causes of cerebral abscess are severe injuries to the skull, disease of the temporal bone in connexion with the ear, ligature or obstruction of a main artery, and pyæmia. Under the three first-named conditions the abscess is usually solitary, but from pyæmia multiple abscesses often result. For practical purposes we may perhaps conveniently discard the latter—since the symptoms will usually be those of general encephalitis—and confine ourselves to those cases in which single, large-sized collections of pus are met with. With this limitation, abscess in the brain is by no means of frequent occurrence.

The best marked, and also the more common examples of large cerebral abscess are met with in connexion with compound fractures of the skull, and by far the most definite symptom which denotes them is the formation of a fungus cerebri. Unless in a compound fracture the brain be directly injured and the dura mater torn, it is very rare indeed for any supuration in its substance to occur. It is not to be denied, however, that now and then, after severe concussion or laceration without external wound, abscess may follow. In such cases we may conjecture that usually some slight laceration or extravasation occurred in the first instance, which constituted a focus for the inflammation. Abscess after simple concussion without lesion is probably a most rare event.

In the article BRAIN, Inflammation of, we shall have to define Encephalitis as a diffuse change of a large part of the cerebral mass, perhaps of a whole hemisphere, attended by the infiltration of cells and fluid. It is obvious that the term abscess in the brain is applicable to one of the results of encephalitis, but it seems clinically probable that the two classes of cases are for the most part distinct, and that diffuse encephalitis has but little tendency to result in abscess, and that abscess is rarely preceded by a stage of encephalitis. Both are usually the consequences of local injury to the brain, or of extension from local disease of its coverings; but whilst encephalitis probably resembles the erysipelalous type of inflammatory action, in localised abscess this tendency is not present. Having distinguished brain-abscess from encephalitis, we must next say a word as to the risk of confusing it with intra-cranial but extra-cerebral collections of pus. Encysted collections of matter may be met with either between the dura mater and bone, or within the arachnoid cavity; and perhaps it ought to be added, though with some hesitation, beneath the arachnoid, in the pia mater. Not infrequently inflammation of the membranes

precedes and attends the formation of an intra-cerebral abscess, and in these cases the symptoms will be mixed. In dealing with published cases it is also necessary to be on our guard as to certain errors which have crept in—cases of meningeal abscess being spoken of as brain-abscess. Sir Prescott Hewett has expressed his opinion that the celebrated case of de la Peyronié's was an example only of extra-cerebral abscess. It is absolutely necessary to make these restrictions, if we would judge correctly as to the symptoms which attend local collections of matter in the brain, and the usual terminations of such cases.

The contents of a cerebral abscess usually consist to some extent of broken-up cerebral tissue, and in some cases there are but very few pus-cells. Especially is this likely to occur when the so-called abscess follows on ligature of the carotid or occlusion of a cerebral artery. In these cases, it is, in the first instance at least, the result of a process of softening rather than of true suppuration.

**SYMPTOMS.**—The symptoms of local suppuration in the brain will vary with the stage, the size of the collection, its precise situation, and above all, with the presence or otherwise of a fistula of relief. In many of the cases which come under surgical care a fistula exists from a very early period, though not infrequently it is liable to occlusion. Under the latter conditions the symptoms of a closed and an open cerebral abscess may be alternately studied in the same case. It will usually be observed that when the exit is closed, and the abscess fills, the patient complains more or less of headache, becomes heavy and drowsy, and experiences twitchings or spasms in the opposite side of the face and limbs, with some tendency to hemiplegia. Of this group the tendency to spasms is probably by far the most significant. The headache may be but trifling, and the patient may even be well enough to leave his bed, when the occurrence of spasm followed by paresis alone gives warning of what is going on. Temperatures are in such cases very misleading. Persistent elevation is a symptom to be attended to, but its absence is no safe ground for negative conclusions. The cases now alluded to are chiefly those in which abscess results from compound fracture of the skull with laceration of the brain-substance. In these the abscess often gives way spontaneously, and a fistula forms, around the orifice of which a mass of pouting brain-granulations, known as *fungus cerebri*, usually forms. In these cases the canal of communication may be very tortuous, and the liability to blocking considerable. Now and then the same result may be met with after syphilitic disease of the skull and meninges. The writer had some years ago a man under his care in whom he had opened a cerebral abscess beneath a hole in one

parietal bone. The patient was able to walk about, and ailed but little so long as the fistula was freely open; but spasms of the face, or even convulsions of the limbs (on the opposite side), always followed its occlusion. The softening gradually extended, and he at length died in consequence, perhaps, of the impossibility of making a counter-opening in a depending situation. Unless the abscess be in the anterior lobe, there will almost invariably be present some degree of hemiplegia, but this will of course vary with the size of the collection and the extent of destruction of tissue. The formation of an abscess after injury is sometimes very insidious, the symptoms being very slight. The cases in which violent headache and pain, vomiting, delirium, and dry tongue, are said to have been present in the early stages are, it may be suspected, usually instances of suppuration between the bone and dura mater. These symptoms occur especially when brain-abscess follows disease of the internal ear, and in these there nearly always is the complication of inflammation around the petrous bone. Such symptoms are very rarely present in traumatic abscesses, which often develop very quietly until they attain a considerable size. It is probable that some degree of rigor, attended with rise of temperature, usually occurs in the beginning of cerebral abscess, but no very precise data are extant on these points.

If a large abscess be permitted to develop without relief, the symptoms of compression will in time ensue: first spasm, then hemiplegia, then hebétude and coma, preceded possibly by violent convulsions. There are cases, however, in which the tendency to increase ceases, and the abscess may pass into a quiescent state and remain for indefinite periods without symptoms.

**DIAGNOSIS.**—The diagnosis between cerebral abscess and meningeal abscess is exceedingly difficult, and often a guess is all that can be made. The almost invariable occurrence of spasm or convulsions in the former, and their frequent absence with the greater degree of pain and headache in the latter, are the most reliable signs. Sometimes—as, for instance, when trephining has been practised, and no inflammatory products are found between the bone and dura mater or under the latter—the diagnosis may be helped by this negative knowledge. In such cases, if hemiplegia, preceded by spasm, have been gradually developed, the surgeon will be well justified in making an incision or puncture into the hemisphere. Optic neuritis may equally be present in both, and its presence or absence will scarcely help the diagnosis.

**PROGNOSIS.**—In addition to the danger of death by compression, there is the risk that the abscess may break into the ventricles or into the subarachnoid space. Some cases are on record in which spontaneous openings

into the nose or into the ear occurred, and profuse discharge followed, the patient in the end recovering. It may be doubted whether these were not instances of meningeal abscess.

**TREATMENT.**—It is needless to say that if abscess be diagnosed with any degree of confidence an opening is essential, and the case must pass into the hands of the surgeon. There is little or no room for medical treatment. Of late years operations with the trephine for the relief of cerebral abscess have been attended with very encouraging success. For the prevention of abscess, in all cases in which injuries likely to produce it have occurred, the utmost precautions should be enforced. Mercury in small doses, frequently repeated, should be given from the first; the injured region should be covered with lint soaked in a strong spirit-of-wine lotion, frequently re-wetted; purgatives should be administered; and the patient should be kept very quiet until long after the healing of the wound.

JONATHAN HUTCHINSON.

### **BRAIN, Anæmia of.**—DEFINITION.—

A condition in which the blood contained within the brain, and especially within the capillaries of the brain, is deficient in quantity, or defective in nutritive quality, or both.

The blood within the brain is contained in arteries, capillaries, and veins. The functional condition of the brain depends on the quantity and quality of the blood circulating in its capillaries, and it is to these that the special symptoms are related. Deficiency in the quantity contained in the arteries or veins can scarcely occur without the condition being shared by the capillary circulation. Defect in the quality of the blood supplied to the brain is always of gradual occurrence, and affects the whole brain; deficiency in quantity of blood may affect the whole brain or part only, and it may be sudden or gradual in its production.

**ÆTIOLOGY.**—*General cerebral anæmia* may be due to the following causes:—(1) It may be a part of systemic anæmia—defect in quantity or quality of the whole blood, and from causes which are considered elsewhere. This is often seen in cases of hæmorrhage, of exhausting discharges, or of defective blood-nutrition, as in chlorosis. (2) The supply of blood to the brain may be deficient, the quantity of blood in the body being normal. The cerebral anæmia is then part of a cephalic anæmia. In consequence of posture in relation to gravitation the supply of blood to the head is that which is especially interfered with by any diminished movement of the blood. At the same time the closure of the cavity in which the brain is situated, and the unreadiness of the cerebro-spinal fluid to undergo rapid change of distribution,

preserve the blood in the head from the extremely rapid diminution to which it would probably otherwise be liable—possibly, indeed, to a degree incompatible with life. Cephalic anæmia may be due to cardiac weakness, or to causes acting through the nervous system on the heart, as in swooning. In systemic anæmia, the lessened cardiac power increases the cerebral deficiency. Whatever lessens the amount of blood discharged from the heart at each systole, such as aortic or mitral disease, may be a cause of cerebral anæmia. Pressure on the vessels conveying the blood to the head, as by an aortic aneurysm, has a similar effect. Unequal distribution of the systemic blood is another cause. The intestinal vessels, if dilated, are capable of containing a large part of the blood of the body, and the effect of their engorgement is often seen after paracentesis abdominis. One theory of shock ascribes its mechanism to vaso-motor dilatation of these vessels, and consequent anæmia of the rest of the system. Severe diarrhœa or dysentery is an occasional cause of symptoms of a very pronounced and prolonged character. (3) The capacity of the cerebral vessels may be diminished by pressure on the brain, exerted by effusions of fluid (hydrocephalus), of blood (in cerebral and meningeal hæmorrhage), or by growths within the skull. (4) General contraction of the arteries of the brain is a rare cause, probably confined to some toxic states, such as that of Bright's disease, in which such contraction can be seen in the retina.

*Partial cerebral anæmia* is due to local obstruction to the passage of the blood through an artery. To be permanently efficient such obstruction must be situated beyond the circle of Willis. Ligature of one carotid causes immediate symptoms of cerebral anæmia, but permanent symptoms are not frequent. Pressure on, or disease of, one carotid, for the same reason, rarely gives rise to symptoms. Obstruction in certain arteries of the brain may cause local anæmia, sudden or gradual, temporary or permanent, according to its cause. Such obstruction may be due to narrowing of the calibre of the vessel by atheromatous changes in its wall, or by spasm of its muscular coat, or may be due to actual occlusion by embolism or thrombosis. The pressure-effects of an intruding substance within the skull (tumour, or clot) act most intensely in one region of the brain, and may influence it only. Arterial spasm is perhaps an occasional cause of local anæmia. Extensively as it has been invoked as a mechanism to explain symptoms, its occurrence has not yet received any definite proof.

It is obvious that of these causes some act suddenly, others gradually, and the symptoms produced will differ accordingly.

**ANATOMICAL CHARACTERS.**—The principal anatomical character of cerebral anæmia is

pallor of the brain, observable chiefly in the paler tint of the cortical substance, and the diminished number of red spots in the white centre. The pallor may be partial or general. The membranes are usually pale, but in some cases of partial anæmia they are hyperæmic. Effusion of serum in the meshes of the pia mater and between the convolutions may be found in general anæmia.

**SYMPTOMS.**—The symptoms of this condition vary according as the anæmia is suddenly or slowly produced, and as it is general or partial.

(1) In *sudden general* anæmia of the brain the sufferer feels drowsy; the special senses are dulled; noises in the ears and vertigo are complained of; the pupils are at first contracted; sight may fail; muscular power is weakened; respiration is sighing; the skin is pale, cold, and moist; nausea is common; and headache is rare. If the anæmia is more intense, consciousness is lost; there is universal paralysis; and general convulsions may occur, epileptiform in character, these being especially frequent in sudden extensive losses of blood in strong subjects. The pupils dilate, and the coma may deepen to death. The loss of sight may persist, in cases which recover, but retinal changes are then found.

(2) When general anæmia of the brain is *slowly* produced, the state of the cerebral functions is usually that of 'irritable weakness.' Their action is imperfect in degree, and excited with undue facility. There is mental dullness and drowsiness; sometimes, however, insomnia is troublesome. Delirium is common in severe cases, and is conspicuous in some forms of imperfect blood-nutrition, as in the so-called 'inanition delirium.' Headache, usually general, is a common symptom. Sensory hyperæsthesiæ, tinnitus, *muscæ volitantes*, and vertigo are frequent. Convulsions are rare, but muscular power is generally deficient. All these phenomena are more marked in the erect than in the recumbent posture, especially when the erect posture is suddenly assumed. It has been remarked that some anæmic persons can think well only when lying down. In young children, after exhausting discharges, as diarrhœa, symptoms referable to cerebral anæmia are common, namely, somnolence and pallor, with depressed fontanelle and contracted pupils. The somnolence may deepen to coma with insensitive conjunctiva, and the coma increase to death. Such symptoms have been called *hydrocephaloid*, from some resemblance to those of acute hydrocephalus.

(3) *Partial* cerebral anæmia causes, if complete, loss of function in the affected area; and if it be permanent, as in obstruction of a vessel beyond the circle of Willis, necrosis of the cerebral tissue results (*see* BRAIN, Softening of). If incomplete and sudden, there is temporary arrest of function.

Ligature of one carotid, for instance, causes transient weakness and numbness in the opposite half of the body. There may be at first an over-action of grey matter, causing, in certain regions, unilateral convulsions. The return of the blood-supply, as when a plug shifts its position or is broken up, may not immediately be followed by restoration of function, which may be put in abeyance for some time by the effect of the sudden influence. The loss of memory is occasionally of a remarkable character, involving only the power of retaining new impressions. If slowly developed, as in atheroma of arteries, pain and vertigo are common, with recurring local symptoms, such as numbness, tingling, and weakness.

In all cases of long-continued cerebral anæmia, prolonged damage to the nutrition of the brain may result. In the child the development of the brain may be arrested; in the adult, loss of memory and of general mental power indicate the deterioration of structure. These may last for many months, but commonly pass away at last.

**PATHOLOGY.**—The symptoms are, as already stated, dependent mainly on the defective quantity and quality of the blood circulating in the brain. Some influence may probably be ascribed to the diminution in the blood-pressure to which the nerve-elements are ordinarily exposed (Burrows). Nothnagel has pointed out that the symptoms indicate an early affection of the respiratory centre in the medulla, and of the cortical grey matter. The convulsions in acute anæmia have been ascribed to irritation of the medulla or of the pons Varolii, but our present knowledge suggests that they are due to the altered function of the motor region of the cortex.

**DIAGNOSIS.**—The diagnosis is not difficult. It rests on the recognition, in a given case, of the cause of cerebral anæmia; and on the exclusion of graver maladies, as organic cerebral disease. With the latter, it should be remembered, anæmia of the brain, local or general, often co-exists. Some symptoms of hyperæmia of the brain closely resemble those of anæmia. A common pathological state of imperfect blood-renewal probably exists in both conditions.

**PROGNOSIS.**—The extent to which the cause of the anæmia is amenable to treatment, and is of transient character, must influence the prognosis. As a rule this is favourable when there is no organic disease of heart, vessels, or brain. In the so-called 'pernicious anæmia,' the prognosis is, of course, unfavourable. Hydrocephaloid symptoms in infants, if met by prompt and suitable treatment, are usually recovered from.

**TREATMENT.**—The treatment necessarily varies in the several forms of the affection, but it is in the main causal. The beneficial effect of the recumbent posture in affording immediate relief to the symptoms, and obviating

permanent damage to the cerebral nutrition, must be always remembered. In acute anæmia from loss of blood, the head must be kept continuously low, stimulants freely administered, and as a penultimate resort bandages applied to the limbs from below upwards may increase the proportionate supply of blood to the brain. If this fails transfusion must be had recourse to. In chronic anæmia sudden change of posture should be carefully avoided, and ferruginous tonics are needed. More important than any other element is physical rest, permitting more of the scanty amount of oxygen that can be conveyed by the blood to be used for the nutrition of the protoplasm, *i.e.* tissues on which the vital functions depend. In spasm of the cerebral vessels nitrite of amyl or nitroglycerine should be employed; and a course of the latter, in a small dose two or three times a day, lessens any liability to irregular contraction that may exist. In the cerebral anæmia of syncope, the recumbent posture, stimulants to the skin, cold water, faradisation, sinapisms, and ammonia to the nasal mucous membrane, assist the recovery of cardiac action and the return of consciousness. In all cases, carefully regulated food and stimulants are needed; beef-tea should be given in small quantities at frequent intervals. The group of symptoms called hydrocephaloid requires similar treatment.

W. R. GOWERS.

**BRAIN, Aneurysm of.**—*See* BRAIN, Vessels of, Diseases of.

**BRAIN, Atrophy of.**—Atrophy of the brain may be congenital, due to arrest of development in very early foetal life; or the constituents of the brain may have been perfectly developed, and may subsequently disappear from one of several causes. This morbid state is regarded as *primary* when there has been no pre-existing disease of the brain or its membranes; *secondary*, either when there has been such pre-existing disease, and the atrophy has occurred from absorption of the part broken down by inflammation, softening, hæmorrhage, &c., when atrophy of some special cerebral organ follows upon destruction of the particular nerve that arises from it; or when localised atrophy has taken place in a very gradual manner from pressure of a tumour; of ventricular effusion, &c. To take these forms in order:—

1. *Congenital Atrophy.*—This is usually associated with weakness of intellect, even to the extent of idiocy. There is atrophy of the body opposite to the side of the cerebral lesion, and this atrophy involves all structures, even the bones. Paralyses of various intensity supervene, often with contraction of the paralysed parts; there being no particular sensitiveness of the special senses, possibly because of the mental hebetude. Epileptic attacks are common; vitality is

low; and the patient easily succumbs to other diseases.

2. *Primary atrophy.*—In this variety there is general diminution both of volume and of weight, affecting most usually the cerebral hemispheres, and that in pretty equal degree. It is most common as a condition of old age—*senile marasmus, atrophia cerebri senilis*. It sometimes in earlier life follows exhausting diseases; and may also be caused by deficient or impure blood-supply to the brain. In close connexion with this mode of causation it is seen after repeated attacks of intoxication, especially after delirium tremens. In this last condition the cerebral atrophy may be acute and rapid.

3. *Secondary atrophy.*—This may be *general* or *partial*. When *general*, the convolutions have a shrunken appearance, and there is always an increase of the subarachnoid fluid. This condition follows various lesions of the brain, especially of the convex surface, such as hæmorrhage of the convexity, encephalitis, or more accurately that form of encephalitis which attacks only the grey matter, or perhaps only one layer of the grey matter, as in some mental diseases. When the atrophy is *partial*, there are found depressions in an otherwise normal hemisphere, at which point a localised hæmorrhage or patch of softening, inflammatory or depending on thrombosis or embolism of vessels, has been absorbed, leaving only a cavity of greater or less extent, filled with fluid and sometimes lined with a thin membrane. A certain portion also of the brain may become atrophied by the gradual pressure of a tumour or any foreign body. Even the little sieve-like depressions seen in various situations after constantly repeated congestion of vessels may be the result of atrophy from compression by the distended vessels. Functional inactivity and atrophy of the optic nerves have led to a similar abnormality of the corpora quadrigemina, and of the higher centres of the perception of vision.

**ANATOMICAL CHARACTERS.**—The atrophied brain, or portion of brain, will vary in appearance on minute examination, according as the lesion has, or has not, been preceded by inflammation. When the atrophy is primary and due to gradual interference with the blood-supply, there is seen a shrunken condition of the nerve-tissue, especially of the calibre of the nerve-tubes. The cells are smaller than usual and pigmented; the arteries are decreased in size or themselves diseased. If the lesion has had an inflammatory origin, the process in order of sequence is, first, inflammation, then softening with fatty degeneration, then partial absorption, and so atrophy; the appearances differing according to the stage of the lesion. Traces of fatty degeneration of all the tissues—vessels, nerve-tubes, and cells—may be found, with the neuroglia either wanting or sclerosed.

Dr. Rudolph Arndt has lately thrown doubt on the possibility of determining atrophy of the ganglionic bodies or nerve-tubes by their size. The size of these bodies varies so greatly, within the limits of health, that he considers this test a very uncertain one. Almost the only trustworthy sign of atrophy, in his opinion, is the appearance in the substance of the ganglionic bodies and in the medullary sheath of nerve-bundles, of black shining globules, somewhat fatty-looking. In process of time these globules increase in number, and at last the whole of the bodies appear permeated by them. These globules are not fatty: they are certainly more or less pigmental. Exactly similar bodies are found in post-mortem examinations, and are a sign of simple decomposition. The duration of this condition, irrespective of pre-existing lesions, is protracted.

**SYMPTOMS.**—The symptoms of atrophy of the brain necessarily vary according to the seat, extent, and aetiology of the lesion. Primary atrophy of the cerebral hemispheres and the first form of secondary atrophy will most usually induce imbecility, or some lesser degree of mental insufficiency, loss of memory, slowness of thought, and other mental diseases. Headache, giddiness, delirium, and stupor are seldom met with. Interference with speech is more common. Affections of sight, and of the motor condition of the eye, do not depend on general atrophy of the brain; they own a more local cause.

Convulsions, paralysis, stiffness of muscles, or contractions are often met with in connexion with this general lesion, as well as various irregularities of locomotion; but it must be remembered that general atrophy of the brain is not seldom associated with atrophic or sclerotic lesions of the spinal cord, and, even where this is not so, several of the morbid phenomena, and particularly convulsions and paralysis, may derive their origin, not from the atrophy, but from the condition pre-existent to the atrophy, such as hæmorrhage of the convexity, meningitis, or periencephalitis.

In secondary atrophy of a more limited extent, the symptoms are apt to be more strictly localised, such as partial loss of power in a single limb, slight imperfections of speech, or strabismus; but here again the positive diagnosis of atrophy is hindered by the complication of pre-existent disease, the local congestions, hæmorrhages, softenings, tumours, or other conditions of which the atrophy is only the sequence. Still less characteristic are the phenomena attending general or partial atrophy of the cerebellum, the pons, and the medulla oblongata.

**TREATMENT.**—Treatment is useless as to the atrophy of the brain: it must be directed to supporting the powers of the patient.

E. LONG FOX.

**BRAIN, Carcinoma of.** See BRAIN, Tumours of.

**BRAIN, Compression of.**—The brain is compressed in the pathological sense whenever its structure is so squeezed that its functions are in any degree interfered with. This squeezing may be effected either by the effusion of blood within the skull, the growth of a tumour, the accumulation of pus or serum, or lastly by the depression of some large portion of the bony parietes. The general belief that depressed fractures are frequently the cause of compression is probably quite a mistake. In such cases the fragment displaced is rarely of sufficient size to cause serious compression of the contents of the skull, and the symptoms usually supposed to indicate that state are really due in most cases to laceration and contusion, or to subsequent inflammation. This point is of great importance in practice, for upon its recognition depends much of the validity of the reasoning by which the operation of primary trephining in compound fractures is defended or condemned. It also offers a most serious fallacy as regards the interpretation of the symptoms due to compression.

By far the best examples of uncomplicated compression of brain are supplied to us by the not very infrequent cases in which a middle meningeal artery is injured, and a large blood-clot is effused between the dura mater and bone. It is from observation of cases of this kind that the assertion is justified, that a very considerable intrusion into the skull may be permitted without the production of any symptoms. No doubt the suddenness or otherwise has much to do with the results, but there seems good reason to believe that, as a rule, the brain will easily accommodate itself to quantities not exceeding an ounce or two, and that usually as large a quantity as four or five ounces is required to cause death. It is very rarely indeed that a depression of bone in the least approaches such an extent of intrusion as this, and the majority of such cases are, as regards the amount of possible squeezing, quite trivial. The manner in which the accommodation is effected is by the removal of the fluid contents of the skull—first the subarachnoid fluid, and secondly the blood. Of the blood-vessels the veins and venous sinuses are probably emptied first, and lastly the arteries and capillaries. A brain in a state of strong compression is an exsanguined brain. In this respect, the brain in the last stage of compression differs very much from that in cases of insensibility from concussion or contusion. It by no means follows that because the brain is pale, the face should be pale also; but it is perhaps usually the case that extreme compression so much enfeebles the heart's action that the pulse is weak or flickering, the respiration shallow and irregu-

lar, and the skin pale and cold. That stertorous breathing, a laboured pulse, and a suffused and dusky countenance are (as according to the classical description) symptoms of compression, is probably for the majority of cases a mistake. Such a group much more frequently denotes laceration, contusion, or central extravasation. Nor is it true that hemiplegia, excepting of the most transitory kind, is often due to compression. A clot of blood poured out over one hemisphere may, if rapidly effused, produce for a while weakness of the opposite limbs, but the brain-mass is soft enough to allow of considerable yielding, and in the course of a few hours the effects of the displacement will have become general, and not local. In a case recorded by the writer in which a post-mortem examination, some weeks later, proved the presence of a large blood-clot, there had been partial hemiplegia without unconsciousness at first, but on the next day all trace of it had disappeared, and it never returned. In many of the cases of bleeding between the dura mater and bone, from the meningeal artery, the hæmorrhage takes place on several different occasions, with, it may be, intervals of a day or two, much as is often observed in wounds of arteries, such as the palmar arch. Thus the observer is able to appreciate the symptoms caused by different degrees of compression, and further proof is afforded that, if the intruded quantity be but moderate, the brain bears it without obvious inconvenience. Often at the autopsy it is quite easy to distinguish clots of very different dates, and to feel sure that the original one was of considerable size. It is clear then that in speaking of the symptoms of compression we must allow for differences in amount of the compressing substance, and also for differences in the rapidity or suddenness of its application.

When compression is produced suddenly, as by a large fragment of bone driven down, the case is almost invariably complicated by contusion. If paralysis or even insensibility be present, it is usually impossible to say to which lesion they are really due. We may, however, hold it almost certain, from what we know of other cases, that the effects in such would be a temporary hemiplegia, with symptoms of shock, if the depression were but moderate; and insensibility, probably soon followed by death, if the depression were very great. The cases in which depression of bone has alone been sufficient to produce long-continued compression with insensibility are possibly somewhat apocryphal. It is possible that compression under such circumstances might be attended by stertor and laboured pulse, but it is possible also that the pulse might be extremely feeble, the countenance pale, and inspiratory efforts weak and irregular.

The depression of bone is perhaps the only

condition which can be supposed capable of producing compression suddenly. When blood is poured out from a ruptured artery, the symptoms come on rather gradually. The patient complains perhaps of headache, and then becomes more or less confused in manner, his gait is unsteady, and the limbs on the side opposite to the injury show special weakness. Vomiting may occur and the weakened limbs may twitch, and unless, as is often the case, the intracranial bleeding stops, these symptoms are soon lost in a state of complete insensibility, with pale face, feeble pulse, and symptoms of shock. Convulsions may now occur, and death often supervenes very quickly. In such a case the whole course of the symptoms may occupy less than an hour. The surgeon ought, of course, to trephine and let out the blood, and he must be prompt, or his patient may die during his preparations. More commonly this rapid termination occurs unexpectedly after one or more previous attacks of temporary head-symptoms, and the patient may have appeared quite well in the intervals. In cases in which the symptoms progress without interruption, their rapidity, no doubt, depends upon the size of the vessel ruptured. Certain special symptoms will also depend upon the precise position taken up by the clot which may chance to press upon special nerve-trunks as well as upon the brain-mass. In ordinary cases the clot is beneath the squamous bone and the lower part of the parietal, and passes downwards into the sphenoidal fossa. In the latter region it may press upon the nerves going to the sphenoidal fissure; and it is of importance for the surgeon to know that dilatation of the pupil on the affected side is often produced. This important symptom is probably due to pressure upon the third nerve.

There is yet another class of compression-cases in which that condition is produced by the slow accumulation of the products of inflammation within the skull. Much will depend, as regards special symptoms, upon the position of the abscess, either within or without the brain. If in the substance of the brain, it must more or less disorganise its structure, and thus cause symptoms due to laceration as well as compression. Under such conditions some degree of hemiplegia, with, probably, preceding spasms of limbs, can scarcely fail to be present. Now and then cases occur in which an irregular sinus leads into an abscess-cavity in the brain, and this sinus being sometimes free and sometimes blocked, the surgeon has repeated opportunity of estimating the effects of filling of the cavity. In such cases, headache, stupor, unilateral twitching of limbs, partial hemiplegia, with perhaps vomiting, and, it may be, general convulsions, are the symptoms to be expected. The position of the abscess as regards different regions of the

brain is also of much importance, but its discussion cannot be entered upon in any detail here, and it obviously concerns rather disorganisation of structure than simple compression.

When a large accumulation of pus takes place between the bone and dura mater, the symptoms produced are much the same as those caused by blood-clot. We must make, however, much allowance for the fact that these cases are almost always attended by meningitis, and thus the symptoms of compression are masked by those due to inflammation. Chronic abscess under the bone without arachnitis may occur now and then in syphilitic and other disease of the cranial bones, but such cases are very rare. They will differ from those of hæmorrhage in that the symptoms are always produced very slowly. The writer once had the opportunity of watching such a case, in which the patient died of compression, very gradually produced by an increasing collection of pus between the bone and dura mater, and without any complication. The chief symptom was constant wearing headache, which prevented sleep. The man was pale and feeble, but not paralysed in any part, excepting that both eyelids drooped. He was rational, but spoke slowly, as if in a state of partial stupor. During the last two or three days of life he had convulsions; and finally, for twenty-four hours, he was in a state of increasing insensibility.

**DIAGNOSIS.**—It will be seen from what has been said above that the diagnosis of compression by symptoms is exceedingly difficult, and that the utmost use must in each case be made of the history of the case. In those of blood-compression after injury to a meningeal artery, there is almost always the fact that the patient between the date of the injury and the supervention of symptoms had an interval during which there appeared to be little or nothing the matter. This history is, if the symptoms have developed rapidly and without the signs of inflammation, by itself conclusive for diagnosis. Under such conditions trephining ought to be at once resorted to, or possibly it might be yet better practice to first tie the carotid artery.

The diagnosis of abscess in the brain-substance has already been discussed, and that of inflammatory collections from meningitis will be examined in its proper place. See **MENINGES, CEREBRAL, Inflammation of, Simple Traumatic.**

**TREATMENT.**—The treatment of compression of the brain is almost wholly surgical, and consists in the use of the trephine and knife to elevate depressed bone, or evacuate collections of blood or pus. JONATHAN HUTCHINSON.

**BRAIN, Concussion of.**—We class under the head 'Concussion of Brain' all symptoms which result simply from the

shaking, more or less violently, of the contents of the skull. It will be obvious, however, that most cases of severe shake of the brain are likely to be complicated by visible lesions. The skull may be broken and the brain may be contused, lacerated, or ecchymosed. It is highly probable, however, that well-marked and even serious symptoms may be produced by shaking only, and without the existence of any lesion discoverable either by the unaided eye or the microscope. We must further clearly understand that this element of concussion (*i.e.* the results of shake independent of lesion) enters into almost every case of injury to the head. Whatever be the other lesions, it is usually the fact that the brain has been more or less severely shaken. Thus it may easily happen in cases in which conspicuous lesions are present, such as fracture of the base or local contusion, that still the results of the shake are the most important. It might be convenient if we were in the habit of speaking of most cases of severe injury to the head as *Concussion plus other lesions*, with the endeavour to assign to each added complication its proper share in the general result. These explanations are necessary before we put the question—Can concussion alone cause death? Although it is highly probable that we ought to reply with a very confident affirmation, and to assert that it is not uncommon for concussion to be the chief cause of the fatal event, yet it is very difficult to prove it, since the cases are extremely rare in which severe concussion is produced without some attending lesion. The symptoms caused by concussion of the brain may be studied in very numerous cases of very various degrees of severity, which yet recover perfectly. From what is observed in these, we may infer as to the part which concussion takes in complicated cases many of which prove fatal.

The results of concussion may be divided into three stages. The first stage is that of *collapse*; the second, that of *reaction*, or of vaso-motor paresis, or, if named from its most prominent symptom, the *sleepy* stage; the third is that of *convalescence*, or recovery. The symptoms of the first stage, or stage of collapse, vary with the severity of the case; but if at all well-marked, consist in feebleness of pulse, pallor of skin, coldness of extremities, and dilatation of pupils. They may approach a condition which threatens immediate death. There is no stertor, for the respiration is too feebly performed. Although the collapse may be very great, the insensibility is rarely quite complete. It is of great importance, in this stage, to establish the negative as regards all forms of paralysis. If any non-symmetrical symptoms are present, the case is more than mere concussion. During this stage nothing should be done, except placing the patient in a condition of comfort, and preventing the cooling of the

body. Stimulants, unless the collapse is extreme, should be avoided.

After the collapse has lasted some little time (half-an-hour to two, three, or more hours), it begins to pass off. The patient moans, manifests discomfort, turns on his side, and draws his knees up. Very often at this period sickness occurs, and it is almost invariable if the patient's stomach was full at the time of the accident. Consciousness is now usually restored, and, by rousing, the patient may be induced to speak, and will tell his name, &c. Gradually, during a period of some hours, the case slides on into the sleepy stage. The pulse is now relaxed and full, the skin is warm or even hot, the face may be somewhat flushed, and the pupils are contracted. The patient is overpowered with sleepiness, and can only be awakened with difficulty. It is, however, always within possibility to awaken him, and he usually rouses himself to the calls of nature. Very commonly the pulse is irregular, especially if the patient be young. At this stage again care must be taken to ascertain whether there are any non-symmetrical symptoms—any weakness of a limb, of one side of the face, or of any single eye-muscle. If the patient passes his urine or fæces in bed, or if there is long-continued retention of urine, it is very probable that there is more than mere concussion—namely, laceration or contusion. The sleepy stage may last for a day or for a week, and it is in severe cases so well-marked that the patient's eyelids may be held open, and the pupils examined, without awaking him. During this stage the measures of treatment called for are spare diet, purgation, cold to the head, and quiet.

When the sleepy stage passes off, the patient is left weary, torpid, unfit for mental effort, and often with distressing headache. These are the symptoms of the convalescing stage, and they may last more or less for a considerable time. The patient should still be kept carefully quiet, no stimulants should be allowed, and purgatives should occasionally be used. Some of the symptoms present during the stage of convalescence may persist so long that they may rank rather as sequelæ. Thus there may be for years nervousness, inaptitude for business, liability to headache, and peculiar susceptibility to the influence of stimulants. As a rule, however, even after very severe concussion, no such ill-results are left, but the patient regains, after a time, perfect cerebral health. This remark must, however, not be held to apply to concussion when received in railway accidents; for in these cases there is a prospect of pecuniary compensation, and the sequelæ are often severe, prolonged, and very peculiar.

JONATHAN HUTCHINSON.

**BRAIN, Hæmorrhage into.**—SYNON.: Cerebral Apoplexy; Fr. *Hémorrhage cérébrale interstitielle*; Ger. *Hirnschlag*.

**DEFINITION.**—Escape of blood, by rupture of a vessel, into the substance or cavities of the brain. Hæmorrhage into the meninges is separately described.

Cerebral hæmorrhage is commonly due to the rupture of an artery, very rarely to the rupture of a vein. Occasionally, minute extravasations are caused by rupture of capillaries, and may constitute, by their number, an infiltration of the cerebral tissue with blood. This is seen in cases of venous thrombosis. Hæmorrhage from arteries or veins may also be due to their laceration by injury.

**ÆTIOLOGY.**—Arterial hæmorrhage is usually due to the coincidence of weakened vascular wall and increased pressure within the vessel. The causes of these states may be regarded as the conditions *predisposing* to cerebral hæmorrhage. Hereditary influence is sometimes distinctly seen, as a tendency to vascular degeneration, or to conditions which, as renal disease, produce such degeneration. Similarity of vascular distribution may also be inherited, and may determine the locality of strain, and, therefore, first of degeneration, and ultimately of rupture. Cerebral hæmorrhage is most frequent after fifty years of age, but occurs at any age, though rare during the first half of life. It is nearly twice as common in men as in women. It is said to be more frequent in temperate than in tropical climates, in winter than in summer, and at high than at low elevations. Certain acquired conditions act as predisposing causes. Chronic Bright's disease leads to early and extreme degeneration of vessels, as well as directly to hypertrophy of the heart and increased blood-pressure; hence it predisposes powerfully to cerebral hæmorrhage. In purpura and scurvy, cerebral hæmorrhage occasionally occurs, and also in pernicious anæmia and leukæmia. In the latter it is a not uncommon cause of death. In these blood-states the mechanism is doubtless impaired nutrition of the walls of the smaller vessels, which permits them to be bulged and thinned by a blood-pressure that may even be below the normal. The state of vascular repletion known as 'plethora' was formerly thought to be a frequent cause of cerebral hæmorrhage. It probably does aid other causes, but rarely co-exists with the more efficient, and so takes a subordinate position. Chronic alcoholism and opium-eating are said to promote vascular degeneration.

Among other remote causes must be reckoned heart-disease and syphilis, both of which commonly act by causing an aneurysm larger than those that are the special mechanism of hæmorrhage. Nevertheless, such aneurysms often escape even the most careful search. Each of these causes, so different in nature, acts by altering the wall

**BRAIN, Congestion of.**—See BRAIN, Hyperæmia of.

of the vessel—the one by a process of growth, the other by inflammation excited by imperfect obstruction with a plug from an inflamed cardiac valve.

The *proximate* causes of cerebral hæmorrhage are the weakened state of the wall of the vessel, and commonly some increase of blood-pressure. The vessel-wall is weakened by degeneration, and is often imperfectly supported in an atrophied brain. The increase of pressure within the vessels may be permanent, as in peripheral obstruction, with or without hypertrophy of the heart; or temporary, as in excited action of the heart, or impeded circulation through the lungs during effort. These causes are considered more fully in the article on BRAIN, Vessels of, Diseases of.

Hæmorrhage from a vein is rare, except as the result of laceration by direct injury, or of ulceration invading the vein from without. Varicose veins in the pia mater may sometimes rupture (Andral).

Considerable capillary hæmorrhage is usually due to the venous obstruction already mentioned. It may also result from cephalic congestion due to any cause, from capillary embolism, and from the blood-states which cause larger vessels to give way. In all these blood-states similar extravasations are often seen in the retina from the same immediate cause.

**ANATOMICAL CHARACTERS.**—In intra-cerebral hæmorrhage, the blood is extravasated into the substance or into the ventricles of the brain—into the ventricles usually by rupture of a previous extravasation within the cerebral substance. In the latter situation the blood occupies a cavity formed by laceration of the brain-tissue; rarely, when very minute and 'capillary,' by merely separating the fibres. In size, an extravasation varies from that of a pea or even smaller, up to that of the fist. The blood is found clotted, and reddish-black in colour; fragments of brain-tissue are mingled with it. The cavity containing it is often very irregular in shape; its walls are uneven, present projecting shreds of lacerated brain-substance, and are blood-stained and softened—at first by imbibition of serum, and later by inflammation. Many small extravasations are often seen in the neighbourhood of a larger clot. Usually there is only one large extravasation; sometimes, however, there are two or three. The extravasated blood exercises pressure: the convolutions are flattened; the falx is bulged to the opposite side (Hughlings Jackson, Hutchinson); and the rest of the hemisphere is anæmic. The effused blood may tear its way into the lateral ventricle; it then speedily distends both lateral ventricles and the third and fourth ventricles, and escapes by the openings at the lower extremity of the fourth ventricle, central and lateral, into the subarachnoid space. Or the blood may escape

to the surface, infiltrate the pia mater, and tear its way into the subarachnoid cavity, often by a very small opening. It is rarely that the artery from which the blood has escaped can be detected. Occasionally the extravasation can be traced to the rupture of an aneurysm of some size. In other cases miliary aneurysms may be found on many vessels. The larger arteries commonly present atheromatous changes, but these are only associated by being the result of some of the causes, and especially of age.

After a time the extravasated blood undergoes changes. The clot shrinks and gradually becomes, first chocolate, then brown, and ultimately a reddish-yellow; and it then contains chiefly fat-globules, pigment and other granules, and hæmatoidin crystals. The rapidity with which it undergoes this change is doubtful, and certainly varies. It is said that the distinctive blood-colour has disappeared as early as the twentieth day. Meanwhile the walls of the cavity undergo changes. The inflammation, in rare cases excessive and purulent, is usually conservative, and leads to the formation of connective tissue. A firm wall is thus developed, the inner surface of which becomes smooth by the softening and removal of the loose fragments of brain-substance; by this means a cyst is formed. It is said that connective tissue may extend across its cavity, and that in rare cases, the fluid being absorbed, the cyst-walls may unite, and a cicatrix result. Such cicatrices are, however, much more frequently due to softening than to hæmorrhage.

Hæmorrhage may occur in any part of the brain, but is more frequent in some situations than in others. The most frequent seat is the corpus striatum and the region just outside it: nearly half the intra-cerebral hæmorrhages are in this situation. Other primary seats, in the order of frequency, are the centrum ovale, the cortex, the pons and peduncles, the cerebellum, the optic thalamus (often affected by an extension of the hæmorrhage from the corpus striatum), the posterior portion and the anterior portion of the hemisphere. The frequency of hæmorrhage in the cerebrum is twenty times greater than into the cerebellum. The frequent extravasation into and outside the corpus striatum, is explained by the vascular supply. As the rupture is often near the edge of the lenticular nucleus the blood tears up the white substance; and this makes the frequency of hæmorrhage into the centrum ovale difficult to ascertain. See BRAIN, Vessels of, Diseases of.

Traumatic hæmorrhage occurs into and from a lacerated portion of brain, and is most frequently found on the surface, occupying mainly the middle of the convex portion of each convolution, and some other regions much exposed to injury, as the surface of the temporo-sphenoidal lobe, and the

under surface of the frontal lobe. Ventricular hæmorrhage sometimes results from traumatic rupture of a small vein on the surface of the corpus striatum (Prescott Hewett).

Soft tumours (especially gliomata) are sometimes the seat of hæmorrhage. The distinction from simple hæmorrhage (sometimes difficult) rests on their position being commonly one in which cerebral hæmorrhage is rare; and on some gelatinous-looking tumour-substance being found, into which hæmorrhage has not occurred, and which has characteristic microscopic features.

Other organs may be healthy, or present the changes which have been mentioned as predisposing causes; the lungs are usually secondarily congested, often intensely.

**SYMPTOMS.**—The occurrence of cerebral hæmorrhage is indicated by cerebral symptoms of two classes, the one general and more or less transient, the other local and more or less permanent. In addition to these there are sometimes premonitory symptoms; and commonly general symptoms, manifested by pulse, temperature, &c., which are secondary to the brain-lesion.

*Premonitory* symptoms, somewhat rare, are those of altered cerebral function due to local vascular disease, headache, vertiginous feelings, local weakness or numbness, slight mental changes, and slight affection of speech (*see* BRAIN, Vessels of, Diseases of; and BRAIN, Anæmia of). But the disease causing them is commonly distinct from that causing the hæmorrhage. The miliary aneurysms cause no symptoms, and hence it is only in the case of aneurysm of larger vessels that there are true premonitory symptoms. Hence they are less frequent than in cases of softening.

The *onset* of hæmorrhage is usually accompanied with apoplexy, *i.e.* loss of consciousness and of power of motion and of sensation, often with relaxation of the sphincters and loss of reflex action (*see* APOPLEXY, CEREBRAL). These symptoms are profound and lasting according to the size of the hæmorrhage and its position; being especially marked in large and double effusions, in intra-ventricular hæmorrhage, and in hæmorrhage into the pons. In a case of moderate severity they last only a few hours, and gradually pass away. In severer cases they may deepen until death occurs from failure of respiratory power. Death is rarely very speedy, life being usually prolonged for some hours even in the most rapidly fatal cases. In rare instances of hæmorrhage into the medulla, and also in meningeal hæmorrhage, death has occurred in five minutes, probably in each case from the rapid interference with the respiratory centre.

The temperature is at first lowered one or two degrees; the pulse becomes less frequent;

and the respiration slow. The Cheyne-Stokes respiratory rhythm often precedes death. After a few hours the temperature rises to the normal and in mild cases stops there, but in graver cases it rises above the normal two or three degrees. In some very grave cases the initial fall or subsequent rise may be extreme and go on until death occurs, sometimes reaching 90° in the one case, and 107° or 108° in the other (Bourneville). The urine is at first abundant, of low specific gravity, and acid in reaction.

In slight cases of cerebral hæmorrhage there may be no loss of consciousness. Vomiting in such cases is not infrequent. In other rare cases of large hæmorrhage, especially between the external capsule of the corpus striatum and the island of Reil (Broadbent), the loss of consciousness comes on gradually, after other symptoms, as of shock, for example, have lasted for an hour or two. These cases have been termed *ingravescent*.

*Local* symptoms, often permanent, and always of longer duration than the loss of consciousness, are present in all cases of circumscribed cerebral hæmorrhage, except in the rare instances in which, by its central position in the pons and medulla, it causes directly bilateral effects only, which are added to and intensify the general symptoms caused indirectly. In a unilateral cerebral lesion, the direct symptoms are unilateral loss of power of voluntary movement and often of sensation, accompanied sometimes with convulsion or rigidity. These local symptoms may commence a few minutes or longer before the loss of consciousness. They coexist with the apoplectic condition, and may often be recognised, even during coma, by the flaccidity of the paralysed limbs, which fall more helplessly than those of the opposite side; by inequality of the mouth and of the pupils; by conjugate deviation of the head and eyes towards the side of the brain injured; by convulsive movements; and, as the apoplexy clears, by the detection of unilateral defect of sensibility. The coma passing away, these signs become more distinct, and all the symptoms of hemiplegia remain, varying in intensity and extent according to the position of the lesion. The apoplectic state may recur after its disappearance—a grave symptom, usually indicating that a fresh hæmorrhage has occurred in the same or the other side of the brain, or more frequently that the blood has escaped into the ventricles. In the former case the unilateral symptoms, conjugate deviation of the head and eyes, &c., are increased on the same, or transferred to the opposite side; in the latter the unilateral symptoms disappear, and general powerlessness and deep coma supervene, with stertor, relaxation of the sphincters, lowered temperature, and impeded respiration. Death always ensues.

Convulsion may be a conspicuous symptom at the onset or subsequently. It is usually unilateral in its course or commencement, beginning on the side paralysed, rarely affecting only the non-paralysed side. In cases in which the blood escapes slowly, convulsions may occur on one side, and in the course of an hour or two may cease, the side at the same time becoming powerless. This is due to the motor structures, commonly those of the cortex, being at first irritated by the hæmorrhage, and afterwards interrupted or compressed so as to be incapable of function. Where convulsion is not met with, muscular twitching or rigidity may occur. General or widely-spread rigidity or twitching points to a bilateral lesion; if with coma, to ventricular hæmorrhage.

After a day or two symptoms of irritation about the cerebral lesion come on, such as headache, delirium, and rigidity in the paralysed limbs. During this period the temperature rises above the normal, and the pulse becomes quick. On their subsidence these symptoms, if the lesion is slight, may be scarcely recognisable, and a stationary period ensues, at the end of which recovery of power over the paralysed limbs begins. In slight cases power may be recovered very speedily. Its return depends upon the structural recovery of slightly damaged tissue, and on other parts taking on an increased function in compensation for that which is destroyed. The electric irritability of the muscles exhibits little change. Sometimes, however, when there is great irritation at the lesion propagated downwards to the cord, a marked initial increase in the irritability may precede a considerable depression, coincident with rapid wasting. Recovery of power is rarely complete except in those cases in which the area of damage is very small; and when the damage is large and affects an important motor region, there may be no recovery, loss of power persisting, commonly with more or less 'late rigidity' in the paralysed limbs. Slight permanent mental change often remains, and as the motor power is recovered, ataxic and other disorders of movement may supervene in the limbs which were paralysed, although much less commonly than after softening.

**DIAGNOSIS.**—The diagnosis of cerebral hæmorrhage rests on the symptoms of a localised cerebral lesion occurring suddenly. The conditions from which it has most commonly to be distinguished are—congestion of the brain; softening of the brain, embolic and thrombic; and sometimes tumour. For the distinction from it of other causes of apoplectic loss of consciousness, uræmia, post-epileptic coma, &c. *see* APOPLEXY, CEREBRAL.

From *congestion* the chief distinction of the cerebral hæmorrhage lies in the transient nature of the loss of conscious-

ness, and in the slightness and general character of the symptoms which characterise the former. Congestion generally, hæmorrhage only sometimes, comes on during effort; and the absence of history of effort is in favour of the latter. Similarly, the premonitory symptoms which are usually present in congestion are generally absent in hæmorrhage. The loss of motor power, and the symptoms of cerebral shock, are much greater in hæmorrhage than in congestion. It must be remembered that the two states frequently coexist. From *softening* consequent on *embolism* cerebral hæmorrhage has also to be distinguished. The subjects of the latter are usually of an earlier age than those of hæmorrhage; their vessels are healthy, but they have organic heart-disease, which is often grave. There may be evidence of embolism elsewhere, in spleen, kidney, or retina. Loss of consciousness is often absent in embolism, and the paralysis often comes on deliberately, and is almost always brief in duration. Deep apoplectic coma should always lead to a suspicion that hæmorrhage is the lesion, and this especially in cases of young persons with heart disease. It is important not to let the presence of heart-disease exclude the idea of hæmorrhage, because it is in such cases that the most severe forms of hæmorrhage are met with, when there is an aneurysm, due to embolism, and this has ruptured. From *softening* due to *thrombosis* the distinction is often difficult. Age, and the state of the vessels, no longer serve as guides. The presence of chronic Bright's disease is slightly in favour of hæmorrhage; atheroma, the cause of softening in late life, being also induced by it, but only to a slight extent. Excitement at the time of the onset suggests hæmorrhage; depression, physical or mental, suggests softening. The occurrence of previous hemiplegic attacks points to softening. In the attack, loss of consciousness is much more considerable, in proportion to the subsequent paralysis, in hæmorrhage than in softening. But the distinction on this ground is often very difficult, since loss of consciousness may be absent in slight hæmorrhage, and considerable in an extensive softening. A deliberate onset is in favour of softening; and so are much mental change, and early rigidity. A definite fall of temperature is generally caused by rupture of a vessel, and so also is a rise within twelve hours. Paralysis of sudden onset, in cases of *tumour*, may be ascribed to hæmorrhage, to which it is indeed sometimes, but not always, due. Usually, enquiry elicits a history of gradual, long-continued symptoms; intense headache and optic neuritis are strongly suggestive of a tumour into which a hæmorrhage may have occurred.

Hæmorrhage into the substance of the brain is distinguished from *meningeal hæmorrhage* by the pain and mental excite-

ment being less conspicuous, convulsion rarer, and by the presence of symptoms of a local lesion. *Hæmorrhage into the ventricles*, which resembles meningeal hæmorrhage in the generality of its symptoms, is usually distinguished by succeeding the symptoms of a circumscribed lesion.

**PROGNOSIS.**—During the attack itself the prognosis in hæmorrhage into the brain must be guided by the intensity of the symptoms, and by the place of the lesion, as far as that can be ascertained. Death is probable if the coma is profound, or has not begun to lessen at the end of twenty-four hours, and if the early depression, or the subsequent rise of temperature and pulse-rate, is great. Most cases are hopeless in which there is marked interference with respiration—even hiccough is a cause of anxiety. When the symptoms indicate ventricular hæmorrhage, or hæmorrhage into the pons, there is little chance of survival. In meningeal hæmorrhage with coma the prognosis is exceedingly grave. Consciousness being recovered, and the danger of immediate death over, the freedom from much secondary pyrexia, from lung-congestion, and from bedsores, is a favourable indication. The chance of recovery from the paralysis is estimated by evidence of position of the lesion, and by any indications of improvement. Early contraction of the flexors is unfavourable. The danger of recurrence is in proportion to the extent of vascular disease, and the existence of irremovable causes of increased arterial tension. Hence the prognosis is rendered unfavourable by advanced age, or chronic renal disease, and by the evidence of general premature decay.

**TREATMENT.**—*During the attack.*—Rest is the most important. The patient should remain, as far as possible, where he is seized; stillness must be secured; and all effort is to be avoided. The posture should be recumbent, with the head raised, and flexion of the neck should be avoided. Any cause of passive cerebral congestion, such as a tight collar, must be looked for and removed. Venesection was formerly almost always employed in such cases—certainly too universally, but is now quite discarded—perhaps too absolutely. Loss of blood lessens the force of the heart and vascular tension; it thus hastens the cessation of external bleeding. Doubtless it acts in the same way in internal hæmorrhage. It may be used with probable advantage if the arterial tension is great (that is, if the pulse is incompressible), the heart acting strongly, and there is reason to believe that the intracranial hæmorrhage is increasing. A small quantity of blood should be taken rapidly. In ventricular hæmorrhage, venesection is probably powerless for good. When this is the case, it may not be equally incapable of doing harm in other ways too obvious to be pointed out. Fashion should neither determine treatment

nor be forgotten as influencing the estimate formed by the friends of a sufferer. Venesection should not be employed where there is any evidence of failing heart-power. Its indications are drawn as much from the state of the patient, as from the fact of hæmorrhage. Bright's disease is no contra-indication.

If bleeding is not employed, the object to be aimed at by other measures must be to lower the arterial pressure within the skull, by keeping down the heart's action, by reducing the volume of the blood, and by diverting the blood as far as possible from the brain, by relaxing the systemic vessels, while endeavouring to obtain contraction of the cerebral vessels. Warmth may be applied to the limbs, aided by sinapisms. Dry-cupping to the surface, and purgatives, as croton oil or calomel, will divert the blood to the surface, or to the capacious intestinal vessels. Drugs which would cause contraction of the vessels are to be avoided, since their influence being on the smallest vessels and universal, their tendency is to increase arterial tension and hæmorrhage. To this, however, digitalis is an exception, since its action seems to be chiefly on arteries larger than those that burst. Diuretics are of great value, and their administration should never be neglected. Contraction of encephalic vessels may be furthered by cold to the head or sinapisms to the neck, according as the head is hotter or colder than normal. The heart should be allowed to fall a little below the normal in force, but failure of power must be warded off by stimulants given with great care. Convulsion is more effectually checked by cold than by bromides; the latter may be given if the convulsion recurs.

*After the attack.*—During the stage of irritation, rest must be maintained, and all sources of annoyance must be avoided. The bowels should be kept gently open by laxatives or by injections. Stimulants must be avoided, and the diet should be light. Pain may be relieved by cold to the head, a blister to the neck, or by Indian hemp, bromide of potassium, or a small dose of hyosine.

During reparation the diet must be nutritious, but carefully regulated; and constipation of the bowels must be avoided. Rubbing of the limbs, and their gentle exercise, will aid their recovery; and after the symptoms of irritation have passed, faradisation will improve muscular nutrition, and is especially indicated where rapid loss of irritability indicates probable wasting. Nerve tonics probably aid a little the recovery of slightly damaged structures: quinine and strychnine, or very small doses of phosphorus if there is no kidney-disease. In anæmia the syrup of the phosphate of iron is good. Warmth, change of scene, and cheerful mental surroundings are useful adjuncts, especially in the later stages of recovery.

W. R. GOWERS.

**BRAIN, Hyperæmia of.**—SYNON.: Congestion of the Brain.

**DEFINITION.**—Increase in the quantity of blood within the capillaries of the brain, other than that which is merely the early stage of inflammation.

Since neither the arteries nor the veins of the brain-substance can be over-distended with blood without capillary hyperæmia, and since it is to capillary hyperæmia that the functional disturbance of the brain is related, this may be justly taken as the essential pathological element in cerebral congestion. The congestion may be *active*, when the capillaries contain, in consequence of arterial distension or dilatation, an increased amount of oxygenated blood passing rapidly through them; or it may be *passive*, when, from venous obstruction, the increased blood in the capillaries is slowly moving, and is becoming, or is in great part already, venous.

'Congestion of the brain' is often alleged because no other explanation of symptoms occurs to the mind of the practitioner, although there may be no real evidence of such a state. Like 'congestion of the liver,' it is often adopted merely because it is a convenient refuge for a destitute diagnosis, and pleases the patient as an explanation which both satisfies and gratifies him. But it should never be assumed on purely negative evidence. Some descriptions of it have been written entirely from cases of hypochondriacal dysæsthesia.

**ÆTIOLGY.**—(A) *Active* congestion of the brain may be general or partial. Of the *general* form the *remote* causes are as follows:—Men are said to be more liable to it than women. Age increases its frequency, chiefly from the greater frequency of its causes; yet children, from the sensitiveness of their vaso-motor system, occasionally suffer from active cerebral congestion. Heredity has only an indirect influence. The plethoric condition, with its tendency to general overfilling of the vessels, is a powerful predisponent. The *immediate* causes of excessive flow through the arteries of the brain may be thus stated: (1) Increase in the blood-pressure—either general, from excessive action of the heart (as in extreme hypertrophy or functional overaction); or partial, from an obstruction elsewhere, throwing an undue proportion of the pressure upon the cerebral vessels. This is seen in contraction of the aorta beyond the origin of the vessels to the head, and in sudden contraction of a large number of the systemic arterioles, as those of the surface, in exposure to cold and in ague. In some states, as Bright's disease, its influence on organs is partially counteracted by the contraction of the smaller arterioles. It results similarly from all cardiac excitement, whether due to mental or physical causes. (2) Active arterial dilatation of vaso-motor origin may be excited

by prolonged mental work, severe emotion, insolation, digestive disturbances, or from the presence in the blood of various poisons, such as alcohol, and amyl-nitrite or nitroglycerine. In these cases the vaso-motor disturbance may precede and cause, or may succeed and result from, the overaction of the brain-tissue, which is intensified by it. It occurs in the second stage of many attacks of migraine. This cause may be conjoined with excited action of the heart, as in exophthalmic goitre. In acute alcoholic poisoning the cerebral congestion is, as Niemeyer suggested, secondary to the disturbance of brain-tissue, although the simultaneous flushing of the face makes this doubtful; in chronic alcoholism the congestion may be primary. In pyrexia the headache and delirium have been thought to be due to congestion, but more likely result from the blood-state. (3) Lessened atmospheric pressure may cause congestion of the brain, as in the 'Zenith' balloon-catastrophe; and it probably occurs when divers pass from the increased to normal pressure. (4) Gravitation in the recumbent posture may alone cause cerebral hyperæmia, or may powerfully aid other causes in producing it.

*Partial active* congestion of brain-tissue occurs chiefly from disease of the arteries, which perverts the local distribution of the blood-pressure; in organic brain-diseases, as tumour, hæmorrhage, &c.; after blows on the head; and probably also from local overaction of special centres. It may also be due to a slight degree and transient duration of the activity of the causes of local inflammation.

(B) *Passive* congestion of the brain, when *general*, is the result of impeded return of blood from the head. It is intense in death from all forms of suffocation or by hanging. It may be due to pressure on the veins in the neck, as by tumours or tight collars; pressure on the innominate veins by tumours or aneurysm; or obstruction to the circulation from violent respiratory efforts, as exertion, cough, or blowing wind-instruments. It may be caused by impediments within the circulation, such as tricuspid insufficiency and its causes in the lungs, or even by disease on the left side of the heart. The recumbent posture assists all these influences.

*Partial passive* congestion may occur from pressure on a cerebral vein, from local venous thrombosis, or from pressure by a growth on one of the cerebral sinuses.

**ANATOMICAL APPEARANCES.**—The capillaries are not visible to the naked eye even when over-distended, but with the microscope they are seen to be dilated, often to twice their normal calibre. Their distension is indicated by a deeper and reddish tint of the grey substance; and the fulness of the small arteries and veins shows itself in an increase, often very great, in the number and size of the red

points visible on section of the white matter. In active congestion the arteries are said to be distinctly larger than normal, and their perivascular spaces lessened in size; the minute vessels of the meninges are distended. Indeed in all forms of general congestion, the state of the vessels is generally more distinct in the pia mater than in the substance of the brain. Even in those, however, it is chiefly the congestion that would pass on to inflammation, or is really its first stage, that leaves distinct traces. In passive congestion the veins and sinuses are gorged with blood. It must be remembered, however, that such engorgement of the veins occurs in all cases of death from interference with the passage of the blood through the thorax, and that the vessels of the most dependent portion are always fullest. The state of the cerebral veins must therefore be carefully compared with that of the veins of other organs. Active congestion may sometimes leave no visible traces. After a time blood-pigment collects outside the vessels (Bastian), and serous effusion into the membranes and ventricles may be found in all forms. After a long-continued congestion, the vessels may be permanently distended; degenerative changes, consequent perhaps on the effusion of serum, may lead to the appearance of wide spaces around them; and the whole brain may have undergone distinct wasting. It must, however, be pointed out that the absence or paucity of post-mortem evidence is equally true of the skin, and does not establish the absence of congestion during life.

**SYMPTOMS.**—It is probable that many symptoms have been erroneously ascribed to cerebral congestion; some because hyperæmia, due to the mode of death, was found *post mortem*; others because an assumed congestion was the readiest explanation.

The symptoms commonly referred to cerebral congestion may be grouped in two classes—those of excitement, and those of depression. Either of these may exist alone; those of excitement may precede those of depression; or they may partially co-exist. They may be slight or severe; acute or chronic. In all cases they are increased by the recumbent posture or depressing the head, by expiration, coughs, and effort; and they are usually aggravated by constipation, and by indulgence in alcohol.

1. *General.*—In general cerebral hyperæmia, among the symptoms of excitement may be mentioned mental irritability; headache—slight or violent, with feelings of fulness or throbbing in the head, and vertiginous or other unpleasant sensations; increased or perverted functions of the organs of special sense, such as flashes of light and noises in the ears; contraction of the pupils; sleeplessness; restlessness, startings, twitchings or slight actual convulsions; and mental

excitement. The pulse is quick. There may be vomiting. The face varies, participating in the congestions of circulatory origin, and in some of vaso-motor disturbance, such as that which may occur during digestion. In other forms of supposed congestion of vaso-motor mechanism, as in those which result from excessive brain-work, the face may be pale, but the nature of these cases is doubtful.

Among the symptoms of depressed brain-function, are dulness of the special senses; motor weakness; mental indifference and slowness; somnolence, especially after meals; dilatation of the pupils; and infrequency of the heart's action. Consciousness may be lost suddenly, and the loss, it is commonly believed, may deepen into coma, but this is seldom absolute. As a rule there is no fever, but in children the temperature may be raised a degree or so.

In the *chronic* forms of cerebral congestion, these symptoms, variously grouped and moderate in degree, continue for days, weeks, or months. Their course is marked by great variability. Durand-Fardel has pointed out that in these cases there is often much viscid secretion of the conjunctiva.

In the more *acute* forms of cerebral congestion, the symptoms of muscular spasm, of mental disturbance, or of loss of consciousness, may be so predominant as to give a special character to the attack:—

*a.* In the *convulsive* form pain or uneasiness in the head commonly precedes the muscular spasm. The latter is usually deliberate in onset. It is said sometimes to be unilateral at first, and afterwards general, but more frequently it is general throughout. Tonic may precede clonic spasm, and the attack resemble an epileptic fit, or clonic spasm only may be present, or slight general tonic spasm. Consciousness may or may not be lost; if lost it is for a very short space of time.

*b.* The *delirious* form is seen under two aspects—(1) in old age, after emotional excitement; in this the wandering is slight, and often related distinctly to the recumbent posture, or to a low position of the head; (2) a much more violent delirium, which is apparently related to cerebral congestion, is seen sometimes after mental work or emotional excitement, or after alcoholic poisoning. It often resembles and may end in mania, but there is no pyrexia. Occasionally death results.

*c.* The *apoplectic* form is marked by sudden loss of consciousness, occurring commonly during effort. The unconsciousness usually lasts only a few minutes, and incomplete general weakness remains for a day or two. In rare cases the loss of consciousness deepens into coma, with stertorous breathing and relaxed sphincters, and death may occur from the extension of the cerebral depression to the respiratory centres.

*d. Vertiginous* sensations sometimes give a character to an attack.

In children congestion of the brain is said to be a rare but occasional cause of convulsion or delirium. Headache and contracted pupils make up a group of symptoms resembling meningitis, but fever is rarely present, and if it exists it is slight, and the symptoms usually come on suddenly and pass away in a day or two. Death, however, sometimes occurs soon after the onset of the attack, especially in the convulsive form. Periodical attacks of this character may change to those of migraine in later life.

2. *Partial*.—Partial hyperæmia leads to localised symptoms of excitement or depression of function. Local convulsion or paralysis may result. Headache is often intense and localised. If nothing more than congestion is present, the symptoms usually soon pass off.

**PATHOLOGY.**—The possibility of the variations in the quantity of blood within the brain has been denied on the ground that the cerebro-spinal circulation is carried on within a closed cavity. But the statement is untrue in the sense that is relevant to the problem at issue, since the communication of the venous sinuses of the skull, and especially of the venous plexuses of the spinal canal within the exterior, with external vessels, is free, and affords a ready mechanism by which the amount of blood, even in the distant cerebral vessels, may be varied by the displacement of the cerebro-spinal fluid. This, it will be remembered, can pass into the perivascular spaces, and can readily yield to increased intravascular pressure, and thus make room for an increase in the quantity of blood within the arteries or veins.

The detailed pathology of cerebral congestion is, however, still obscure, since we know little of the relative part played by the blood-vessels and the nerve-elements in determining the form which disturbance of function shall take. An excessive supply of arterial blood is, in all organs, attended with functional activity, and it is easy to understand that active congestion should result in symptoms of excitement. Subsequent depression of function has been accounted for by inferring compression of brain-tissue by effused serum. The reaction of exhaustion may contribute. In passive congestion the nerve-tissue is imperfectly supplied with oxygenated blood, and compressed by distended vessels, and its functions are impaired by the presence of effete products. Hence the predominance of symptoms of depression over those of irritation.

**DIAGNOSIS.**—The diagnosis rests on the discovery of circulatory and other causes of cerebral congestion; on the circumstances of posture, effort, &c., under which the symptoms came on; on the existence of concomi-

tant congestion in other parts supplied by the carotids, as the face (by no means invariable); on the general character of the cerebral symptoms; on their speedy disappearance; and on the absence, in the adult at any rate, of elevation of temperature. The diagnosis of the special forms of cerebral congestion from the diseases which they most resemble is considered under the head of those diseases. It may, however, be mentioned that, on the one hand, persistent focal symptoms exclude mere congestion, and that, on the other hand, the condition is likely to cause an error in cases in which the various cephalic sensations of pressure, fulness, heat, &c., which seem to the hypochondriacal sufferer suggestive of congestion, are generally pseudo-neuralgic in nature. It is in these cases that it is within the power of the doctor to do great harm by increasing the apprehensions of the patient, by encouraging the erroneous opinion and so increasing the influence of attention in augmenting the distress.

**PROGNOSIS.**—The prognosis is generally immediately favourable; but in a severe attack of any form, death may occur. The apoplectic form is most dangerous, the convulsive least so. Degenerated vessels increase the immediate risk to life. After many attacks, permanent nutritional changes in the brain often supervene.

**TREATMENT.**—The most important elements in the treatment of cerebral congestion are posture, removal of blood, purgation, cold to the head, and warm and stimulating applications to the surface. Whatever be the cause, it is important to raise the patient's head, so that gravitation may impede the flow and aid the return of the blood. By this means alone, insomnia from hyperæmia may often be prevented. The removal of blood is useful in extreme forms of congestion, either active or passive, especially in those forms of active congestion in which the face participates. Venesection or leeching may be employed, according to the severity of the attack; in active congestion the blood which is taken should be removed quickly. The relief which in such cases follows an epistaxis illustrates the value of this method of treatment; and we may take a lesson from nature and, in some cases, apply a leech behind the ear, where there are communications between the circulation inside and outside the cranium. It is not advisable in those cases in which, from overaction of brain-tissue, or from cold to the surface, dilatation of the cerebral vessels results while the surface remains pale. In all forms of congestion, purgation is useful. It removes from the blood some of its serum, and it affords immediate relief to the cerebral circulation, by causing an afflux of blood to the capacious intestinal vessels. In plethoric states, diuresis is also most useful, and has

succeeded where venesection and purgation failed. Cold to the head is of most value in reflex or secondary dilatation of the cerebral vessels, as after mental work, insolation, fatigue, and in some toxæmic states. In the same class of cases, stimulation of the peripheral nerves by sinapisms, blisters, &c., applied to the neck, will not only draw blood to the surface, but also, by reflex influence, assist in obtaining arterial contraction. Hot applications to the limbs act in a similar manner, causing local afflux of blood and thus lessening the tendency to encephalic engorgement. Recourse may also be had to mechanical appliances for increasing the quantity of blood in the limbs. This element in treatment is most useful in active congestion. Alcohol and opium must be avoided in all forms of active congestion, but in passive congestion they may be of service. Bromide of potassium is useful in those cases in which the congestion is produced by vaso-motor mechanism, excited either by stimulation of brain-tissue or of distant nerves. In passive congestion from heart-disease the treatment is that of the cardiac condition, digitalis, strophanthus, and diuretics being of especial service. All persons liable to congestion of the brain should live regularly, avoid hot rooms, and attend carefully to the stomach and bowels, relieving the latter by frequent moderate purgation.

W. R. GOWERS.

**BRAIN, Hypertrophy of.**—Hypertrophy of brain is a misnomer. True hypertrophy would consist of increase in number or in size of the nerve-cells, nerve-tubes, connective stroma, and supplying vessels; and there might be expected, as a result of this condition, some manifestation of a higher intellectual development. Such a condition is never found. A so-called hypertrophied brain is one that is larger and heavier than normal. On removing the skull-cap, the encephalon seems to expand, so as to render it difficult to affix the bone-covering in its place; the membranes are dry; the sulci have nearly disappeared, and the whole organ appears pale and bloodless. On section there is a sensation of toughness, though less in degree than in a case of general sclerosis. There is no sign of pressure upon the orbital plates, such as is met with in chronic hydrocephalus. The lesion affects only the cerebral hemispheres, especially on the convex surface, and perhaps the posterior lobes in particular. The base of the brain and the cerebellum are unaffected except by pressure.

On minute examination, the nerve-cells and nerve-fibres, far from being found augmented in number or in size, may even be compressed and diminished; and there is often also some interference with the normal calibre of the vessels. Gintrac, however, records a case in which the calibre of the nerve-tubes was almost double that of the normal. What in-

crease there is affects the white matter of the brain, and this structure is very pale and of an elastic consistence. The real and sole lesion is hyperplasia of the connective tissue. It differs from sclerosis in affecting the cerebral hemispheres more universally than is the case with sclerosis; and also that in sclerosis there is not only increase of the connective tissue, but subsequent retraction, and, as a frequent consequence of this, an absolute destruction more or less of the nerve-elements of the organ. When the disease is far advanced it may possibly cause absorption of the inner table of the skull, and thus produce a roughness or thinning of the bone, or, in extreme cases, even perforation. The sinuses are generally distended with blood. Hypertrophy of smaller portions of the brain is rare: still various cases are on record in which, under the name of neuromata of the nervous centres, white or grey matter has been found in certain parts of the brain over and above the normal constituents of this organ. Hyperplasia of the pineal gland is closely allied to glioma. It should be distinguished from encysted dropsy of that organ.

In most cases of so-called hypertrophy of a small part of the brain the lesion is an infiltrating growth.

**ÆTIOLOGY.**—Hypertrophy of the brain has been said to be sometimes secondary, and caused by the irritation of morbid growths. This, however, is rare. It is generally primary, and may be a disease of intra-uterine life; but it is generally developed after birth, especially in rickety children. Some forms of encephalocele, without hydrocephalus, seem to be due to the growth of compact masses of cerebral substance in excess of what is normal. The conditions for the production of this morbid state are infancy; bad diet; repeated congestion of the cerebral vessels, such as might be induced by frequent cough; and perhaps lead-poisoning.

**SYMPTOMS.**—The symptoms of so-called general hypertrophy vary according as the sutures are closed or not. If, from insufficient occlusion of the sutures, the head expands in proportion to the increase in size of the encephalon, the symptoms may be very slight. Children thus affected show no intellectual hebetude. In them, as long as the abnormality is uncomplicated with local inflammation of membranes or with hæmorrhages, there may be no headache, no affection of sight, no sensory or motor paralysis, and no convulsions. Convulsions, however, are common in cases in which the occlusion of the sutures has prevented expansion of the head proportionate to the internal increase of tissue. In such cases also there is generally more or less motor paralysis, often some anæsthesia of the limbs, headache, vomiting, and mental hebetude—symptoms,

in fact, either of marked interference with the intra-cranial circulation, or of irritation from inflammatory complications. The prognosis is always bad, but in rachitic cases the course may be chronic.

TREATMENT.—All treatment seems to be ineffective in diminishing cerebral hypertrophy.  
E. LONG FOX.

**BRAIN, Inflammation of.**—**SYNON.**: Encephalitis.—Encephalitis is a term which ought perhaps to be strictly limited to inflammatory changes in the brain-substance itself, to the exclusion of all forms of meningitis. It may be either *diffuse* or *local*, but for our present purpose we have chiefly to do with the diffuse form. Local encephalitis will generally result in abscess (*see* BRAIN, Abscess of), and will usually be met with, if we put aside the results of injuries, in association either with disease of the ear, with tubercular growths, or with pyæmic deposits.

It may perhaps be doubted whether the occurrence of diffuse inflammation of the brain-substance as an acute disease has as yet been proved, excepting as a result of wounds. Even as a traumatic lesion, its special features have by no means been accurately studied. It is, however, highly probable that, after penetrating wounds of the brain, its substance may inflame, just as the cellular tissue of a limb may, the inflammatory processes beginning at the site of the wound and rapidly spreading through a large part of the hemisphere. It is probably in the perivascular spaces that the process chiefly spreads, and it is in these that the microscope will detect the most abundant results. Such a condition of diffuse encephalitis may exist without there being any visible changes in the brain. It may perhaps be a little softened or a little congested, but very probably there is nothing about which the most experienced pathologist could feel certain until the microscope is resorted to.

**SYMPTOMS.**—It is not possible in the present state of our knowledge to speak with any certainty of the symptoms of diffuse encephalitis. They will vary, of course, with the region affected; and disturbance of function, followed by more or less complete loss, will be the most frequent occurrence.

**TREATMENT.**—When the symptoms of encephalitis are once recognised, it will usually be too late for treatment, and measures of prevention are those of chief importance. The early use of mercury, beginning in anticipation of symptoms, rather than waiting for them, is probably the most important; and next to it come cold to the head, purgatives, and counter-irritation.

JONATHAN HUTCHINSON.

**BRAIN, Laceration of.**—In the more strict sense of the word, the brain is but little liable to laceration from injury. Yet, in

connexion with injuries, such as penetrating wounds of the skull, fractures with great depression of bone, and even with violent concussions, the brain-substance is not infrequently, to some extent, torn. In so soft a structure, however, and under the influence of modes of violence which are usually rather of the nature of blows than of anything likely to cause stretching, we rarely meet with results comparable to laceration of any of the firmer textures of the body. Whenever the brain is 'lacerated' it is also contused, and the contusion often extends widely around the rent, and is by far the more important lesion. In the peripheral parts of the brain-mass this is especially true, and it is of little practical use to speak of lacerations excepting as complications of very severe contusions. In the central parts, the crura especially, we meet now and then with a laceration properly so called, and it is not very infrequent to find the trunks of single nerves torn across. The consideration of those forms of laceration which are produced by the effusion of blood from ruptured vessels of size sufficient to supply a stream forcible enough to break up the surrounding substance, will be found in the articles APOPLEXY, CEREBRAL; and BRAIN, Hæmorrhage into.

In the case of injuries to the head from falls or blows without perforation, certain definite parts are prone to suffer from contusion and laceration. Usually some slight evidence of injury is found immediately beneath the part of the skull upon which the blow was received, but by far the chief bruising will be at the opposite point. If the occiput be struck, the anterior lobes will be contused; and if one parietal eminence, the opposite sphenoidal lobe. This law, however, is greatly modified by the differing conditions under which different parts of the brain-mass are placed as regards their surroundings. In the posterior half of the skull the brain-mass is bulky, and between its hinder lobes and the cerebellum is a strong flexible membrane, well calculated to break vibrations gradually, and thus to prevent contusion. Nor are there in these regions any strongly marked bony ridges against which the brain might be dashed. These conditions are reversed as regards the anterior lobes and the middle lobes, and the consequence is that, whilst severe contusions are often seen in the latter, they are much more rare in the cerebellum and posterior two-thirds of the brain-mass. In cases of compound fracture, with tearing of the dura mater, and deep depression of bone, the brain-substance may, of course, be injured at any part; but even in respect to this kind of violence the hinder regions of the skull are specially protected.

**SYMPTOMS.**—We know enough of surface-laceration of the brain in parts other than the anterior and middle lobes, to be able to assert that, unless the lesion extend very widely or

deeply, it does not reveal itself by any special symptoms. If very extensive, weakness of the opposite limbs and side of the face is usually observed. Injury to the anterior lobes, unless extensive, cannot be diagnosed, but it may be guessed at in a few cases where the sense of smell is lost in one or both nostrils; for it is very common for the olfactory bulbs to be damaged at the same time. If the anterior lobes are severely lacerated, the symptoms will be those of very severe concussion, with the difference that insensibility is more nearly complete, and that it increases instead of diminishing as the days pass on. When a sphenoidal lobe is contused there is usually, according to the writer's observation, incomplete hemiplegia of the opposite side, involving sensation as well as motion, and the face as well as the limbs. From these symptoms the patient may, in the course of months, wholly recover. It is usually the apex of the sphenoidal lobe which is lacerated, but if the lesion extends higher, and if it occurs on the left side, aphasia may be present.

In connexion with recent research (Dr. Hughlings Jackson, Dr. Ferrier, and others) as to localised functions, we are now able to diagnose with considerable accuracy the precise regions injured.

**TREATMENT AND PROGNOSIS.**—In the treatment and prognosis of lacerations and contusions of the surface of the brain, much depends upon whether or not the case is complicated by compound fracture and the admission of air. If air have been admitted there is risk of meningitis or encephalitis, denoted in either case by the occurrence, within a few days of the injury, of hemiplegia of the opposite side. To prevent this must be the object of treatment. The scalp should be shaved, the wound closed with sutures as far as practicable, and lint wetted in a spirit-and-lead lotion should be laid over the part and systematically re-wetted every hour. If the case be treated in hospital it may be well, in addition to this, to wash the wound with the lotion before closing it, or to dress with Lister's antiseptic precautions. In cases of laceration without access of air, death may ensue from diffuse softening around the part. If this happens the case will probably end within a week or ten days. It is probable that many cases of fractured base with more or less severe laceration of brain recover; in some with permanent paralysis, but in others without. It must be added that many of the cases in which death occurs within a few hours or a day or two after fracture of the base are attended by laceration. In these the symptoms are often difficult to distinguish from those of compression. Profound insensibility, a bloated face, stertorous breathing, and a full pulse, are often present; but they may be substituted by pallor and a feeble pulse in connexion with

great depression of the heart's action. If any deviation from symmetry as regards the paralysis of the limbs can be proved, it is in favour of laceration and against compression, but the differential diagnosis is a matter of extreme difficulty.

**Laceration of Cranial Nerves.**—Lacerations of single nerves within the cranial cavity are not by any means uncommon. This occurrence is to be suspected whenever the parts supplied by a cranial nerve are completely paralysed, without accompanying symptoms indicative of severe lesion of the brain-mass. Cases of laceration of the brain itself may be complicated by laceration of nerve-trunks, and thus the symptoms may become difficult to interpret with confidence.

Of single cranial nerves the olfactory bulbs are the most liable to suffer from contusion; and the third, fourth, and sixth nerve-trunks are those most frequently torn through. JONATHAN HUTCHINSON.

**BRAIN, Malformations of.**—The malformations of the cranium and its contents may be divided into two series:—A. Those which are scarcely compatible with life; and B. Those in which life is possible, although the intellectual power may be more or less modified from a healthy condition.

A. The *first series* will include at least seven forms, in all of which life is so rare that it is impossible to speak of more than the pathological anatomy.

1. *Dicephalia*—in which two heads are found upon a single body, or upon two bodies pretty extensively connected. In the first variety, one head may be attached to the vault of the palate of the other, or may be united to the convexity of the skull. In the second variety the heads may sometimes spring from a single neck. This dicephalous condition is frequently accompanied by malformation affecting the spinal column and spinal nerves, as well as by some incompleteness in the development of the brain.

2. *Monocephalia*—the union of two heads into one, on two separate bodies. The two cranial cavities are united into one. Dissection of the dura mater points to this membrane having been formed out of two, and in like manner the cranial contents are either double, or appear to be single from the union of double organs.

3. *Acephalia*—the complete absence of head. An acephalous monster is usually a twin; and when this is not the case, it is associated with the morbid condition of the uterus of the mother known by the name of uterine hydatids.

4. *Paracephalia*—the head not entirely wanting, but deprived of most of the cranium and of the face. A monster of this kind is generally a twin.

5. *Anencephalia*.—The absolute meaning

of this term would be the absence of all cranial contents, but it is made to include certain varieties, differing according to the amount of the encephalon developed. The aspect of the head, resembling that of a frog, the considerable projection of the eyes, the flattening of the forehead, and the absence of the cranial vault, are the chief characteristics of this abnormality.

In the first degree, there is absence of cerebrum, cerebellum, mesocephale, and spinal cord. In cases of this kind the cranial vault is generally absent, and the bones at the base of the skull are convex and thickened.

In the second degree, the cerebrum, cerebellum, and mesocephale are absent, but a portion of the spinal cord is found. This portion of cord is most usually the lower part.

In the third degree, the spinal cord is pretty complete, but there is still an absence of the cerebrum, cerebellum, and mesocephale.

A few cases have been recorded of the fourth degree, in which no cerebrum or cerebellum is found, but a normal spinal cord, and a pretty complete mesocephale.

In the fifth degree, the cerebrum alone is entirely or almost entirely absent, whilst the rest of the nervous centres are present, though not always in a perfectly complete condition. The seat of the absent cerebrum is often filled by fluid.

Lastly, one case has been recorded in which the cerebrum was present, whilst the cerebellum, mesocephale, and spinal cord were wanting.

Anencephalia, like the other previously mentioned malformations, is due to arrest of development, such arrest depending either on physical injuries to the uterus at a very early period of pregnancy, or to some mental shock experienced by the mother during the first two months after conception. It differs from acephalia, not only by the partial formation of the head, but by the presence of the heart, and other thoracic organs. The ganglia of the sympathetic are usually well-developed.

6. *Pseudencephalia*.—In this malformation there is anencephalia plus a very considerable thickening of the meninges, which take the place and often imitate the aspect of the brain. Its varieties exactly correspond to those of anencephalia. The tumour formed by the development of the membranes is of variable size and position. It may be frontal, fronto-parietal, or occipital. The real seat of the lesion is the pia mater. The abnormality consists in extreme hypertrophy of this structure, with complete arrest in the development of the encephalon, or of some portion of it. Several observers have recognised certain vesicles in the interior of the membranous tumour, and these have been supposed to be cerebral cells in process of development. It is more in accordance with observation to believe with Gintrac that they

are connected with the development of the choroid plexus.

7. *Cyclocephalia*.—In this monstrosity there is an approximation or actual fusion of two eyes in a common orbit. It is connected with certain abnormalities in the brain, that militate against viability, or at least prolongation of life. The brain itself is generally more or less deficient, especially in its anterior and central portions, and in some cases the nose and mouth are very ill-developed.

B. The *second series* of cases owe their abnormal conditions to injury arising in the course of foetal life; and some forms at least may be due to lesion occurring at a later period than in the first series.

8. *Atelencephalia*—incompleteness of brain or of membranes—is the chief of these forms. This incompleteness manifests itself in seven varieties, according to the part of the encephalon injured by the lesion.

In the first variety, the dura mater is somewhat deficient, being altogether absent in certain situations at the base of the brain. The falk cerebri may be wanting; or from incomplete development it may seem perforated with holes; or the tentorium cerebelli may be absent. There are no symptoms which allow a positive diagnosis of any of these lesions during life.

In the second variety, there is general incompleteness of the brain, or imperfection of several portions of it at one and the same time. Whilst the cranial vault is thrown back, and the lower jaw is short, the base of the skull is large, the cerebral convolutions almost absent, and the cerebellum large. The head is almost always small, and it may present various irregular forms. This coincides with certain internal lesions, partial or general atrophy with consequent serous effusion under the membranes, inflammatory conditions, or thickening of the cranial bones and of the meninges. The incompleteness of the brain varies exceedingly, from a condition in which the whole brain is atrophied, to spots of deficiency, such as the absence of a single convolution, of the septum, or of the pineal gland. The symptoms will necessarily vary much according to the amount of cerebral incompleteness. Where this is general, affecting in some degree all the convolutions, the intellectual powers, as in the microcephalous Aztecs, will be very slightly developed, and their language of the very simplest form. With the brain still more imperfect, there is generally complete idiocy, or a condition closely allied to it. The special senses are dulled, particularly sight and hearing. Speech is in abeyance, or is limited to monosyllables. There are various motor phenomena, such as muscular debility, hemiplegia, paraplegia, contraction, convulsion, loss of power over sphincters, dysphagia, vomiting, or feebleness of respiration.

The third variety includes incompleteness

of the central parts of the brain. The corpus callosum, the septum, the fornix, the corpora striata, and the cornua ammonis may be imperfectly developed. The cerebral hemispheres may thus be in some sort fused together, and the shape of the ventricles altered. The symptoms differ from those of the preceding variety, in that the special senses are seldom involved; and that, although complete idiocy may accompany these lesions, it is more usual for the brain to be found capable of some slight intelligence, though unequal to the conception of abstract ideas.

In the fourth variety, the lateral portions of the brain are incomplete. This lesion generally occupies one side of the brain, leaving the other hemisphere intact. Several points in the hemisphere may be affected, or the whole of a single lobe. Most usually there is a depression occupying the seat of one or more convolutions. Such a lesion occasionally attacks the whole hemisphere, giving it the appearance of a large pouch filled with fluid. Sometimes also the neighbouring ventricle communicates with it; or there may be much ventricular effusion, with imperfect development of the corpus striatum, the optic thalamus, the cornua ammonis, the mammillary tubercle, the crus cerebri, and the optic and olfactory nerves of one side. In a considerable proportion of patients so affected, the lesion is accompanied by idiocy, and possibly the inability to speak is connected with this mental condition. Some few patients, however, possessed with some intelligence, have yet been unable to speak; this has been the case even when the lesion has existed on the right side. Deafness is rare; feebleness of sight, various forms of strabismus, and nystagmus common. Very frequently there is hemiplegia of the side opposite to the lesion, and certain other affections of the limbs—emaciation, incomplete development, contraction, various deformities of the hands, &c. Sensation even in the paralysed limbs is normal; convulsions are not uncommon.

In the fifth variety, there is incompleteness of the anterior portion of the brain. Here both the anterior lobes are affected together. This condition may be associated with some deficiency of the corpus callosum, fornix, and corpora striata. Idiocy is not uncommon; mutism is the rule, but in some patients not idiotic a few words have been possible. Other phenomena—amaurosis, strabismus, and various motor abnormalities—have occurred so irregularly in these patients that it is probable they were symptoms not so much of this lesion as of certain further complications.

Incompleteness of the cerebellum forms the sixth variety. This is sometimes associated with a similar condition of one side of the brain. Usually one lateral lobe only is

affected. General hydrocephalus is an occasional complication, and a collection of fluid under the tentorium cerebelli is very common. The symptoms are very negative. In general terms it may be said that there is no loss of muscular co-ordination, and no loss of sexual power.

In the seventh variety, there is incompleteness of the mesocephale and medulla oblongata. This is not carried very far. Certain modifications in form and volume are alone compatible with the preservation of life. It is not a common lesion, and has generally been associated with idiocy.

9. *Congenital hydrocephalus*.—One variety of this congenital effusion of fluid is rare, viz. when the fluid is outside the dura mater, between this membrane and the pericranium, and the cranial bones are found floating in the midst of the fluid.

The second variety is that in which the fluid lies outside the brain. The writer believes that serous effusion in this position is not the cause of the atrophy, flattening, or induration of the subjacent cerebral organs, but the effect: that where fluid is found in this situation it is only the consequence of some one of the lesions already mentioned notably atrophy of brain from whatever cause, and of atelencephalia. This view is, however, opposed to that of some authors.

The third variety is congenital hydrocephalus of the ventricles. In some such cases the increase in the size of the head occurs before birth; in others, not until after. The head increases rapidly in size in the first four weeks after birth. The sutures are widely separated, the cranial bones very thin, the integuments of the head injected, and the hair deficient. The muscles are badly developed, locomotion is imperfect, the intellect is generally obtuse, but the special senses are not particularly affected, unless it be that sight is deficient.

10. *Synencephalia* is merely a matter of pathological interest. The head of the fœtus is sometimes found adherent to the membranes or to the placenta, as a consequence of intra-uterine inflammation. At the point of adhesion the place of the cranial bone is taken by a thin vascular membrane. This condition is sometimes accompanied by, and indeed perhaps causes, encephalocele.

11. *Exencephalia*.—Here a large portion of the brain is situated outside the cavity of the cranium. Practically it includes all the other malformations of the brain that are yet to be spoken of. Thus, if only a limited portion of the brain finds its way outside the skull by an abnormal opening, the displacement is known by the name of *encephalocele*, or *hernia cerebri*; if this hernia coincide with a hydrocephalic condition of the ventricles, it is called *hydrencephalocele*; and if the hernia is composed not of the brain, but of the membranes, distended with

fluid it may be, the lesion is called *meningocele* or *hydromeningocele*. Exencephalia proper may be subdivided into *frontal*, *sincipital*, and *occipital*, according to the direction taken by the extruded brain.

In encephalocele only a portion of the brain, more or less limited, is found outside the skull. The exit takes place most frequently at the occipital, and next in the frontal region; but the temporal and parietal regions are occasionally the seat of this lesion. The symptoms may be very negative. Encephalocele, unless pressed upon externally, is not often attended by convulsions or paralysis, by intellectual feebleness, or by difficulty of speech. This latter symptom is sometimes found when the hernia includes the cerebellum. Neither is this lesion incompatible with the prolongation of life. The chief diagnostic difficulty is the possibility of the tumour being cephalæmatoma, but this is frequently situated over the parietal bones, an unusual position for encephalocele: and external pressure of the former tumour causes none of the cerebral phenomena—stupor, dilatation of pupils, paralysis, convulsion, so constantly seen from compression of an encephalocele.

E. LONG FOX.

**BRAIN, Malignant Diseases of.**—*See* BRAIN, Tumours and New Growths of.

**BRAIN, Membranes or Meninges of, Diseases of.**—*See* MENINGES, Diseases of.

**BRAIN, Morbid Growths of.**—*See* BRAIN, Tumours and New Growths of.

**BRAIN, Œdema of.**—DEFINITION.—Infiltration of the brain and pia mater with serum.

ETIOLOGY AND PATHOLOGY.—In chronic maladies attended with general œdema, especially Bright's disease, there is an increased amount of fluid around the brain—in the meshes of the pia mater and between the convolutions—and also in the ventricles. Occasionally the cerebral substance is infiltrated; but this is uncommon, and it probably occurs only when there is some change in the cerebral structure. The perivascular canals normally afford a ready means of escape for effused serum, and in Bright's disease, at least, the substance of the brain is often remarkably firm, so as to present a contrast to the condition of other organs. But apart from any general disease, all spaces that result from a diminution in the volume of the structures within the skull, are filled by serum. Thus in senile atrophy of the brain, the space between the shrunken convolutions is occupied by serum, and the ventricles contain an excess of fluid. The brain-tissue may also appear to contain more fluid than usual, in consequence of the presence of serum in the enlarged perivascular canals. Such effusion is also met with

in those forms of insanity in which there are degenerative changes in the brain with a diminution in its volume. It is seen also in cases of rapid course, in which functional excitement is attended with some vascular disturbance. Conditions of hyperæmia and inflammation are attended with effusion of serum in the brain, as in other organs, and it is in cases of inflammation that the greatest amount of œdema is met with, but this condition is not included in the common use of the name. In passive congestion, however, such as results from heart-disease, enough serum may escape from the distended vessels to give rise to a condition of œdema; but it is probable that when the excess is more than trifling, room is made for it by wasting of the nerve-elements, the result of the continued pressure to which they are exposed.

When the degree of œdema is considerable, the cerebral substance may be enlarged, the convolutions being flattened; and the tissue is lessened in consistence owing to the infiltration of the cerebral tissue by the liquid, and the separation by it of the nerve-elements. A microscopical section shows empty round and oval spaces between the cells and fibres, limited by delicate tracts of the separated neuroglial tissue. The same softening is seen in the neighbourhood of effusions of fluid into the ventricles; the brain-tissue, for a depth of some lines from the ependyma, being softened to a pulpy consistence. Post-mortem imbibition always increases the apparent amount of the œdema and the degree of the softening, because the nerve-elements quickly begin to break up after death. In simple œdema the tissue remains pale, and the grey substance may be even paler than usual.

SYMPTOMS.—Little is known of the symptoms of œdema of the brain. The pathological state is usually secondary to some other condition, the symptoms of which mask any that the œdema itself might possibly cause. General œdema seems attended by slow diminution of mental power and motor force, which may be in part due to the patient's state, but has many other possible causes. The effusion of serum in cases of congestion, and consequent pressure on the nerve-elements, has been considered as the cause of the symptoms of depression common in that condition. Cases occasionally occur in which effusion of serum into the ventricles and the pia mater is the only post-mortem condition to be found after an apoplectic seizure, and such cases are often spoken of as instances of *serous apoplexy*. In so far as the effusion of serum is sometimes possibly related to the apoplectic attack, it can be merely as the consequence of a cerebral congestion which has left no recognisable post-mortem hyperæmia. In point of fact we know nothing of symptoms that can be ascribed to the œdema itself, and it is not

easy to conceive a mechanism by which symptoms can result from it, so far as we are acquainted with its intimate pathology.

**TREATMENT.**—The treatment of cerebral œdema is usually secondary to the condition, commonly conspicuous enough, which is its cause—Bright's disease, passive cerebral congestion, &c. If œdema be suspected where no causal indication for treatment exists, purgatives and diuretics, with iron if there be debility, are the remedies most likely to be of service. But where a diagnosis is speculative, treatment necessarily lacks a confident basis.

The effusion of fluid into the ventricles is described under **HYDROCEPHALUS**.

W. R. GOWERS.

**BRAIN, Sclerosis of.**—See **SPINAL CORD, Special Diseases of: Multiple Sclerosis**.

**BRAIN, Softening of.**—**DEFINITION.**—A pathological state of brain-tissue, attended with diminished consistence; usually local; and indicated, during life, by mental, motor, and sensory symptoms, which vary according to the seat of the lesion. It is produced rapidly in the vast majority of cases, and is then usually dependent on vascular obstruction. Very rarely it is chronic in development, and the nature of the actual process is not known. The following description applies to the acute form.

**ÆTIOLOGY.**—Acute softening of the brain, occurring during life, and not due to traumatic cause, is referable to one of two conditions, inflammation or vascular obstruction. Most cases were formerly thought to be due to inflammation; but it is now known that very few are. Inflammatory softening is described in another article (*see* **BRAIN, Inflammation of**). The vascular obstruction, which is the usual cause of softening, may be arterial or, rarely, capillary. Venous obstruction also causes softening, but the loss of consistence does not involve the whole of the tissue of the part, and enough remains to permit an ultimate restoration of firm tissue and even the production of induration. The arterial obstruction may be due to a coagulum formed *in situ* (thrombosis), or to a plug of fibrin conveyed to the spot by the blood-current (embolism). The chief predisposing and exciting causes of softening of the brain will therefore be the causes of these conditions (*see* **BRAIN, Vessels of, Diseases of**). The common concomitant conditions are—in thrombosis, vascular disease; in embolism, valvular disease of the heart; and as predisposing conditions we usually find—in cases of thrombosis, advanced age, Bright's disease, chronic alcoholism, gout, or syphilis;—in cases of embolism, acute rheumatism, chorea, or scarlet fever. Senile vascular degeneration is the most common cause of softening, and hence the disease is met with most frequently in the old. In rare cases, thrombosis occurs

without disease of the vessels, from a state of the blood alone. It is probable that this is sometimes a cause of hemiplegia coming on a few days after child-birth, when there seems to be a physiological increase in the coagulability of the blood. Embolism, due to valvular disease of the heart, and thrombosis due to syphilitic disease, are the most frequent causes of acute local softening in the young and middle-aged. All these ætiological conditions are considered in more detail in the article on Diseases of the Vessels of the **BRAIN**.

**ANATOMICAL CHARACTERS.**—The characteristic feature of cerebral softening is diminished consistence. This may, however, arise from either ante-mortem or post-mortem changes. In each case the diminished consistence depends on the breaking-up of the material, of which the nerve-elements are composed, into globules and granules, and the separation of these by an increased quantity of fluid. Thus the continuous structures of which the brain consists are broken up into disconnected fragments, and the consistence of the tissue is accordingly diminished. In post-mortem softening there is nothing more. The globules of myelin are often large, and the separating fluid abundant. The softened tissue has the tint of the normal cerebral substance. The process is the result of the imbibition of fluid from some collection of serum, in the ventricles or elsewhere, and occurs in the greatest degree in the immediate vicinity of this. In ante-mortem softening there are, in addition, certain changes in the tissue-elements. The process of segmentation of myelin results in the formation of finer granules. These are in part aggregated into 'granule-corpuscles,' round or oval masses of globules and granules, sometimes contained within a distinct cell-wall. Some of these bodies may arise by simple aggregation, others by the degeneration of connective-tissue cells, and some by the aggregation of products of nerve-degeneration within cellular elements of various kinds. The walls of vessels in the softened area also present fatty degeneration. No further change may exist, and the area affected may present simply a diminution of consistence, its colour remaining unchanged. It is then called *white* or *grey softening*. Very frequently, however, in the part thus diseased, distension of capillaries with blood occurs, most considerable in the periphery, and blood is actually effused, chiefly by rupture of capillaries, in trifling degree perhaps by migration of corpuscles. In proportion to the amount of blood mingled with the softened brain-tissue, the colour of the affected area is changed, and thus *red softening* is produced when the amount of blood is considerable. In the change which results from closure of a vein, the tissue is crammed with punctiform extravasations, and a purplish colour results;

but, as already stated, there is not the general diminution of consistence that brings the alteration into the category of 'softenings.' After a time, the blood effused in red softening degenerates, its tint becomes altered to yellow or orange, and *yellow softening* is produced. Ultimately, it is said, the colour, if at first moderate, may be removed and white softening result, but the pigment that gives rise to the yellow tint may remain for many years.

*Red softening* is found chiefly in the grey substance, where the vessels are numerous, especially in the cortex and central ganglia. The tint varies; the red colour is usually punctiform, or mingled with yellow and white. If the extravasations are large and numerous, 'capillary apoplexy' results. According to the amount of effusion of serum and blood there is swelling, and the diseased area may project above the cut surface. Inflammatory changes result from the vascular distension, and in proportion to these, increase in the nuclei of the neuroglia is found, especially at the circumferential portions. From this cause, and from the migration of white corpuscles, pus-like cells appear among the products of degeneration in varying quantity. The vessels are dilated, and may present a moniliform appearance. Their perivascular sheaths are often distended with blood.

*Yellow softening* results from red softening, by degenerative changes in the blood effused attended with a change in its pigment. It has a similar seat, being frequently met with in the convolutions, where it constitutes the *plaques jaunes* of the French. Its consistence is usually slight, its aspect granular. The colour depends on the presence of minute pigment-granules, diffused colouring matter, and hematoidin crystals.

*White softening* has the tint of the normal cerebral substance. In consistence it varies: it may be only a little below that of the cerebral substance, or it may be diffuent. Its aspect is uniform, or white flakes are scattered through it. The limits are usually gradual. Under the microscope it presents the detritus of nerve-elements, a few nuclei from the connective tissue, granule-corpuscles, and, ultimately, corpora amyloidea. White softening is chiefly found in the white substance of the hemispheres. It occasionally has a gangrenous odour, and then may be found in the white or in the grey substance; probably this form results from the obstruction of capillaries by septic material. It is theoretically probable that when softening is white from the first, the capillaries are the seat of the primary obstruction. It occurs also in the zone of cerebral tissue in which meet the regions supplied by the arteries of the cortex and of the central ganglia.

*Ultimate changes.*—White and yellow softening may remain for years unchanged. Usually the products of disintegration of the nerve-elements are gradually removed, so

that a cavity remains, across which bands and trabeculae pass—the remains of vessels and other structures that have escaped total necrosis. Sometimes the changes in the elements of the neuroglia and the extravasated white corpuscles, &c., result in the formation of a considerable quantity of connective tissue, consisting of fine fibre-cells and fibres, most abundant in the margins of the softened area, which become firm and dense, whilst the trabeculae of the connective tissue, crossing the cavity, are thickened in like manner. After a time, when the amount of this tissue is considerable, the fluid may be absorbed, and a sort of cicatrix result from the union, partial or complete, of the walls. In other cases the walls alone are thus altered, the solid particles are removed from the softened tissue, and a cyst is formed. The outer portion of the cyst or cicatrix may be limited by a zone of dilated blood-vessels, presenting, under the microscope, a peculiar and quite characteristic feature.

*Seats of softening.*—There is no part of the brain in which softening has not been found, but its most frequent seats are the cortex, the corpus striatum, and the optic thalamus. In the pons Varolii and medulla it is also frequently found, but it is rare in the cerebellum. Its frequency in the medulla is underrated because only minute areas of softening are compatible with a duration of life sufficient to permit them to assume a characteristic aspect. Its occurrence, position, and characters depend on the distribution of the vessels. The small arteries of the corpus striatum and optic thalamus are 'terminal arteries,' having only capillary communication with other vessels, insufficient for a collateral circulation adequate to maintain the vitality of the tissue. The arteries to the cortex of the brain are sometimes terminal, but sometimes possess anastomoses by arterioles with other branches. Hence obstruction in the central arteries leads invariably to softening, while obstruction in the superficial arteries may also cause softening (which involves the grey substance of the convolutions and some of the subjacent white centre to which the vessels penetrate), but often the anastomoses of the superficial vessels are so free that softening does not result. An obstruction of a main trunk (as the middle cerebral) may lead to softening of the central region (corpus striatum), while the convolutions escape; but usually both suffer. For the same reason, the softening of the cortex is apt to be irregular in distribution, and partial even within the region of the obstructed artery, a feature considered further in the article on Diseases of the Vessels of the Brain.

*Symptoms.*—The *premonitory* symptoms of softening of the brain depend upon its mechanism, and are considered in greater detail in the articles describing its causes. In em-

bolism other symptoms than those of cardiac disease are usually absent. Occasionally a slight attack of loss of cerebral function, due to a slight embolism, may precede a graver attack. In softening due to arterial disease, premonitory symptoms of local cerebral anæmia are frequently present; these may exist for months before the onset, or only for a few days, or even for a few hours. There is also, in many cases, sufficient disease to impair the nutrition of many parts of the brain, revealed by symptoms of wide range—mental deterioration, numbness, and pains in the limbs, pain in the head, or slight local weakness. These symptoms are of especial significance when associated with evidence of degeneration elsewhere in the arteries; with the conditions—as chronic Bright's disease, alcoholism, and senility—in which atheroma of the cerebral arteries is common; or with constitutional syphilis.

The symptoms of *actual* softening are those of loss of function in the damaged portion of the brain. Strictly, indeed, these symptoms are those of the anæmia that causes the softening, and which, depriving the brain-tissue of the material for its functional action, arrests the latter with a rapidity which seems to suggest that other influences may co-operate in the arrest. The onset of the symptoms may be actually sudden, as in embolism, and sometimes in thrombosis; or it may be gradual, as occasionally in thrombosis. In the former case, the symptoms of initial shock are added to those due to the structures involved (*see* CONVOLUTIONS OF THE BRAIN AND CORTEX CEREBRI, Lesions of; &c.). Hemiplegic symptoms and mental disorder are the most common. Hemiplegia especially occurs in embolism, on account of the frequency with which the middle cerebral artery is obstructed, and of the important motor regions (corpus striatum and motor parts of the convolutions) to which that artery is distributed. From the distribution of the artery to the lower frontal convolutions and adjacent region, aphasia is frequently present when the obstruction is on the left side.

When the symptoms come on suddenly, they often occur after some fatiguing exertion, or during exhaustion from any cause. If the area damaged be extensive, there is loss of consciousness, and there may be all the symptoms of an apoplectic seizure. The loss of consciousness is rarely profound, and the symptoms of apoplexy soon pass off. In the most severe cases, however, they may deepen to fatal coma, especially when an important artery is occluded in each hemisphere. Thus the closure of both middle cerebrals causes symptoms indistinguishable from those of ventricular hæmorrhage and equally fatal in ultimate result. Symptoms of irritation commonly succeed those of apoplexy as the collateral hyperæmia sets in, or they may be marked at the onset. Convulsions, often

unilateral, may occur and be repeated for days, chiefly when the softening involves the motor region of the cortex, and spares the path from it through the central ganglia. The patient may pass from the apoplectic condition into one of delirium. In the old, delirium may be the chief symptom of the onset in cases in which the softened areas are small, multiple, and cortical in situation. According as these symptoms are chiefly marked at the onset three varieties have been described, the *apoplectic*, *convulsive*, and *delirious* forms.

Recovery from the special symptoms of the attack is often incomplete; permanent weakness may remain, as hemiplegia, and mental power is weakened, the patient passing into the chronic state about to be described. The persistent hemiplegia is often accompanied by rigidity, or, when the loss of power is incomplete, by mobile spasm, such as, in its most marked form, has been described as *athetosis*. When the cause is arterial degeneration, return or relapse is common, and it is almost invariable where senile arterial disease is widely spread.

*Chronic softening of the brain* is a term applied to a group of symptoms, of wide range, indicative of failure of cerebral power. It is a term that has become firmly rooted in popular nosology, where it denotes chiefly general paralysis of the insane, but includes also all maladies attended by a similar mental failure, profound in degree and permanent in duration and progressive in course. This use of the word is a source of considerable inconvenience, since it may be strictly accurate to deny the application of a name to that which, nevertheless, it perfectly connotes; and it is generally wiser to explain the change which has come over the use of the word, rather than to give a simple answer to the question in the form it commonly assumes, Is it softening of the brain? The symptoms indicated by the term may supervene on more acute symptoms of softening, or may be gradual in their onset. There is mental dulness, defective perception, drowsiness, loss of memory (especially for recent events), often slight wandering; emotional manifestations are easily excited. Physical power is defective—as a rule generally, sometimes locally. The more delicate motor actions are imperfectly adjusted: articulation becomes indistinct, and the handwriting imperfect. These symptoms may progress into actual imbecility, or may be cut short by some more profound cerebral seizure, or by some intercurrent pulmonary affection, rendered grave by the deficient muscular respiratory power. They are met with chiefly in the old, and are probably due to concomitant degeneration of cerebral tissue and of the arteries supplying it with blood—the two pathological elements varying in relative degree and in relation. Spots of softening, often widely

spread, may be found, and are, indeed, the cause of many of the symptoms. But the state may also come on without any local softening, and without the degenerated vessels to which such softening is usually due. A similar state often follows any grave local lesion of the brain in advancing life.

Progressive symptoms, focal or general, or both, sometimes of the character just described, and sometimes such as are caused by a cerebral tumour, have been met with in a few cases in which extensive local softening of the cerebral tissue has been found, without vascular disease to explain it, and without correspondence with arterial territories. Most subjects have been old, and the disease has not commonly been diagnosed during life. Recorded cases are at present too few to permit an adequate history of the disease to be inferred. Altogether, not only is the term one of loose usage, but the lesion is one of such varied relations, and its symptoms of such equivocal significance, that the subject is a region of cerebral pathology in which very much remains to be done to establish even a firm outline of scientific knowledge.

**DIAGNOSIS.**—The *acute* form of softening has to be distinguished from acute congestive apoplexy and from cerebral hæmorrhage. It is distinguished from the former by the occurrence of the symptoms indicative of local mischief, and by the absence of evidence of cephalic hyperæmia. From hæmorrhage the diagnosis is often difficult, except during the first half of life, when hæmorrhage is so rare that it should only be thought of when loss of consciousness is profound and prolonged. In softening from thrombosis, the initial apoplectic symptoms may be absent, or, if present, slight and brief. They are more often preceded by slight local cerebral symptoms, due to the vascular disease, than is the onset of cerebral hæmorrhage. In the latter such symptoms are merely associated, and are not due to the cause of the hæmorrhage, except in the rare cases in which there is an aneurysm of one of the larger intracranial arteries. In these, however, preceding arterial symptoms are often more considerable in degree and duration than those which precede softening. Most important is the indication afforded by the state of the heart; evidence of its hypertrophy is seldom absent in cerebral hæmorrhage, while it is feeble and often irregular in the softening from atheroma, and presents evidence of valvular disease in embolism. Improvement occurs earlier than in cerebral hæmorrhage. The temperature rises soon after the attack, but falls in a day or two; in hæmorrhage the rise occurs later (Bourneville). There is more marked mental change than in hæmorrhage, shown at first in excitement, subsequently by depression and deterioration of power. In the cases in which the onset is sudden and the apoplexy profound, a diagnosis from hæmorrhage is often impos-

sible. In embolism the onset of the attack is commonly sudden, but the loss of consciousness is less profound than in hæmorrhage, and is often absent. This is true also of softening from syphilitic disease, in which the diagnosis is often aided by a history of the causal malady. Too much weight must not be laid on the absence of such a history in patients in whom the disease is possible, and especially little significance can be attached to the absence of a history of constitutional symptoms, while neither the thoroughness of former treatment, nor a considerable period of freedom from symptoms, has any value whatever as a contra-indication. But with the lapse of time, it does become unlikely that softening is due to this cause; still, it is only after twenty years, and in the absence of any suspicion of a second infection, that the improbability has considerable weight. Optic neuritis, developing after the lesion, occurs in rare cases of softening from embolism, and is practically absent in hæmorrhage. In all cases the various indications must be compared and balanced, and their relative weight noted in the estimation, special caution being taken to allow no weight to negative facts in the presence of positive evidence of opposite significance. In capillary embolism, if extensive, a distinction from hæmorrhage often cannot be made—the loss of consciousness is profound and lasting. Bilateral softening, such as sometimes results from disease of both middle cerebral arteries, may also (as already mentioned) cause symptoms indistinguishable from those of ventricular hæmorrhage.

The distinction of softening from other cerebral diseases is described in the accounts of these.

**PROGNOSIS.**—The immediate and ultimate prognosis in an attack of softening of the brain depends on the degree and extent of the symptoms, as indicating the extent of the lesion; and on their character, as indicating the region of the brain damaged. Both the near and the remote prognosis is much graver in damage to the medulla and pons Varolii than when the corpus striatum or cerebral hemispheres are affected. Locality is of especial importance in regard to the prognosis in softening, because it is by far the most frequent lesion of the medulla or pons that is survived. Hence the prognosis is of high practical importance at the onset as well as later on. The intensity of the coma indicates a greater degree of gravity than does its duration. Youth and general health favour the rapidity and the degree of recovery. Where actual softening has occurred, the damaged tissue never regains its functional power. The congested periphery may recover in proportion to the integrity of the tissues, and to the freedom of the vessels from disease. The chances of a recurrence of softening in another situation depend on

the extent to which its causal condition is widely spread or can be removed. In vascular degeneration recurrence is almost certain. In embolism it is rare. The prognosis in syphilitic disease of the vessels depends upon the recognition and treatment of the syphilitic influence so far as recurrence is concerned; but the prognosis of developed symptoms is independent of the fact of this syphilitic cause, or of any treatment to which the patient may be subjected. Treatment can only influence the disease of the wall of the vessel, and this can have no effect in restoring the circulation, since the vessel beyond is closed by clot, or in restoring the structure of the destroyed tissue of the brain. As a matter of fact, the course of hemiplegia due to this cause is precisely the same as that of hemiplegia due to softening produced by any other vascular mechanism; improvement or recovery is determined by the position of the lesion, and the extent to which the symptoms are not due to the actual destructive softening, and to which they can be compensated. Improvement or persistence of symptoms may coincide with treatment, but the one is not due to it, nor the other the result of its absence.

**TREATMENT.**—The treatment at the onset of cerebral softening is of great importance, because, although nothing can probably be done to undo the mischief that has occurred, much may be done to prevent its extension or increase, and in many cases it is by such preventable extension or increase, after the patient comes under treatment, that the degree of ultimate disability, or death itself, is determined. It is essential that the attention of the practitioner should be fixed on the pathological process and its mechanism; he should endeavour to picture to himself clearly what is taking place and how it is being brought about, and arrange his therapeutic measures so as to counteract the morbid influences in the utmost practicable degree. The closure of a vessel by clot is the great fact of what may be termed the therapeutic pathology of these cases, and the measures to obviate the increase in this process have much in common in the various forms of softening. The variations rendered necessary by the differences in mechanism are considered in the articles on the several vascular diseases. During the acute stage the patient must be kept at perfect rest, with the head moderately raised, flexion of the neck being avoided. During the initial stage of shock, warmth by hot-water bottles, &c., should be applied to the extremities, to equalise the circulation. The bowels, if confined, should be made to act gently; but, unless the evidence of encephalic congestion be early and conspicuous, purgation should be avoided. The determination of blood from the brain to the intestines, involved in purgation, and desirable in cerebral hæmorrhage,

is to be carefully avoided in cerebral softening. It is important also to maintain the circulation steady and uniform, avoiding alike undue quickening or slowing of the blood-current. Hence, digitalis is useful in all cases in which there is cardiac weakness or irregularity. In all senile cases, or when there is a gouty diathesis, nitrous ether or other diuretic drug is useful, and, in the latter case, some lithia may be added. Thus the tendency of the blood to clot is lessened, and the risk of the extension of the lesion—always a danger in the early stage of the affection—is lessened or obviated. Stimulants must be given or withheld according to the state of the heart and circulation; when there is doubt whether they should be given or not, it is better to give a small quantity in softening, and to withhold them in hæmorrhage or if the diagnosis from hæmorrhage is doubtful. After the stage of depression has passed, the irritation due to secondary inflammation, indicated by headache and elevation of temperature, needs quietude, laxatives, and sometimes cold to the head. When convulsions are an early and recurrent symptom, mustard plasters to the neck and extremities, and bromide of potassium, are sometimes effective; in the early period, cold to the head may arrest them. The fits that attend the actual onset are seldom influenced by treatment; they cease because the tissue is destroyed that is essential for their occurrence. When these fail, hyoscyne may be given cautiously, and in small doses ( $\frac{1}{10}$  gr.).

After the attack has passed, recovery must be aided by maintaining the general health in the best possible condition. The secretions should be kept free, the digestive organs in good order, the habits strictly regulated, and nerve tonics—cod-liver oil, hypophosphite of sodium, strychnine, quinine, and iron—may be given with advantage. The symptoms of chronic softening, whether occurring after an acute attack or coming on gradually, should be treated in a similar manner.

W. R. GOWERS.

**BRAIN, Syphilitic Disease of.** See BRAIN, Tumours and New Growths of.

**BRAIN, Tubercle of.** See BRAIN, Tumours and New Growths of.

**BRAIN, Tumours and New Growths of.**—The intimate connexion of the brain with its membranes makes it impossible, except in the most general terms, to draw any marked distinction between tumours of the cerebral substance and tumours arising from its envelopes. A growth pressing inwards from a membrane must impinge upon brain-tissue; a growth originating in brain-tissue must in many situations involve the membranes.

**ANATOMICAL CHARACTERS.**—The tissues from which tumours have their origin seem to present the best ground for a scientific classification of these lesions; and it is not

devoid of interest to mark that the absolute nerve-elements of the brain are never primarily the source of a morbid growth.

Cerebral tumours, then, may be roughly separated into three series:—

1. Those whose centre of origin is some one or other of the membranes, external to the brain, or dipping into the ventricles.

2. Those which spring from the blood-vessels.

3. Those which own the neuroglia as their starting-point.

Dr. Gowers considers such a classification of small practical value, and believes it is more convenient to describe the various growths in the order of their frequency. Tumours, therefore, may be grouped in six categories:—

(1) *Diathetic*: Tubercular and Syphilitic. (2) *Sarcomatous*: Glioma, Sarcoma, Myxoma. (3) *Carcinoma*. (4) *Osteo-fibroid*: Fibroma, Osteoma, Osteo-fibroma. (5) *Miscellaneous*: Cholesteatoma, Lipoma, Vascular or Erectile Tumours, Psammoma, Neuroma. (6) *Parasitic*: Echinococcus and Cysticercus.

Taking, however, this original subdivision, which is Rindfleisch's, the tumours which are placed in each series differ somewhat from his arrangement. In the first series five kinds of tumour are found, arising from the membranes or from the free surface of the ventricles. These are Pacchionian granulations; spindle-celled sarcoma; myxoma of the membranes; psammoma; and lipoma. The second series will include, first, aneurysms, depending upon disease of one or more of the arterial coats; and, secondly, such tumours as have their origin in the sheaths of the vessels, comprising carcinoma cerebri simplex; fungus of the dura mater; cholesteatoma; epithelioma myxomatodes psammosum; papilloma of the pia mater and vessels; papilloma myxomatodes; and tubercle. In the third series may be counted glioma; myxoma of the nerve-substance; syphilitic gumma; and fibroma. Included under none of those headings, echinococci and cysticerci cellulosa must be mentioned, as they affect the brain.

Each of the growths enumerated will now be briefly described.

1. *Pacchionian granulations*.—These are granulations of the arachnoid, sometimes met with in childhood, very constantly from middle age onwards, and scarcely recognised as morbid lesions. Their ætiology is unknown. They do not give rise to any symptoms. They are chiefly situated along the superior longitudinal sinus, which in rare cases is perforated by them. They form groups of papillæ, consisting of striped connective tissue, poor in cells, and proceeding directly from a thin but a continually renewed layer of sub-epithelial germinal tissue.

2. *Sarcoma*.—This sometimes has its origin in the nervous tissue itself, but more fre-

quently arises from the dura mater, especially at the base of the skull. From their situation sarcomata are especially apt to interfere with one or more of the cerebral nerves. They may attain the size of a pigeon's or even of a hen's egg.

When sarcoma attacks the dura mater it originates from its internal side. The most usual situation is the membrane about the sella turcica and the pars petrosa. It forms a depression in the brain, while the bone becomes atrophied behind it. The growth is composed of fusiform cells, with tolerably numerous and sometimes dilated vessels. Sarcomatous growths are not freely developed above the surface, but rather in the depths of the tissue; they distend the cerebral convolutions, form deep depressions on the surface, and even penetrate far into the brain. They occur under two forms—*hard sarcoma* with compact fibrous fundamental tissue and small cells, often called fibrous tumour; and *soft sarcoma*, with a loose scanty intercellular substance, and numerous cells of comparatively large size. The cells are mostly fusiform, but sometimes round and multinuclear, and the two latter may be surrounded by the former. Sarcoma in this situation is generally single. It may attain the size of a nut or even of an apple, and is frequently hæmorrhagic.

In the cerebral tissue itself the hard sarcoma attains a great degree of density: it is sometimes fibrous, at other times cartilaginous, of a dense homogeneous structure, whitish or bluish-white, with a yellow tinge here and there, and with very few vessels. It is distinguished from the brain that surrounds it by a very vascular zone. It can be easily separated from the parenchyma, and may thus be recognised after death from simple sclerosis and hard glioma of the brain. The softer form—*fibro-cellular sarcoma*—is generally either a myxo- or a glio-sarcoma: but pure fuso-cellular sarcoma is met with. The tumour is a clear grey, almost like the grey matter of the corpus striatum. It is often vascular, with a reddish tinge. These sarcomata are often almost spherical, and easily detached from the surrounding brain-substance. Others, however, seem to be continuous with the neighbouring substance, and to be little more than simple hypertrophies of the cerebral tissue. Especially is this the case in tumours of the corpus striatum and optic thalamus. The cells of cerebral sarcoma are frequently the seat of fatty degeneration, and the whole tumour may be hæmorrhagic. Its most frequent situations in the brain are the ganglia at the base.

3. *Myxoma*.—Myxoma of the membranes is rare, and generally has its origin from the convexity of the brain, being connected with the inner surface of the dura mater. It is a small growth, soft, fragile, having a gelatinous aspect.

Myxomata are frequently met with in the cerebral hemispheres, and then take their origin from the neuroglia. Such growths are probably malignant, the proofs of their malignancy being that they are often multiple locally; that they frequently recur when removed from a peripheral nerve; and that they not seldom affect internal organs. Myxoma possibly includes all that has been called colloid cancer. When this lesion affects the cerebral hemispheres it may be of large size. The mucus is a constituent part of the tissue; it is not a product of secretion, as in mucous cysts. It is most commonly a mucoid form of glioma.

4. *Psammoma*.—Psammomata have been met with in the brain, spinal membranes, spinal cord, and nerves; they are not uncommon in the choroid plexus, but are most usually found in the pineal gland. There are two kinds of these growths. In the first, the sand occupies the interior of the meshes of the connective tissue in very varied forms, as compact cylinders, as pear-shaped masses, as spines, or as globes, surrounded by connective tissue, and connected by it with the other parts of the tumour. In the second form, the sand lies without cohesion in the parts and between them, so that the different grains of sand may be easily isolated. In this latter form, the psammoma is composed most generally of round elliptical corpuscles, and sometimes also of large complex conglomerations. These little tumours have usually a concentric arrangement internally.

5. *Lipoma*.—This is a rare tumour in the brain. It may be connected with the inner surface of the dura mater, or with the ependyma of the ventricles. The fatty matter is contained in cells, and the cells are surrounded by an organised membrane. Lipoma is usually single, seldom multiple; of irregular shape; and varies in size from a small nut to a hen's egg. Small pieces of carbonate of lime have been found in these tumours.

6. *Aneurysms*.—The larger cerebral aneurysms have been observed from early times. More recently Liouville has called special attention to the subject of miliary aneurysms, and has shown that they are common; that they are multiple; that they frequently give way in the brain or in the pia mater; and that they often co-exist with aneurysms of the larger vessels in other parts of the body. Aneurysm of the middle meningeal, of the internal carotid within the cavernous sinus and at its exit from it, of the anterior cerebral, of the anterior communicating, of the arteries of the corpus callosum, of the middle cerebral, of the posterior communicating, of the vertebral, of the basilar, of the posterior cerebral, and of the arteries supplying the cerebellum, are all met with not infrequently. The middle cerebral and the basilar, however, are the vessels most usually affected with this lesion.

The minute miliary aneurysms have been observed in the pia mater, at the surface of the convolutions or in their substance, in the optic thalami, the pons, the corpora striata, cerebellum, crura cerebri, and medulla oblongata; more rarely in the centrum ovale. A large one was found by Dr. Bastian in the lenticular nucleus. These miliary aneurysms may be visible to the naked eye. The smallest are seen under the microscope as ampullæ of the vessels, containing coagulated blood or granules of hæmatoidin. The arterial walls have generally undergone some form of degeneration. The vessel, dilated at some parts, is constricted at others. The lesion may be a consequence of atheroma of the vessel, but far more commonly it is the result of arterio-sclerosis of the inner coat of the vessels, either at the seat or in the immediate neighbourhood of the aneurysm. Embolism is a frequent cause. See BRAIN, Vessels of, Diseases of.

7. *Carcinoma cerebri simplex*.—Cancer, excluding from this term sarcoma and glioma, may originate in the cranial bones, the dura mater, the pia mater, the cerebrum, the cerebellum, the pons, and the medulla oblongata. The medulla oblongata, the fornix, and the corpora quadrigemina are the regions least often affected, whereas the cerebral hemispheres are the most favourite localities. All forms of cancer are met with, in all cases having their origin in the coats of the vessels. Epithelial cancer has been generally believed to have its starting point in the peripheral layer of the arachnoid, the tissue that lines the under surface of the dura mater. Encephaloid cancer is, however, the most common form met with in the brain.

8. *Fungus of the dura mater* can scarcely be separated from the preceding form. It arises from the outer surface of the dura mater, penetrates with the vessel from which it springs into the compact tissue, destroys the vitreous table, and spreads out in the diploë: in its progress it may penetrate the external table and lift up the integuments of the cranium. The internal table invariably suffers more than the external. Sometimes there is coincident passage of the tumour inwards, and the subjacent membranes become glued to the dura mater and to the cerebral substance. There may result simply the depression of surface consequent upon pressure from above, but more commonly cell-growth similar to that of the original tumour takes place, first from the vessels of the pia mater, and afterwards from the vessels of the cerebral convolutions.

9. Closely allied to the epithelial cancer that has its origin in the dura mater is the *cholesteatoma*, which is generally situated at the base of the brain. Rindfleisch considers it a squamous epithelioma, whose cellular cylinders are wholly converted into a mass of pearly nodules with a silky lustre. It is

covered by the arachnoid, and springs either from the vessels of the pia mater, or more rarely from the perivascular sheath of the vessels in the substance of the brain.

10. Last of the cancers is a tumour that has been found in the third ventricle—*epithelioma myxomatodes psammosum*—consisting of globes and cylinders of epithelial cells, embedded in a very bulky stroma of mucous tissue.

11. Springing also from the vessels, two forms of *papilloma* are met with—papilloma of the pia mater and vessels; and papilloma myxomatodes. The former is composed of a number of branching papillæ, each of which contains a blood-vessel with a small amount of connective tissue, and a double coat of epithelium, of which the outer layer is columnar. In the latter, which is probably a mere variety of the former, the structure of the tumour is the same, but the columnar cells secrete a vast amount of viscid mucus.

12. *Tubercle* springs from the middle tunic of the small arteries of the pia mater, or of the nerve-substance. It rarely attacks the membranes in the form of tumour, rarely also the white matter of the brain, but prefers, as its principal seat, the grey matter of the convolutions and of the deeper parts. Tubercle of the dura mater is, however, sometimes met with, and it may induce obliteration of sinuses. The cerebellum is a frequent seat of tubercle, which exists here in the form of superficial granulations. The pons also is frequently affected with tubercle, both in the form of small tumours of its substance, and as polypous tubercles of the fourth ventricle. Tubercle is separated from the surrounding cerebral substance by a very delicate reddish envelope.

Tubercular tumours of the cerebral substance are often multiple, and not infrequently large; they are of very slow growth; persist long in the caseous state; and may be found cretified. Sometimes there is cerebral softening around them. Virchow states that the increase of tubercles takes place by apposition or juxtaposition, and that the apposition takes place not by layers primarily caseous, but by zones of new grey proliferation, usually in the form of miliary tubercle. A very delicate layer of connective tissue of new formation, a species of encyusting false membrane, represents the mother-tissue for the subsequent generation of young tubercles.

13. *Glioma*, called by Billroth *granulated sarcoma*, or *round-celled sarcoma*, is practically a local hyperplastic development of the neuroglia. It may appear in three forms, either as *soft glioma*, rich in cells—the most common kind; as *hard glioma*, fibrous, and, if the vessels are much developed, telangiectasic; or thirdly, as a *myxoglioma*, a complex tumour, in which part of the tumour takes the appearance of mucous tissue. The nature of the tumour is partly

determined by the nature of the tissue from which it springs; thus glioma of the brain is generally, but not always, soft, glioma of the ependyma hard. The soft gliomata are closely allied to myxomata. The intercellular substance is found in moderate quantity. In the more mucous gliomata the network is regular and large, and the tissue has little cohesion. If the meshes are larger still, and the mucous element abundant, this variety passes into a myxoma. If there be a considerable increase of cells, whilst the trabeculæ become narrower, we get a medullary glioma, which may be transformed into a medullary sarcoma if the cells continue to grow and multiply. These transitions are not uncommon even in the same tumour, especially in the posterior lobes of the brain. If the vessels are developed in great abundance we get hæmorrhages and a kind of fungus hæmatodes. Hard glioma is closely allied to fibroma, with which indeed it may be combined to form a fibro-glioma. In hard glioma the fibres are not arranged in a network, but in parallel lines, like felt. The nervous elements, naturally contained in the neuroglia, are absent in these tumours. The walls of the vessels are frequently thickened.

The glioma of the ependyma is of little importance; it is seen as fine granulations on the surface of the lateral ventricles in chronic hydrocephalus; on the floor of the fourth ventricle it may grow to the size of a cherry. In the cerebral substance, gliomata may attain the size of a fist, or even of a child's head; and they are often mistaken for cancers or sarcomata of the brain. Hard glioma may be distinguished from sclerosis, in that sclerosis encloses the normal nerve-elements. In glioma, too, there is great proliferation of neuroglia cells. There is no distinct limit between glioma and the surrounding brain-substance, but the tumour on section shows greater vascularity, greater consistence, and a more transparent constitution, as well as often a bluish-white appearance compared with the white brain-matter. The demarcation in grey matter is imperceptible to the naked eye, especially if the glioma be soft. Soft glioma is generally single; hard is often multiple. The membranes may adhere, but form no part of the tumour.

Virchow thinks that glioma is not malignant; that hard glioma has an inflammatory origin; and that the soft variety is set up by local causes, such as injury. Gintrac, on the other hand, unites gliomata and sarcomata under the head of cancers.

Soft glioma is most frequently situated in the posterior lobes, less often in the upper and lateral parts of the cerebral hemispheres. It may be congenital. It gives rise to complications—namely, first, great congestion, causing cerebral compression, irritation, pain, excitement, or apoplexy; and, secondly, hydrocephalus of the ventricles, which in

protracted cases is seldom absent. The latter occurs most rapidly in glioma of the optic thalami, or of the posterior lobe, compressing the choroid veins, the venæ Galeni, or the transverse sinus.

14. *Myxoma of the nerve-substance*.—This is not common in the brain. It owns the same origin as glioma, having the neuroglia as its starting-point.

15. *Syphilitic gumma*.—Gummy tumours of the brain are generally found at the circumference, and especially at the base of the brain. Their origin is either from the membranes, from the vessels, or from the neuroglia of the cerebral substance. They have infective properties, or at any rate they are multiple, and may be met with at the same time affecting the dura mater, the pia mater, the brain, nerves, and cranial bones. They are often accompanied by inflammatory phenomena, a point which distinguishes them from large tubercles. Gumma is not the usual form in which syphilis attacks the dura mater on its external surface. It may, however, affect the arachnoid surface of the dura mater. In this situation the gummata may vary in size from a hemp-seed to a nut. They may be found just above the convexity of the hemispheres, or at the anterior part of the base of the brain, especially about the sella turcica, or on the tentorium cerebelli. They have been met with in the falx cerebri. The inflammatory condition around these tumours often unites them to the pia mater, and the subjacent portion of brain is frequently softened, either by the inflammation, or by arterial obliteration.

When the pia mater is united to the dura mater, gummata very small in size may form in the former membrane. The subjacent brain may be softened or sclerosed. Much larger gummata, however—from the size of a nut to that of a hen's egg—originate from the pia mater, and are most usually situated in the region between the optic chiasma and the pons, or on the crura cerebelli. On the convex surface they are much smaller. Gummata of the cerebral substance occur in situations most subject to traumatic influences. The chief seats are the cerebral hemispheres, the large ganglia, especially the optic thalami, and next in frequency the pons, and crura cerebri and cerebelli. The tumours attain to a good size, but are not so large as those of the pia mater. They may be multiple, but often exist singly. See BRAIN, Vessels of, Diseases of.

16. *Fibroma*.—True fibromata, distinct from hard gliomata and sarcomata, probably do not exist in the brain or its membranes. Fibromata are essentially composed of connective tissue. Such increase in this tissue is sclerosis, and its arrangement is too indefinite, its amount too small, to be considered a tumour. Rindfleisch is probably wrong in stating that there are solitary tubercles of

the brain which deserve rather to be called fibroid tumours; although it may be true that in some cheesy nodules of the nervous centres the growth of fibres and the condensation predominate enormously over the corpuscular structure. Practically, however, both *enchondromata* and *osteomata* are fibrous tumours. An enchondroma is a heterologous tumour not developed from a pre-existing cartilage, but produced by a change in the type of formation by proceeding from a non-cartilaginous matrix. Although osteoid enchondromata may be malignant, yet true osteomata are not so. These tumours may attack the cerebral dura mater, and on the convexity are multiple. If the tumour attack the falx cerebri it is solitary. Its starting-point is the internal surface of the dura mater. It is distinguished from exostosis of bone by having a fibrous layer between it and the bone. It may set up irritative pachymeningitis. Such tumours are also found small in connexion with the cerebral arachnoid, as simple united patches or pointed prolongations. Their favourite seat is the convex surface of the anterior lobes. The nervous centres are very rarely the seat of these tumours. Their matrix is formed by connective tissue, not cartilage, the product of irritation of the neuroglia, and so a consequence of circumscribed encephalitis.

17. *Hydatids* are rare within the skull, but are met with occasionally in all parts of the brain, between the membranes, in the ventricles, and lying free at the base of the brain. They are more common in children than in adults. The brain may suffer from pressure either in the way of softening from interference with the vessels, or from sclerosis.

*Cysticerci* are also met with in various parts of the brain or its membranes. They may be surrounded by connective-tissue capsules; or may lie free, arranged in a racemose form. They may be single, or may attack the same individual in several hundred places at once.

**SYMPTOMS**.—Even in tumours of considerable size all symptoms may be latent. The more tolerant portion of the brain will include the hemispheres and the white commissural regions, whilst the mesocephale, the optic thalami, and the corpora striata are amongst the least tolerant portions. It is not unusual, moreover, to meet with decided intermissions, especially in the early period of the disease; such intermissions depending on temporary lesions in the immediate neighbourhood of the tumour.

Even with these intermissions the diagnosis of the presence or position of the cerebral tumours would be comparatively easy, if the symptoms invariably depended upon direct excitation. Many of the phenomena pass the limits of the immediate sphere of the tumour, and are the results of reflex action.

Sometimes also the symptoms due to direct and reflex excitation may coincide, and this is particularly the case in tumours of the base. The main difficulties lie in the possible latency of all symptoms; in their internission; in the distinction and combination of direct and reflex excitation; and in the remissions following physical or psychical excitement.

Symptoms, then, may depend on direct or reflex excitation, and consist of exaltation of functional activity, such as contractions, partial or general convulsions, hyperæsthesia, and hyperideation. Others are produced by secondary lesions in the neighbourhood of the tumour—congestion, hæmorrhage, inflammation, &c.—and these may include not only all in the previous division, but temporary or persistent paralysis, fever, and other phenomena. Then there may be symptoms of direct compression, definite paralyzes, and gradual enfeeblement of the sensorial and intellectual faculties.

Taking some of the more common conditions in order, and viewing them as dependent on direct or reflex irritation, the most frequent certainly is headache; and except in tumour of the cerebellum, when the headache is almost invariably occipital, there is no symptom less useful in determining the position of the lesion. It is less frequently due to direct excitation than to reflex. The pain is very severe, indeed, more so than in any other disease, excepting, perhaps, meningitis; it persists through the whole malady; and is increased by vibration of all kinds, light, sound, or movement of the head. It may be confined to a single spot, or be diffused over the whole head. Connected with headache in many cases, and often equally the effects of radiated influence, are *tinnitus aurium*, morbid acuteness of hearing, or painful sensitiveness to sound; disturbances of vision, *diplopia*, *muscæ volitantes*, and *strabismus*, which may be transient; *formication*, and sometimes *hyperæsthesiæ* of greater or less extent. In some cases there is an agitated condition of the intellectual faculties, and even *delirium*. Disturbances of sight are very common. The retinal lesions will be subsequently described. It is a remarkable fact, as bearing upon the decussating arrangement of the optic nerves, that the affections of sight usually implicate both eyes, even where the tumour has involved only one optic nerve, and is not situated near the optic chiasma or the *corpora quadrigemina*. Hearing is far less often affected than vision. Generally a slight diminution only of this function is observed; and, in the rare cases in which complete deafness is met with, it is unilateral. Taste and smell are seldom interfered with. When these special senses are morbidly affected, the tumour will probably be situated in the *uncus gyri hippocampi* (the hippocampal lobule) or its immediate vicinity.

The symptoms of compression may be included in the expression 'lowering of function,' comprising apathy, feebleness of memory, want of attention, confusion, and a general enfeeblement of ideas. These conditions are often accompanied or preceded by certain diffused symptoms, such as *vertigo*.

*Vertigo* is the first symptom in many cases: it is felt especially when the patient is in the upright position. It often produces uncertainty of gait, even where the tumour is not in the cerebellum. Strange sensations in the head are also complained of, a feeling of liquid in the head, or of a mobile body; or the sensation may be that of a solid body filling the head, or pressing upon some portion of it.

These phenomena often coincide with evidences of irritation of the *mesocephale*, whether due to direct compression or to radiated irritation of the *medulla oblongata*. Chief amongst these symptoms is vomiting. It is not accompanied by nausea or other manifestations of *dyspepsia*, and it will occur when the stomach is empty. It can frequently be checked only by keeping the patient in a recumbent position. Constipation also is often obstinate.

*Epileptiform convulsion* has an important bearing on the diagnosis of these lesions. Very frequently convulsion is preceded by many of the phenomena already touched upon, such as headache or *vertigo*. Frequently, however, convulsion precedes all other morbid phenomena, and the patient may be in perfect health in the intervals of the attacks. Given, therefore, convulsion as the one factor in forming an opinion, it is necessary to consider the liability of the patient to convulsive attacks from causes other than tumours; to realise whether the family history shows any suspicion of *epilepsy*; and to eliminate from the case the possibility of *saturnine*, *alcoholic*, and *uræmic* poisoning. If this is done, and especially if we find early convulsion associated with headache and with vomiting, this symptom will prove an important aid in the diagnosis of tumour.

The phenomena depending on the presence of tumour itself may be associated with others due to complications, such as *œdema*, *congestion*, *encephalitis*, or *meningitis* of the surrounding parts. A high temperature, for instance, will point to inflammation either of the nervous substance or of the meninges near the lesion; and *meningeal inflammation* seems to be accompanied by the highest temperature. *Syphilitic gumma*, however, may coincide with *syphilitic meningitis* on some other portion of the *encephalon* not directly connected with the immediate surroundings of the tumour.

Passing for the moment the subject of definite paralyzes with the remark that the *sphincters* are seldom affected, even in cases

in which the paralysis takes a paraplegic form, it may be mentioned that a want of equilibrium seems to be a not unusual evidence of the presence of tumour in the cerebellum. Aphasia may not only be due to the special localisation of tumour in Broca's region of the left anterior lobe, but also to the presence of this lesion in any part of the tract (corpus striatum, optic thalamus, or crus cerebri) which unites this portion of the hemisphere to the medulla oblongata, the highway by which the centre for the production of articulate speech is connected with the co-ordinating centre for this function, or to tumour affecting those centres lesion of which is associated with word-blindness, or word-deafness. Anæsthesia of the skin is seldom met with as a symptom of cerebral tumour. When present it is found only in the limbs affected with motor paralysis, and is scarcely ever complete.

*Symptoms of special localities.*—It remains to take special regions of the encephalon separately, and to endeavour to differentiate the position of the tumour by the symptoms attending its presence.

Tumour in the *medulla oblongata* will be accompanied by various disturbances of sensibility, especially headache, and sometimes by convulsions. The pressure of a tumour is seldom limited to the medulla oblongata, and the symptoms therefore are complex. In several of the cases recorded there has not only been amaurosis and deafness of one side, but interference with taste and smell. Aneurysm of the basilar artery has been known to cause temporary albuminuria and convulsions coincident with each other.

Tumour of the *fourth ventricle* may manifest itself by the presence of sugar or inositol in the urine. Tumour here, as in the medulla oblongata, frequently destroys life quickly, before there has been time, so to speak, for much local lesion to be set up. Vomiting is a frequent symptom.

Tumour of the *crura cerebelli* and of the *corpora quadrigemina* cannot be diagnosed by any peculiar symptoms. In one case, in which the corpora quadrigemina seemed wholly transformed into a tuberculous mass, the sight remained good, but there was double ptosis.

In tumour of the *cerebellum* there is little disturbance of sensibility, except occipital headache. There are various disturbances of motility, especially convulsions and irregularity of locomotion, but no true paralysis. Amblyopia, amaurosis, and convergent strabismus are common. There is no interference with the psychological functions, or with speech, as a general rule. Vomiting is very common. In 76 cases collected by Ladame, there was no abnormality in the genital functions, except in 4 instances.

In tumours of the *pons*, the disturbances

of sensibility are general or partial anæsthesia, and in some cases more or less headache. Hemiplegia, of unequal degree on the two sides, and other forms of paralysis are observed, but no convulsion. Various and manifold disturbances of the special senses; phenomena of depression of mind; frequent alteration of speech; and early disorders in swallowing, occur.

Tumours of the *crura cerebri* follow the example of the same lesion in the pons with reference to disturbances of sensibility. Equilateral hemiplegia opposite to the lesion is met with, and paralysis of the oculo-motor nerve on the same side as the tumour, often gradually extending itself to both oculomotors.

Tumours of the *pituitary gland* are accompanied by intense frontal headache; by no definite disturbances of sensation or of motion; by double amblyopia or amaurosis, unequally developed; and by no loss of speech.

Tumours of the *middle cavities of the cranium* seem to affect mainly the third and the fifth nerves, anæsthesia or pains in the face and ptosis being the prominent symptoms, with some interference with the free action of the other muscles of the eyeball supplied by the third. In one case, in which a scirrhus tumour of the left side was situated on the inner surface of the sphenoid bone, extending laterally to the internal auditory meatus and backwards to the pons, not only were the third and fifth nerves paralysed, but colour-blindness supervened some time before death.

In tumours of the *corpora striata* and *optic thalami*, headache is less frequent than in other regions. Hemiplegia and convulsions are frequent, the former especially so. Hardly any disturbance of the special senses is observed. Intelligence and speech are frequently disordered.

In tumour of the *corpus callosum*, there is frequently some mental aberration, and often convulsions; but not rarely a total absence of morbid symptoms.

In tumour of the *middle cerebral lobes*, headache is frequently a prominent symptom, but otherwise the sensory disorder is mainly anæsthetic. Hemiplegia is common, as is also convulsion of an epileptiform character. These convulsive attacks are not rarely unilateral, and sometimes affect at first one limb only. In fact the position of a tumour towards the anterior portion of the middle lobe may be determined by symptoms with tolerable accuracy. Various disturbances of sight and of hearing are met with; as well as various psychological abnormalities, ranging from mere confusion of ideas to absolute imbecility.

In tumour of the *anterior lobes* there is general headache, seldom of the frontal region particularly. No other sensory disturbance

occurs. Sight and smell are frequently affected, speech seldom. Hemiplegia, convulsions, and psychical disturbance will occur, much as in tumours of the middle lobes.

In tumour of the *posterior lobes*, there is general headache, seldom localised in the occiput; no other sensory disturbance. Slightly marked hemiplegia occurs, and convulsive attacks are very frequent. There is no disturbance of the organs of special sense. The mental faculties are greatly altered, particularly in the tendency to depression.

All three lobes may be affected with tumour coincidentally, and the headache is then very intense; the epileptiform convulsions exceed the paralytic phenomena; the organs of special sense are little affected; and there are various mental disturbances.

In tumour of the *convexity*, the headache is generally limited either to the frontal regions, to one side of the head, or to the occiput. There is neither anæsthesia nor paralysis, but intense convulsions occur. The special senses are not disturbed. The mental condition is one of irritation, evidenced by delirium and excitement.

Lastly, very various regions of the brain may be simultaneously the seat of tumour, and the morbid phenomena will be necessarily complex.

*Retinal changes.*—It has seemed more convenient to speak of retinal changes dependent on cerebral tumour separate from the other symptoms. Great variations in the lesion occur—according to the position of the tumour; its direct interference with the optic centres; its complication with meningitis; and its pressure on the optic nerves and chiasma.

Taking choked disc, optic neuritis, and atrophy of the optic nerve as the three chief lesions, cerebral tumour may very frequently induce choked disc, by interfering with the venous ebb from the eye; optic neuritis, if meningitis is associated with the tumour; optic atrophy, by pressure of the tumour on the optic nerve, or by this pressure of the tumour or of hydrocephalus secondarily induced by it on the optic centres or tracts, or by softening around the tumour, such softening implicating the optic centres, or, lastly, by the propagation of sclerosis. Any tumour situated far back in the encephalon may interfere with the venous flow through the venæ Galeni, and so produce hydrocephalus of the ventricles, and the retinal effects of hydrocephalus.

There are no retinal changes from tumour in the corpus callosum, nor, as a rule, from tumour in the optic thalami. Tumours of the cerebral hemispheres all influence the optic nerve, if they interfere with the base of the brain.

Tumours of the cerebellum may cause pressure on the lateral sinuses, the straight sinus, the venæ Galeni, or the torcular Herophili:

pressure in any of these situations may produce choked disc. Or the tumour may affect the corpora quadrigemina; or softening around it may spread to these organs, and atrophy of the optic nerve be the result. *A fortiori*, tumour of the corpora quadrigemina themselves will lead to atrophy of nerve.

Tumour of the *crura cerebelli* causes hydrocephalus, and its effects on the retina.

In a similar way tumours springing from the bone or the membranes at the base of the brain may produce choked disc or atrophy, according to the position of the pressure, optic neuritis by complications with meningitis, or neuro-retinitis by irritation of the connective elements of the nerves (*see OPHTHALMOSCOPE IN MEDICINE*). Hemianopsia bitemporalis fugax has been shown by Oppenheim to be a valuable diagnostic symptom of syphilitic disease in the base of the skull.

*COURSE.*—The clinical course of cerebral tumours is intermittent and paroxysmal. In many cases, such as those of glioma, it is very slow. Two groups of phenomena may complicate its ordinary course, namely, those associated with meningitis and apoplexy.

*SYMPTOMS OF SPECIAL TUMOURS.*—Aneurysmal tumours may sometimes be distinguished by the sense of throbbing in the head; by the patient being of adult age or young; by his being attacked in the midst of perfect health; by vomiting being rare, apoplexy frequent, paralysis of cranial nerves early and unilateral; and by absence of mental phenomena. Aneurysm situated in the cavernous sinus produces exophthalmos. *See BRAIN, Vessels of, Diseases of.*

The symptoms of echinococci differ very little from those of other slow tumours: headache, dizziness, vomiting, syncope, and epileptiform attacks are most frequent. Disturbances of the motor and sensory functions, and also of the mind itself, are sometimes met with, and vary according to the situation of the lesion. The prognosis is unfavourable; the diagnosis impossible, unless echinococci exist at the same time in the liver.

Cysticerci manifest their presence in the brain by epileptic attacks, which augment in number and severity; the health of the patient between the fits is at first good, then apathy and torpor supervene; hemiplegia is rare, and never early; cranial nerve paralyses are exceptional; the symptoms are diffuse and bilateral, owing to the position of the parasite in the grey convolutions, and in many spots at a time. The age of the patient is above forty. Perhaps there may be evidence of the presence of cysticerci elsewhere.

Syphilitic tumours coincide with actual or previous syphilis. The headache attending this form of tumour is generally intensified at night.

In tubercle of the brain there is often a

tuberculous family history, or the presence of tubercle elsewhere in the body. It seldom compresses cranial nerves. Its clinical evolution is often by paroxysms, and grave cases are combined with tuberculous arachnitis and hydrocephalus of the ventricles. Tubercle in the cortical substance of the brain and cerebellum may, however, be attended by no special symptoms.

In cancer also there is frequently a family history of this malady; and the cancerous cachexia may be present. Cancer seldom exists elsewhere when it is cerebral. This growth is much less often accompanied by symptoms due to congestion and hæmorrhage than glioma or sarcoma, being less vascular.

**PROGNOSIS.**—The prognosis of cerebral tumour is always bad, except in syphilitic gumma, and perhaps aneurysm.

**TREATMENT.**—In syphilitic gumma and in aneurysm large doses of iodide of potassium may be used with more or less success. This remedy is also useful in dispersing the results of the meningitis which so often accompanies tumour of the base. Beyond this there is little to be done, except in the endeavour to relieve pain and to support the strength of the patient. In a few instances cerebral tumours have been successfully removed by operation.

E. LONG FOX.

### BRAIN, Ventricles of, Diseases of. *See* VENTRICLES OF BRAIN, Diseases of.

### BRAIN, Vessels of, Diseases of.

1. **Aneurysm.**—The larger arteries of the brain, and their minute branches in the cerebral substance, are both liable to aneurysmal dilatation.

(a) *Aneurysm of the larger cerebral arteries* is more common than that of vessels of a similar size elsewhere. The large vessels of the base, or their primary branches, may be affected. The basilar and middle cerebral arteries are those most frequently diseased, aneurysms of these two vessels constituting more than half the cases. Next in frequency is the internal carotid. The vertebral, anterior and posterior cerebrals, anterior and posterior communicating, and anterior cerebellar arteries are occasionally, but less frequently, affected. In one or two recorded cases the aneurysm has been situated in the interior of the pons Varolii or cerebellum. The disease is rather more frequent on the left side, but this is chiefly due to the greater proclivity of the left middle cerebral compared with the right, a fact which is explained by the ætiology. There may be more than one aneurysm, situated on different arteries, or on different branches of the same artery. The aneurysm is usually sacculated, rarely dissecting. Its size usually varies from that of a pea to that of a nut, but aneurysms have been met with as large as a hen's egg. When the size is considerable the brain-

tissue is pressed upon and softened, as by any other kind of tumour.

**ÆTIOLOGY AND PATHOLOGY.**—These aneurysms are rather more common in men than in women, after actual childhood, and are far more frequent before the ordinary degenerative period than aneurysms elsewhere. Nearly half the recorded instances have occurred between ten and forty years of age, and about one-sixth between ten and twenty. The change in the arterial wall resembles that giving rise to aneurysm elsewhere—a fibroid degeneration, with loss of muscular and elastic tissue. Distensible tissue, which yields permanently before the blood-pressure, is substituted for the resilient elements of the wall, which, normally, bring back the vessel to its previous size after each diastole. The alteration is occasionally part of a widely-spread arterial change, but more frequently is local. It is then due to some local process, usually one of three kinds: (1) syphilitic disease, which has altered the wall, but has not obliterated or greatly reduced the lumen of the vessel; (2) injury—a blow or fall on the head, which probably acts by causing a local arteritis, whilst extensive traces of old inflammation are sometimes found in the neighbourhood of the aneurysm in such cases; (3) embolism, imperfect occlusion of the vessel by a plug, from a valve affected with active endocarditis. Dr. Church first pointed out the association in young persons of cerebral aneurysm with valvular disease of the heart; and so many facts have been published which support the opinion, that the relation must be regarded as beyond question. It is an occasional cause of aneurysm in other situations. There is often evidence of inflammatory and degenerative changes (thickening, calcification) in the arterial wall after embolic occlusion, especially when the plug comes from an inflamed endocardium. It then seems to have the power of exciting a considerable degree of inflammation in the tissue with which it is in contact, and the elements of the wall of the artery may be altered as by traumatic arteritis. If the obstruction is incomplete, the altered wall may yield to the blood-pressure. This cause explains the greater frequency of aneurysm in the left than in the right middle cerebral, and also the fact that those who suffer from cerebral hæmorrhage frequently have valvular disease of the heart, and in a still larger proportion in young persons. Whatever is the cause of the alteration in the arterial wall, the efficient agent in the production of the aneurysm is the high blood-pressure in the cerebral arteries, coupled perhaps with a slighter external support than is possessed by the vessels in other parts.

*Rupture* has occurred in more than half of the recorded cases of cerebral aneurysm. The blood may escape rapidly or slowly, and

the hæmorrhage may take place into the subarachnoid space, or into the adjacent cerebral substance. In the former case, meningeal hæmorrhage is the result. Rupture into the cerebral substance is not uncommon. An aneurysm in the fissure of Sylvius may cause a hæmorrhage into the substance of the brain, bursting into the lateral ventricle; and an aneurysm on the posterior cerebral artery may burst into the substance of the pons. Two causes may determine this rupture into the substance of the brain—first, thickening of the subarachnoid tissue adjacent to the aneurysm, hindering its rupture outwards; secondly, the small size of the opening and consequent gradual escape of the blood, producing a slow disintegration of the brain-tissue, and thus preparing a channel for the effusion. In such cases only a small quantity of blood may have trickled into the ventricles or subarachnoid space. In rare cases a communication with a venous sinus forms, and constitutes an arterio-venous aneurysm. This has occurred between an aneurysm of the internal carotid and the cavernous sinus.

**SYMPTOMS.**—Symptoms of the existence of an aneurysm may be entirely absent. When present, they depend on the pressure which the tumour exerts on neighbouring parts. They vary widely according to its seat, and they are rarely by themselves distinctive. Mental disturbance is uncommon. Headache is a very uniform symptom. It is often intense, sometimes throbbing, and may be localised, as in the occiput in basilar aneurysm. Giddiness is not uncommon, and is complained of in aneurysms in all situations. Convulsions are not frequent except in cases in which the aneurysm is in the neighbourhood of the motor region of the cortex, *i.e.* is on the middle cerebral or its branches. Palsy in some situation often occurs, and depends on the pressure of the tumour; the most common is that of the cranial nerves which lie adjacent to the aneurysm, as of the nerves of the orbit in aneurysm of the internal carotid. Optic neuritis is occasionally present. The symptoms are thus those of an intracranial tumour, and they suggest an aneurysm when they are such as to show that the tumour is in the position of one of the arteries liable to be affected; and the probability is increased if one of the known causes of aneurysm can be traced. Aneurysms elsewhere would, of course, give great additional significance to the symptoms, but the coincidence is too rare, an instance being almost unique, to be of practical importance. In some cases a murmur can be heard by the patient, and in still rarer cases (of aneurysm of the internal carotid) it has been audible on auscultation. But such an objective murmur alone can not afford actual proof of the existence of an aneurysm, since pulsation, the chief sign of an accessible aneurysm, can never be perceived.

Rupture of an intracranial aneurysm gives rise to symptoms which vary according as the blood escapes quickly or slowly. If quickly, the blood usually escapes into the meninges and causes sudden apoplexy with general paralysis, rapidly deepening to a fatal issue. If slowly, the symptoms are less sudden, and unilateral paralysis or convulsion may occur. This is especially the case when the blood escapes slowly into the cerebral substance; the unilateral symptoms commence suddenly, and gradually increase during a few hours or days, with or without initial loss of consciousness, but ending in fatal coma.

**TREATMENT.**—Little can be done in cases where intracranial aneurysm is suspected. Even when it is of syphilitic origin, drugs are powerless to alter the damaged and dilated vessel, since the change which has given rise to it is of the nature of a cicatricial transformation of the syphilitic tissue, on which drugs can no longer exert the influence they possess over the earlier stages of the newly-formed growth. Hypodermic injection of ergotin ( $\frac{1}{8}$ -grain) has been recommended by Langenbeck and advocated by Bartholow. Iodide of potassium may also be given in the hope of promoting coagulation within the sac, from the undoubted evidence of the power it has to effect this in aneurysms elsewhere. Rest is important. All causes of increased intravascular pressure, such as effort and low positions of the head, are to be avoided. The bowels should be kept regular. In rare cases where progressive paralysis of orbital nerves suggests the probability of aneurysm of the internal carotid, and a murmur renders the diagnosis certain, ligature of the common carotid may be, and has been, resorted to with success. It is, moreover, probable that intracranial surgery may be able to cope with aneurysms on some other vessels, but on this point experience has still to be gained.

(b) *Minute 'miliary' aneurysms* occur in the small arteries of the pia mater and substance of the brain (Virchow, Charcot, and Bouchard). They are met with almost exclusively in the second half of life, and increase in frequency with age up to about seventy-five, the less frequency of cerebral hæmorrhage in extreme old age making it probable that they are less common in very late life. They may involve vessels not more than the  $\frac{1}{100}$  inch in diameter, but are most common on vessels a little larger than this. The walls suffer fibroid degeneration of the outer and middle coat, commencing, it is said, as nuclear proliferation. The muscular tissue of the middle coat disappears, and the whole wall at the spot becomes dilated into a sacculated aneurysm, varying in size from the  $\frac{1}{25}$  to the  $\frac{1}{15}$  of an inch. These dilations have been found in all parts, but most frequently in the central ganglia, and next most frequently in the pons Varolii, the con-

volutions, the cerebellum, the medulla oblongata, the cerebellar peduncles, and the centrum ovale (Bouchard). They are to be found in most cases of cerebral hæmorrhage; and it is probable that such hæmorrhage is usually due to their rupture. Similar minute aneurysms of the retinal arteries co-exist in rare cases.

The rupture of a minute artery into its perivascular sheath distends this with blood, causing what has been termed a minute dissecting aneurysm. Such are frequently met with in cases in which the vessels are exposed to extreme pressure, as in death from asphyxial conditions; or in the increased tension in collateral vessels when vascular obstruction has occurred.

**SYMPTOMS.**—No symptoms are known to be associated with the existence of these minute aneurysms, and this fact explains the absence of indications of a liability to cerebral hæmorrhage. The symptoms of rupture are described under **BRAIN, Hæmorrhage** into.

**2. Degeneration.**—(a) *Of Arteries.*—The larger cerebral arteries are very common seats of the thickening of the inner coat, called by Virchow 'endarteritis deformans,' and which, when fattily degenerated, constitutes 'atheroma.' In the cerebral vessels the fatty change occurs quickly and frequently; and opaque yellow thickenings are the result. Only one or two of these may be present; or the change may involve the whole of the larger vessels at the base and extend for a considerable distance along the chief cerebral branches. The distribution of the degeneration may be symmetrical. It may coexist with a similar change in arteries elsewhere, or may be isolated. Degeneration of the cerebral arteries is common after middle life, being found in seven-tenths of the subjects examined (Bichat). It occasionally occurs much earlier, especially in cases of chronic Bright's disease and alcoholism. The exciting cause of this disease is probably the strain to which the badly supported cerebral vessels are exposed in consequence of their early origin from large trunks, perhaps rendered more effective by the deficient support from adjacent structures. The influence of strain explains the relation of atheroma to Bright's disease, in which the arterial tension is so much increased. It is not easy to explain the occasional freedom of these vessels from atheroma when this is abundant elsewhere.

The degenerated tissue occasionally softens and a cavity forms and opens into the vessel, so as to permit the formation of a dissecting aneurysm. More commonly it undergoes calcification. The result of these thickenings of the wall is generally to lessen the calibre of the vessel, sometimes to close it altogether, to cause local anæmia of the brain, and to favour the formation of a coagulum in the unnarrowed part beyond, with the result that

softening occurs in the part supplied by it. Where the change in the wall is slight, the new tissue may undergo fibroid change only, the wall remains thin, the artery may be dilated at the spot, but the tissue formed in this process is seldom extensible enough to permit a true aneurysm to result. Hence it is rare for arteries affected with atheroma to give way, and permit hæmorrhage to take place; the change in the wall differs from that which permits the extension (into an aneurysm) which precedes rupture. The minute arteries of the cerebral substance may undergo fatty degeneration, but they do not suffer from the change above described. There is not, in them, the formation of a new tissue that undergoes fatty degeneration, although their elastic elements may be replaced by the inelastic extensible tissue that permits the formation of an aneurysm.

**SYMPTOMS.**—Atheroma of the cerebral vessels leads to the symptoms of local anæmia of the brain, and is a common cause of the transient cerebral symptoms so frequent in the old. Tingling in the limbs of one side, and slight loss of power, may be due to this cause, and are frequently the precursors of an attack of hemiplegia from the closure of the vessel. It is possible also that the loss of memory and mental failure common in the old may be the result of more general interference with the supply of blood to the brain in consequence of this arterial disease, or of the deficient supply to certain parts subserving more general psychical functions. But caution should be observed in ascribing such senile symptoms to this mechanism; general symptoms are probably more frequently due to senile failure in the nutrition of the cells of the cortex.

**TREATMENT.**—Tonics, cardiac stimulants, and substances which, as cod-liver oil, promote the nutrition of the tissues generally, may be given with possible advantage. It is not likely, however, that any treatment can modify the state of the arterial wall, or that its tissue ever resumes, in any degree, its normal characters. The maintenance of the general nutrition in a good state may lessen the tendency to fatty degeneration and subsequent calcification of the tissue, and thus postpone or prevent the disastrous consequences of occlusion of the vessels. In this, however, we can only adjust our treatment on rational grounds, since the nature of the case precludes any possible evidence of beneficial result. Of especial importance is it, in any case in which there is reason to believe or to suspect the presence or future development of atheroma, to do all in our power to keep the arterial tension within normal limits, and to preserve the blood from the presence of the products of imperfect digestion and the accumulation of metabolic products, which favour thrombosis.

(b) *Of Veins.*—Degeneration of the walls

of the veins is much less frequent than degeneration of the arteries, perhaps on account of the less degree of pressure to which they are exposed. Disease of them is generally the result of inflammation communicated from without. Occasionally the veins of the pia mater may be found varicose in advanced life, and in one case recorded by Andral rupture of such a dilated vein was the cause of meningeal hæmorrhage.

**3. Embolism.**—**DEFINITION.**—The obstruction of arteries or capillaries of the brain by solid particles carried by the blood-current from some other part of the vascular system.

**ÆTIOLOGY.**—The source of the embolic particles is almost invariably situated between the pulmonary capillaries and the obstructed vessels, *i.e.* in the pulmonary veins, the left side of the heart, or the arteries. In arterial embolism it is necessarily so, since no particles large enough to obstruct even a small artery could pass through the capillaries of the lungs. In almost all cases the heart is the source of the plugs, a particle of fibrin being washed by the blood from a deposit on a diseased valve or in some recess (as the auricular appendix). Endocarditis, or chronic valvular disease, therefore, usually coexists with the embolism. Mitral stenosis is an especially frequent source of emboli, probably because the surface is commonly much altered, and the blood-current is in part slow (in diastole, allowing deposit), and in part very rapid (in auricular systole, detaching loose fibrin). Moreover the dilatation of the left auricle, and the imperfect degree in which the blood is expelled from it, frequently lead to a coagulum in the appendix. Disease of the aorta—atheroma or aneurysm—is the next most frequent source, and, less frequently, disease of the carotid or vertebral arteries, and coagulation in the pulmonary veins, large or small—the latter in some rare cases of inflammation and growths.

Particles obstructing capillaries may come from some softened atheromatous patch or fibrinous deposit, from pigmentary formations, or from deposits in ulcerative endocarditis. In the last case the obstructing material has a septic character, probably due to bacterial organisms, and the inflammation it causes may be suppurative.

Embolism occurs with equal frequency in both sexes, and at all periods of life, but most frequently between later childhood and middle life. This is explained by the fact that it occurs more readily from valvular disease due to endocarditis, than from that which results from the degeneration of age, but most frequently when the lapse of time has led to secondary changes in the valves damaged, or during second attacks of endocarditis—inflammation of valves previously diseased being especially prone to give rise to embolism. Hence most subjects of embolism have suffered or are suffering, from one of the

diseases known to cause endocarditis, and especially from rheumatic fever.

**ANATOMICAL CHARACTERS.**—Almost any of the cerebral arteries may be obstructed; but the vessels most prone to suffer are the middle cerebrals, in consequence of the more direct circulation into them. Next in frequency are the internal carotid and the anterior cerebral. Embolism is less frequent in the arteries to which the vertebals convey blood, but has been met with in most of them; even the basilar may be obstructed by a clot small enough to pass through the vertebals, but too large to enter the posterior cerebral. The cerebellar arteries are those least frequently affected. Obstruction of several vessels is sometimes found, having occurred at the same or different times. Embolism is more common on the left side, but the difference is not so great as is often stated, being not greater than as four to three. It is due to the more direct origin of the left carotid, into which a plug can therefore be more easily carried than into the right; hence it depends on the greater frequency of the occlusion of the branches of the internal carotid, and especially (because it is the vessel most often affected) of the middle cerebral. The left vertebral is also rather more often plugged than is the right, but embolism of this artery is too rare materially to influence the numerical relation between the vessels of the two sides in their liability to accident.

The plug is usually arrested at some spot at which the vessel is narrowed by a branch being given off. Here the fragment may be found, usually decolourised, and commonly closing altogether the lumen of the vessel. On each side of this is a secondary clot: the distal extends far into the contracted branches of the vessel, the proximal as far as the next large branch. The obstruction may lead to inflammation of the wall of the vessel at the spot, especially when the plug has been carried from a place at which inflammation is going on. The inflammation leads to change of texture and degeneration, fibroid or fatty; the former may permit an aneurysm to be formed, the latter may cause a thickened patch, in which calcification may occur. The inflammation may spread to the adjacent tissue, leading to induration around the spot. It is most intense when the plug comes from a valve the seat of active endocarditis, and may then determine a peculiar irritative character in the secondary inflammation, as is shown by the fact that optic neuritis sometimes develops in such cases.

The effect of embolism is to arrest the blood-supply to the part to which the artery is distributed, unless the blood can reach it by some other channel; the cerebral tissue, deprived of its circulation, undergoes necrosis and the necrotic softening described in another article. This result, however, is often

prevented by the anastomoses that exist, and as these are very irregular in the cortex of the brain, both in different persons and in different parts of the same arterial area, considerable variations exist in the extent of softening that results from occlusion of the same artery. But in the central ganglia anastomoses are absent, and softening uniformly follows. Thus embolism of the middle cerebral may cause softening of the corpus striatum, and the cortex supplied may be intact, softened in irregular areas, or totally destroyed. Capillary embolism also causes softening, and when the obstruction is from a septic source, 'metastatic abscess' may result. Thus cerebral abscess results from suppurative disease in the thorax and other parts. One of the rare curiosities of pathology is the production of such abscess by the embolic obstruction of the vessels with fragments of *oidium albicans*. A plug of fibrin sometimes quickly breaks up, and is carried into the capillaries, so that the artery is pervious, although the brain-tissue it supplies is necrosed. Necessarily, when the capillaries are obstructed, a collateral blood-supply is of no avail to prevent softening.

For the SYMPTOMS, DIAGNOSIS, and TREATMENT of cerebral embolism see BRAIN, Softening of.

4. **Rupture.**—Rupture of cerebral arteries is common and is the cause of cerebral hæmorrhage; and rupture of capillaries is not infrequent. Rupture of veins is extremely rare, except as the result of injury.

(a) *Of Arteries.*—The proximate causes of rupture are weakening of the arterial wall, and increased pressure within the vessel. The conditions which give rise to these two factors are the remote causes of rupture. The actual rupture is commonly due to a temporary sudden excess of intravascular pressure.

**ETIOLOGY.**—The wall of the vessel is weakened, especially by degenerative disease—chronic periarteritis, or (rarely) simple fatty degeneration. Aneurysmal dilatation and thinning may have resulted from the chronic change. In some diseases attended with a tendency to extravasation (purpura, hæmophilia, &c.) it is probable that the vascular walls undergo rapid degeneration (in the acquired), or are unusually thin (in the congenital) maladies. Defective external support, from atrophy of the brain, causing increased size of the perivascular canals, was formerly thought to be a potent cause, and is now perhaps underrated. The mobile perivascular fluid which surrounds the vessels must afford a less efficient support than cerebral tissue, and sudden variations in the size of these canals will expose the vessels to sudden differences in the amount of tension to which their walls are exposed.

When vessels are much weakened, they may rupture when the extravascular pressure

is at, or even below, the normal; very commonly, however, there coexists increased pressure. Loss of arterial elasticity leads to a jerky pressure. Arterial degeneration, and still more constantly, arterial contraction, in Bright's disease, cause increased tension by obstruction; and the hypertrophy of the heart, which develops to overcome the obstruction, adds materially to the pressure within the arteries. Hypertrophy to overcome an obstacle near the heart has probably no influence in causing rupture of cerebral vessels.

The immediate cause of rupture is generally some temporary increase of the blood-pressure due to effort—as in cough, straining at stool or vomiting; excited action of the heart; suddenly developed heart- or lung-disease obstructing the circulation; local obstruction to return of blood; contraction of the arterioles, general or local; or the action of gravitation in the recumbent posture. The last two causes, acting together, probably determine the frequent occurrence of rupture during sleep.

The conditions which produce these proximate causes are the remote causes of rupture. The most efficient are those which determine weakening of the vascular wall, and have already been spoken of (*see Degeneration*). Age is an important element—rupture is most common after fifty, but may occur from local vascular disease at any age. Hereditary predisposition is effective by a tendency to early degeneration, and is often very conspicuous; several members of the same family die at the same time of life from this cause. Position of degeneration is probably largely influenced by the distribution of the vessels; and the latter may be hereditary, as the retina sometimes strikingly shows us: the same distribution of the retinal artery may be seen in parent and child. So, too, correspondence in the way in which the cerebral arteries of the two sides divide and are distributed may be observed to coincide with a close correspondence in the position of spots of visible degeneration, and doubtless also determines a like symmetry in the *invisible* degeneration of these aneurysms; hence rupture sometimes occurs at the same place in each hemisphere. Sex tells probably by exposure to the greater pressure entailed by muscular effort (men suffer from rupture twice as frequently as women). Alcoholism leads to early degeneration. But the most efficient predisponent is Bright's disease, which leads to great intravascular pressure, and weakens the cerebral vessels by causing degeneration. It is probable that some acute diathetic diseases in which rupture is common act in a similar manner.

Some of the cerebral arteries give way more frequently than others, especially the arteries of the corpora striata and pons Varolii. This seems due (1) to their origin

at right angles from vessels of very considerable size (basilar and middle cerebral), and their consequent exposure to the full pressure within the parent trunk. (2) To their 'terminal' character, which precludes collateral relief when a general increase of intra-arterial pressure augments disproportionately that in an artery in which it is normally above the average for the size of the vessel. One artery, which very frequently gives way, passes from the middle cerebral through the anterior perforated spot, outwards between the island of Reil and the lenticular nucleus, the outer part of which it perforates, and then passes through the white 'internal capsule,' between the lenticular and caudate nuclei, to ramify in the anterior part of the latter. The arterioles supplying the convolutions on the surface of the brain are not often ruptured, except from injury. They are exposed much less directly to the blood-pressure, and sometimes possess considerable anastomoses.

**SYMPTOMS.**—The consequence of rupture of an artery is cerebral hæmorrhage, the symptoms and treatment of which are described elsewhere (*see* BRAIN, Hæmorrhage into). In traumatic laceration of the brain the arteries are torn, and often cause much hæmorrhage.

(b) *Of Capillaries.*—The minutest arteries, and veins, and the capillaries rarely rupture, except when exposed to sudden pressure by venous thrombosis, when the obstruction in the part from which the vein proceeds may determine numerous capillary hæmorrhages into the cerebral substance. In general venous congestion, as in asphyxial states, such hæmorrhage may occur; but a more frequent result is rupture of a vessel within its perivascular sheath, which thus becomes distended with blood.

For SYMPTOMS *see* BRAIN, Hæmorrhage into.

5. **Syphilitic Disease.**—The arteries of the brain are occasionally diseased in constitutional syphilis, between the first and twelfth year after infection, but occasionally in the earlier or later stages of syphilis. The large arteries at the base are those commonly affected; very rarely a similar change invades the minute arteries of the central substance. Usually more than one vessel is affected. It is generally the result of the acquired, but sometimes of the congenital malady. The frequency of the lesion is not known, since it is only recognised when the affected artery becomes occluded or greatly narrowed by the change. The disease may occur without this consequence and probably does so not rarely, and is removed by treatment adopted on account of simultaneous manifestations in other situations. The wall is thickened at circumscribed areas by a fibro-nuclear growth, which causes a nodular projection on the exterior, and diminishes also the calibre of the vessel. The structure

of the growth resembles that of syphilomata elsewhere. It begins by a nuclear proliferation between the inner coat and the elastic lamina, and it attains its chief development in this situation, the elastic lamina being pushed outwards and the lumen of the vessel obliterated. The middle coat may ultimately disappear. Vessels may form in the substance of the growth, and may subsequently be obliterated, leading to the irregular fatty degeneration so characteristic of these formations. The disease is sometimes symmetrical on the arteries of the two sides. The growth or secondary thrombosis occluding the vessel, softening may result in the area of brain-tissue supplied by it. Since the symptoms due to the occlusion are usually sudden, it is probable that the final arrest of the diminished blood-supply is always through the agency of thrombosis. As in other forms of arterial narrowing, when a certain degree of constriction is reached, the condition of the circulation in the vessel beyond must of necessity be such that clot forms on the altered wall, and at last the blood-flow suddenly ceases. The softening resembles in its occurrence and characters that which is produced by degenerative changes in the arteries, but is more varied in its seat, and it affects younger persons. The diminished elasticity of the diseased wall, when the thickening is slight, may permit the dilatation of the vessel into an aneurysm. Possibly the same result may follow the fatty degeneration of the new tissue.

**SYMPTOMS.**—No symptoms are produced by the arterial disease until it causes local anæmia or softening, the symptoms of which resemble those due to other causes.

**TREATMENT.**—This is that of the later stages of syphilis. It must be remembered that the removal of arterial disease may not restore the damaged cerebral tissue.

6. **Thrombosis.**—Thrombosis—the coagulation of the blood *in situ*—may occur in the cerebral arteries; or in the cerebral veins and sinuses.

(a) *In the Arteries.*

**ÆTIOLOGY.**—The causes of arterial thrombosis are the following:—

(1) An alteration in the wall of the artery, by which the blood comes in contact with an abnormal surface. The most common condition is atheroma, and hence arterial thrombosis is most frequent when atheroma is most common—in the old. Syphilitic disease of the artery sometimes leads to it. Much more rarely it is caused by an arteritis, spontaneous, or the result of adjacent inflammation or traumatic damage.

(2) Retardation of the blood-current. This may result from weakened action of the heart in debilitating diseases (as phthisis and cancer), and in extreme fatigue. It may be part of the effect of a convulsive fit; or it

may be caused locally by the arterial diseases mentioned already, which lead to narrowing and loss of elasticity.

(3) An increased tendency of the blood to coagulate. This is seen in many diseases, especially in marasmic states in young and old (such as are caused by phthisis and cancer), in acute rheumatism, and in the puerperal condition. A slight cause then suffices to produce coagulation, and the weak heart, so common in many of these conditions, may cause sufficient retardation of the blood-current.

(4) Lastly, thrombosis in an artery may be secondary to its complete or partial obstruction by an embolus.

**ANATOMICAL CHARACTERS.**—The arteries occluded may be one or several, and large or small. Thrombosis occurs most frequently in the internal carotid, middle cerebral, vertebral, and basilar arteries, or their branches. It is not uncommon for a clot to form in a branch that comes off at a diseased spot, while the main artery continues pervious. The orifice of the branch is narrowed so that enough blood does not enter to maintain the circulation in the part beyond. The vascular wall may present any of the local causal conditions already mentioned, or it may be healthy. After a time thickening from secondary arteritis occurs. Within the vessel is a coagulum which usually fills its interior, and is adherent to the wall. It may not fill the vessel, either because originally imperfect, or because the clot has shrunk. A recent quickly-formed coagulum is red, but after a time it becomes pale and yellow. A slowly-formed coagulum is pale, and may be laminated. A secondary clot usually forms far into the contracted distal branches, and on the proximal side as far as the nearest large branch. Ultimately the clot may, rarely, soften, the channel being sometimes re-established. More commonly it undergoes calcification, or, with the artery, contracts and becomes atrophied. The brain-tissue, in which the artery was distributed, may be at first anæmic, but quickly becomes congested. It ultimately undergoes softening—red, yellow, or white, according to the amount of vascular distension. If the collateral circulation is free, it may remain unsoftened.

**SYMPTOMS.**—Where chronic arterial disease is the cause of thrombosis, it may be preceded by the symptoms of local cerebral anæmia (see BRAIN, Anæmia of). The thrombosis itself leads to the symptoms of loss of function in the part to which the artery passed. The onset of these symptoms is slow or sudden, according to the rapidity with which the coagulum forms; and their degree depends on the size of the vessel occluded, its position, and its relation to other vessels which may supply blood to the area involved. Thrombosis of a small vessel in the

cerebral substance usually leads to transient brain-disturbance, headache, vertigo, tingling, and temporary weakness in the limbs, which soon pass away if a collateral circulation is established; more slowly, if softening ensues, by compensatory action elsewhere. The occlusion of a large vessel causes commonly more marked symptoms. Complete hemiplegia is frequent, and its onset may be marked by loss of consciousness. See BRAIN, Softening of.

**DIAGNOSIS.**—The diagnosis rests on a combination of the symptoms of local cerebral disease with the causal condition—vascular mischief (indicated by probable age, degeneration elsewhere, or syphilis), and with conditions leading to relaxation of the blood-current, or increased coagulability of the blood. The diagnosis is rendered more probable by the symptoms if slight being transient, if severe being of gradual onset, and, whether slight or severe, being preceded by the premonitory indications of local cerebral anæmia.

The PROGNOSIS and TREATMENT of thrombosis in arteries are considered under its consequence, BRAIN, Softening of.

(b) *In Cerebral Veins and Sinuses.*

**ÆTIOLOGY.**—Thrombosis in sinuses may be primary, and due to changes in the constitution or the circulation of the blood; or secondary, and due to local causes inducing coagulation directly at the spot affected. Secondary thrombosis is the more common. The same conditions of retarded circulation and altered blood-state which permit coagulation in arteries, favour it also in veins, and it is often seen in such conditions as phthisis and cancer, and especially in marasmic states in children. Hence the primary form is most common in children, but the secondary form occurs with nearly equal frequency through life. Local retardation of the circulation from narrowing of the sinus, or compression of the jugular vein, occasionally assists. The blood-current in the front part of the longitudinal sinus is one of extreme sluggishness, on account of the physical conditions of the circulation. Local change causing coagulation is usually the extension to the sinus of adjacent inflammation, or of a clot produced in a tributary vein by such inflammation. Caries of the bones of the skull, especially of the temporal bone, and meningitis are common causes. Lastly, injuries of the skull involving the sinuses sometimes cause coagulation in them.

**ANATOMICAL CHARACTERS.**—Of primary thrombosis the superior longitudinal sinus is the most common seat, and thence the clot spreads into the veins on either side, and often also into the lateral sinuses. When secondary, the thrombosis occurs in the sinus nearest to the local mischief; in disease of the petrous bone, the lateral sinus is usually involved. The sinus is distended by firm

clot, commonly (not invariably) adherent, sometimes in concentric layers. The walls of the vessel are healthy when the thrombosis is primary or secondary by extension of clot, but thickened and brittle when invaded directly by adjacent inflammation. After a time the clot may soften and break down.

The consequence of venous thrombosis is local arrest of the blood-current, the tributary veins and capillaries becoming enormously distended with blood and rupturing, and the cerebral substance being crammed with minute capillary extravasations which often coalesce. The condition is frequently seen in the convolutions. Blood is also effused into the meshes of the pia mater, and into the subarachnoid space. Into the looser tissues and into the ventricles serum may escape. Thrombosis of the veins of Galen is one cause of ventricular effusion. Ultimately the brain-tissue, the seat of the ischæmic congestion, undergoes softening, first red, and then yellow or white. Occasionally the softening of the clot leads to pyæmia.

**SYMPTOMS.**—The symptoms vary in their character. Sometimes they are at first those of mental excitement, namely, intense headache and muscular spasm, shown as contractions in the limbs, or as convulsion, often beginning locally, according to the position of the congestion. These symptoms, after one or several days, are succeeded by those of depression; with coma, and dilatation of pupils. In other cases the coma may come on suddenly, and the stage of excitement, above described, may be absent or little marked. Now and then hemiplegia slowly comes on, without initial loss of consciousness, and develops to a complete degree in the course of a few hours or a few days. Convulsions, when they occur, often continue till death. When the superior longitudinal sinus is plugged, epistaxis, œdema of the forehead, and exophthalmos have sometimes been observed. When the lateral sinus is obstructed, there may be painful œdema behind the ear, and the jugular vein on that side has been noticed to be less full than on the other.

**DIAGNOSIS.**—The diagnosis rests on the occurrence of severe cerebral symptoms in association with a causal condition, constitutional or local.

**PROGNOSIS.**—This is always serious, death being, in most cases, speedy.

**TREATMENT.**—The indications for treatment are mostly causal. In primary thrombosis stimulants and nutritious diet are necessary, and tonics if they can be taken. The patient should lie with the head and shoulders a little elevated, and care be taken that the neck is not bent, and that there is no constriction of it by tight clothes. In secondary thrombosis, occurring in robust individuals, leeching or cupping is recommended;

purgatives should be given; and in the less severe cases a blister may be applied to the neck, and the utmost care taken to afford free exit for pent-up inflammatory products. Pain and convulsion are relieved most effectually by cold to the head.

W. R. GOWERS.

**BRAIN FEVER.**—A name popularly applied to any kind of febrile state in which symptoms of cerebral excitement are prominent; as well as to cases of inflammatory disease of the brain or its membranes.

**BREAK-BONE FEVER.**—A synonym for dengue. See DENGUE.

**BREAST, Diseases of.**—SYNON.: Fr. *Maladies de la Mamelle*; Ger. *Brustdrüsenkrankheiten*. This subject will be treated under the following divisions:—

- I. *Diseases before puberty*, in both sexes.
- II. *Diseases about the age of the establishment of puberty; and after that period*, in the female.—(A) in the *active* state of the gland; (B) in the *passive* state.
- III. *Diseases affecting the rudimentary organ in the male.*
- IV. *Diseases of the nipple.*

The diseases of an organ composed essentially of glandular structures have here to be described. The mammary gland is classed with those termed racemose; but it differs from every other organ in the body of a similar class, inasmuch as it only arrives at maturity when its function is to be subservient to the nourishment of the offspring. In its perfection it appears, normally, only in the female sex, and even then it does not become developed until the internal organs of generation are capable of performing their functions.

Hence, to describe systematically the diseases of the breast, it is requisite to treat of them not only in reference to sex, but also in relation to the different periods of life at which certain diseases appear. Briefly then, from a histological point of view, they occur: (a) in the rudimentary state of the gland; (b) in its mature state; (c) when it has become a secreting organ; (d) during a state of degeneration.

I. In the *rudimentary period* of the gland the tissues composing it are rarely liable to morbid derangement. Usually, soon after birth, especially in male infants, the rudimentary nipple and the skin of the region within the zone of the areola become slightly elevated and of a pink hue. In some infants a secretion, slightly milk-like, oozes from the ducts. In this state, the injudicious rubbing practised by the attendant excites inflammation, which, if not arrested by desistance from that pernicious interference, may advance to suppuration. When that happens, the abscess must be incised and treated on ordinary principles.

II. *About that age, in both sexes*, when the development of the generative organs

advances with greater rapidity to maturity than in early life, the breast-gland enlarges, and may be painful, thus causing anxiety to the individual. Development usually takes place symmetrically in the female; but, when the gland of the one side takes precedence of that of the other, the circumstance need only be regarded as a departure from the ordinary rule, for no trouble will result, and in due time both will attain their normal proportions. In the male, pain or uneasiness sometimes occurs for a few days about this period, very often excited by the pressure of the dress. The removal of this cause is sufficient to arrest further mischief.

In the female the development of the breast having reached maturity, the gland becomes associated by sympathetic influences communicated through nervous stimuli with the functions of the pelvic generative organs. This physiological fact should always be remembered when investigating the nature of the morbid affections of this organ.

From a clinical point of view, it is essential to examine the diseases of the breast under the two states before mentioned, namely—(A) whilst the gland is undergoing metamorphosis into a secreting organ, and during lactation; (B) as a mature gland, but passive as regards its function.

Glancing at the various morbid states of the body of the gland as a whole, they may be divided, primarily, into two groups:—the *functional* derangements; and the *structural* or histological diseases.

We will now proceed to discuss these affections, as they are presented at the different periods mentioned above.

A. After conception the breasts soon begin to enlarge, and minute pisiform indurations may be felt at their borders and surface. Occasionally, but very rarely, this normal increase in bulk is attended with considerable pain and irritation extending throughout the nervous relations of the gland. The pain is referred to the back, neck, inside of arms, shoulders, and side of thorax—over, in fact, the area of distribution of the branches of those intercostal nerves which send filaments to the breasts. It affords a good example of reflected irritation. This state usually occurs after the first conception, and in women of excitable, nervous temperament. Attention to ordinary hygienic measures during the progress of the metamorphosis of the organ into a secreting gland, with its accomplishment, affords relief to the pain. In large, lax, pendulous breasts, the separate lobes of which each is composed may excite apprehension of the existence of a tumour. But tumours composed of new tissue are so extremely rarely developed during pregnancy, that the greatest caution must be exercised in the diagnosis of their nature.

**Agalactia.**—Very rarely, no change whatever in the breasts accompanies pregnancy,

under which circumstances there is an absence of the secretion of milk after parturition.

**Inflammation of the Breast.**—SYNON.: Mastitis.

**ÆTIOLOGY.**—Before lactation, a acute inflammation of the breasts is very uncommon. After lactation, on the contrary, it is very frequent. This morbid state is often the result of carelessness or ignorance on the part of the nurse. The slightest unusual fulness or ‘knottiness’ discovered after the infant has been sucking, and when the ducts and their terminal secreting vesicles should be empty, requires immediate attention. Congestion of a lobule or lobe with milk produces the nodule, and the cause of the impeding to its escape should be sought for. The state of the nipple is generally the origin of the difficulty. Either the orifice of a duct may be obstructed by epithelium, or a superficial ulceration exists around it. The morbid or defective states of the nipple are the most fruitful causes of inflammation and its results in the breast. Prophylactic measures should always be instituted when there is reason to fear that a defective development of the nipple will interfere with the free flow of the milk. Even with some mothers it would be advisable to resign the duty of suckling, rather than subject themselves to the almost certain misery arising from persistent and ineffectual attempts to do so. Inflammation, generally passing on to suppuration and abscess, either within the body of the breast or on its surface, is most frequent in primiparæ, and within the first month after parturition.

**SYMPTOMS.**—First, hardness is felt, ‘a knot,’ in some part of the substance of the organ; this enlarges, and may attain to considerable dimensions before causing pain or even uneasiness. Next, pain is felt during suckling; this increases each time the infant sucks, and ‘the draught’ is produced. The integuments then become pink, and afterwards red, tense, shining; more or less of the breast feeling very inelastic, firm, prominent, and heavy. Pain is now often very severe, and great constitutional disturbance is excited. In the centre of the redness the skin becomes of a purplish tint; around this it is œdematous; and with the finger, at the centre of the purple zone, a slight depression and softening spot can be detected. An abscess now exists, and fluctuation is more or less marked in proportion to the quantity of pus. At the purple centre the cuticle has probably by this time separated from the cutis; and a vesicle containing serum, either yellow or slightly tinged with blood, indicates that ulceration of the cutis is proceeding, and that the pus will soon escape. The above is a brief description of the objective signs indicating the morbid processes noticeable in all cases of local inflammation advancing to and terminating in suppuration and abscess. It is not possible to state with any degree of exactness the

period of time required for the accomplishment of these definite changes. It varies according to so many local and constitutional circumstances, that it would be idle to attempt to predict any certain definite period or stage for each phase. It will be more useful to describe the treatment by which the progress of the disease may be arrested or limited, and its painful course mitigated.

**TREATMENT.**—Great attention should always be given to the nipple of primiparæ. In many women, this important division of the gland is very small and undeveloped, perhaps only on one side, so that the infant, especially if not very strong, has great difficulty in obtaining sufficient milk to appease the appetite, and its efforts cause pain in the part. This circumstance induces the mother to prefer suckling most with that breast the nipple of which is perfect, and the infant soon appreciates the advantage of that side. Consequently the gland-tissue of that breast having the imperfect nipple becomes congested. Every time the infant sucks it becomes worse, more and more pain and irritation are excited, the orifices of one or more of the ducts in the nipple become blocked, and perhaps the infant refuses to suck the breast. But the gland becomes more and more distended, the nipple deeply buried, until at last suckling is impracticable. Probably none of these increasing troubles have been stated to the attendant surgeon; and, when he is consulted, he finds the breast to be in the state above described. The perfect development of the nipple should be always a subject of anxious solicitude on the part of the obstetric practitioner. If this organ be imperfect, precautions should be taken to prevent the gland itself from becoming congested, and if the infant cannot draw the milk sufficiently, some mechanical means should be employed to effect this object. The nipple itself should be examined. If its end be more than usually coated with secretion, or the openings of the ducts seem to be obstructed with an excess of epithelium, attempts may be made to remove it. If abrasions, ulcers, 'cracks or chaps,' are visible between the rugæ, a soothing application, or a weak solution of carbolic acid, should be used. Ablution with warm water, or the contact of a little moist cotton covered with gutta-percha tissue, is preferable to the dressing; or, if there be much secretion from the glands on the nipple, after cleansing its surface, some dry powder is beneficial, such as carbonate of magnesium, oxide of zinc, or starch powder.

When actual congestion of the gland-tissue exists, mechanical means should be used to reduce it. Supporting the gland with strips of plaster and a bandage is sometimes very useful. When inflammation is excited, local applications of warmth and moisture are indicated, and the constitutional condition of

the sufferer demands special attention. The application of glycerine of belladonna and a suitable support of the breast are also of great service. When suppuration has taken place, its relation to the adjacent parts and the exact size of the abscess when formed should be carefully ascertained. There is great diversity in the progress, duration, and sufferings of the patient, depending upon the locality of the pus. It may be situated over the body of the gland, within it, or beneath it.

When *overlying* or superficial to the body of the breast, the course of the disease is rapid. In those cases the constitutional disturbance is usually trifling.

Both the local and constitutional symptoms are much more severe when inflammation affects *the body* of the gland, and pus collects between its lobes. The progress of the disease is tedious, pointing of the pus is slow, and the exact spot at which it may reach the surface is for a long time doubtful. In the majority of cases it makes its way between the ducts, and reaches the surface near the areola or within its area; usually to the sternal side of the nipple, where the gland-tissue is thinnest. In either case, as soon as the presence of pus is ascertained, it should be evacuated, by means of a short incision radiating from the nipple. This must be kept open by as large a drainage-tube as the incision will admit, and the strictest antiseptic treatment should be employed. The omission of the latter is very likely to lead to burrowing of pus and the development of troublesome sinuses.

When the abscess forms *beneath* the breast the local appearances are quite characteristic. The gland itself seems little involved, but it is pushed prominently forwards and seems to rest upon a cushion of fluid. To the touch the elasticity of the swelling is very striking, and, without producing additional pain, a slight bulging of the walls of the abscess may sometimes be produced at the periphery of the gland when, with the palm of the hand and outspread fingers, compression is made from the front backwards against the thorax. The patient should be recumbent. The pus in these cases often points somewhere around the borders of the body of the gland, and must be evacuated at the earliest possible date.

During the time occupied in the formation of a mammary abscess and its local treatment, the constitutional powers of the patient must be well supported, and the general health maintained by every means.

The *sequelæ* of suppuration in an organ composed of so much connective tissue, and endowed with its peculiar function, sometimes cause great trouble; they are, however, much less frequently met with now than before the introduction of the antiseptic treatment. They are protracted induration, sinuses, and fistulæ, through which last the

milk persistently escapes. Induration of the whole or part of the breast subsides when lactation ceases, and the organ in due time resumes its healthy state. Sinuses and fistulæ may require incisions, but the ordinary methods for their cure should be adopted before having recourse to a treatment often involving much subsequent deformity.

At the time for weaning the infant inflammation rarely occurs. Considerable milk-congestion of the secreting structure may sometimes produce irritation and inconvenience, to be relieved by mechanically drawing the milk in just sufficient quantity only to diminish the fulness.

**Galactoele.**—An accumulation of milk, to which the above term is given, forms a tumour in the connective tissue of the organ, and results from the bursting of a lactiferous tube. The swelling always appears first during lactation. It may vary in size from time to time; sometimes enlarging rapidly as suckling goes on. Two varieties are met with. In the one form there is a single swelling near the nipple, quite superficial, and quickly recognisable by its objective signs. In the other there may be several swellings distinguishable in the substance of the gland, as well as on its surface, all of comparatively small size, very firm and globular. In the same gland they vary very much in size, and in the degree of resistance they offer to manipulation. The discrimination between these tumours and others in the breast is easy, if the surgeon is able to ascertain with exactness that the swelling appeared somewhat suddenly during suckling, and that its size varied conformably with that function.

In cases of long standing, the contents of the cyst become solid in proportion to the quantity of the fluid constituents of the milk absorbed, and the cyst-wall itself is very often rigid and even may become gritty.

**TREATMENT.**—The treatment of this malady consists in cutting into the cyst, removing its contents, and drainage.

B. The diseases affecting the mature gland, in its *passive* state, from the age of puberty to that period of life when the *catamenia* cease, may be grouped as follows:—the *functional* affections, or those which are characterised by changes in the secreting portion of the gland, accompanied by more or less induration, inconvenience, and pain; the *structural*, or those diseases characterised by some new-growth, which is altogether a superaddition to the organ, and—growing within its sphere of nutrition—often resembles, more or less, the gland-structure in composition; and others the minute elements of which are nucleated cells of various shapes, definitely and diversely arranged.

In this state of the breast *inflammation* rarely occurs. Nevertheless, both the acute and chronic varieties of that morbid process

terminating in abscess are met with, and careful discrimination is necessary to avoid mistaking such diseases for new-growths, especially in patients above forty years old; for the amount of matter in a chronic abscess is often small, while the surrounding inflammation is often great and not infrequently an enlargement of the axillary glands, and some retraction of the nipple, make the diagnosis from cancer very difficult. The history of the case, tactile examination, and the variations occurring during the progress of the affection, commonly suffice for the detection of such cases. The treatment should be the same as for abscess in general.

**Functional Derangements.**—The functional derangements of the breast in its passive state demand special attention. They are characterised by a peculiar activity of its secreting portion, which undergoes structural changes of a specific kind. When the tissues composing a mature but perfectly passive gland are examined with the microscope, the caecal terminations of the ducts are found to be small, and little else than fibre-tissue is seen. Here and there cæci may be detected containing minute aggregations of epithelium. But when, under some sympathetic excitement, with derangement of the functions of the pelvic generative organs, the secreting cells of the gland become active and are distended with epithelium, they induce more or less enlargement of the breast. This condition of things is called *chronic lobular mastitis*, and should perhaps more properly be included amongst the inflammatory diseases of the organ. But of such affections there is this important fact to be noticed. The whole breast need not be necessarily involved. On the contrary, one lobe only may be excited, and when this occurs the existence of a tumour is declared. When, after excision, such enlarged lobes are carefully examined with the microscope, normal gland-tissue is seen, the caecal ends of the ducts are readily recognised, and their immediate association with the excretory ducts may be observed. The former are gorged with epithelium; and true gland-tissue, less its peculiar secretion, has been developed.

**SYMPTOMS.**—Associated with this state of the tissues of the breast, the patient complains of pain, both locally and spread over a very wide area. To express as briefly as possible the superficial regions affected and over which pain is felt, the reader must be reminded of the distribution of the descending cutaneous branches of the cervical plexus, and of the lateral and anterior cutaneous branches of the second, third, fourth, and fifth intercostal nerves. From these, special filaments are distributed to the breast; and to the site of exit of one or more of them at the intercostal foramina, the source of the pain is referred by the patient. The skin of the neck, shoulder, side of thorax, and inside

of arm receives filaments from the same source. Hence an explanation of the widely-diffused pain.

It is of the first importance to discriminate between this state of the gland-tissue and substantial new-growths, especially because the latter cannot be removed by natural processes, whilst the former most probably will be. The objective signs are the following: to the touch the excited gland-tissue is nodular, irregular over its surface, much identified and mingled with the body of the organ. If the whole breast be large and relaxed, the tips of the fingers may be insinuated between the borders of the indurated lobe and the lobes not affected. If the entire body of the gland be morbidly firm, it feels like a disc-shaped mass lying on the thorax, under the borders of which the fingers can be pressed. Occasionally, at one or more spots along the periphery of the gland irregular nodules are perceptible, projecting into the connective tissue around them. When one lobe is affected, the shape of the induration may be detected corresponding with that of a lobe, namely, broad at the periphery and gradually narrowing towards the areola. Pain as a subjective indication is of great assistance in forming a diagnosis of these cases; but the source and course of the pain must be carefully traced. Generally manipulation of the induration produces increased pain; occasionally touching the induration, even however gently, is intolerable, and persistence in or repetition of the act is strongly opposed by the sufferer. Light pressure should be made over the points of exit of both the anterior and lateral branches of the intercostal nerves, when the pain excited thereby will correspond with the nerve-filaments of the affected lobe. Pressure along the upper dorsal spinous processes usually excites pain also. The morbid affections above described occur in single women, married but sterile females, and young widows, at ages between twenty years and forty. More or less disturbance of the catamenia co-exists, either in relation to the frequency or quantity of the discharge. The patient complains of languor and inability for bodily or mental exertion; and she is desponding and often alarmed for the possible consequences of the affection suggested by sympathising friends. She is irritable, restless at night, loses appetite for food, as well as all desire for social enjoyments, and becomes highly susceptible and emotional.

**DIAGNOSIS.**—An exact diagnosis of these histological changes may be made if the manipulator examines the organ methodically. He should, first, gently grasp the induration between the thumb and fingers, when it will be distinctly appreciable. Afterwards, placing the palmar surface of the fingers over the surface of the breast and gently pressing backwards against the thorax,

the induration cannot be detected. Should there still remain any doubt on the subject, let the patient recline on her opposite side on a sofa, and in this posture, if there exists a substantial new-growth, the integument is usually elevated by it.

**TREATMENT.**—The treatment of these cases consists in attention to the general health. Every hygienic direction should be enjoined, and such medicines administered as conduce to its improvement. Local soothing applications are usually futile, and, except in those cases of extreme pain, are not advisable; but strapping with belladonna plaster may give relief, and will serve to support the breast and to prevent manipulation of the part, and the consequent frequent recurrence of the patient's thoughts to it. Should the gland be heavy and pendulous, a suspensory bandage, as thin as possible, may be adjusted.

**New Formations.**—We shall next describe the diseases of the mature gland arising from the development of new formations—either of tissues constituting new-growths; or of conditions causing collections of fluid of distinct and specific kinds. All of these may be thus arranged in three groups:—first, the *fluid* tumours; second, the *solid*; and, third, those composed of both *solid* and *fluid*.

**A. Cysts.**—The *fluid tumours*, commonly termed *cystic*, consist of a membranous sac with its contents. Now, calling to mind the histological divisions of the breast, and dwelling on the structural differences between its parts, the secreting apparatus and the excretory, there is little difficulty in assigning to the cysts their true histological affinities. Thus there are cysts associated with the secreting apparatus; others with the excretory, the ducts; and some due to the extravasation of the secretion of the gland, the milk, into the connective tissue. Effusions of blood also give rise to the development of cysts, either independently of other diseases or associated with them. Another variety of cyst is produced by the development of entozoa, notably of *echinococcus hominis*; and it must not be forgotten that cysts as well as abscesses may be developed in connexion with various kinds of tumours. An ordinary examination of the fluid derived from these cysts at once establishes the fact that heat and the admixture of nitric acid produce coagulation in that abstracted from some of them, whilst that from other cysts remains unaffected by the treatment. The cysts are thus divisible by the nature of their contents into two distinctly defined groups:—

a. Those containing fluid without the admixture of any coagulable element.

b. Those enclosing fluid which does contain coagulable material. Other characteristics of these two fluids are not less conspicuous.

From an objective point of view all cystic

tumours of this gland may be classed in two groups, thus:—first, those associated with its ducts, the evidence of which is afforded by the escape of fluid at the nipple; and secondly, those not connected with the ducts by any such evidence.

The following tabular arrangement will place before the reader, at a glance, all the varieties of cysts met with in the breast.

I. *Cysts associated with the ducts*, communicating and connected with them.

1. Containing milk.
2. Enclosing growths; with serum, coagulable and often tinged with blood:—
  - a. Adenoid growths.
  - b. Granulation cell-growth.
  - c. Cancer.

II. *Cysts not connected with the ducts*.

1. Surrounding effused blood.
2. Enclosing milk.
3. Simple cysts.
4. Entozoon cysts.
5. Investing growths; with serum coagulable, tinged with blood and containing cholesterine:—
  - a. Adenoid growths.
  - b. Granulation cell-growth.
  - c. Cancer.

1. We have already described, under the name *galactocoele*, a tumour observed during suckling and composed of milk. But the surgeon is occasionally consulted about a swelling which, at first sight, would seem to have no reference to that function. Nevertheless the milk, or all that remains of that secretion, constitutes its entire bulk. If exact inquiry be made, the patient states that a tumour has existed, unaccompanied by pain, from the period of the last weaning, perhaps not having been observed until the gland ceased to secrete, and that its size slowly diminished until a certain period, since which it has remained of unvarying bulk. This decrease is due to the absorption of the serum of the milk, whilst the solid parts remain. The cyst should be incised, the contents removed, and the wound should be drained.

Cysts containing the solid parts of the milk are sometimes associated with perfectly new growths of the glandular type.

2. True *sero-cysts*—that is to say, collections of serum circumscribed by a fibrous membrane—are frequently met with, and are most commonly associated with adenoid, granulation, and cancerous growths. They occur in the breasts of middle-aged women. The fluid which these cysts contain is sometimes quite clear, and of a yellow tint; at other times it is tinged with blood-colouring matter, and is turbid. It always contains some constituent coagulable by heat and the admixture of nitric acid. These are the exudation-cysts of the mammary gland.

3. Other cysts containing a fluid like serum, until its composition is carefully ex-

amined, are developed in the breast, and are probably associated immediately with the secreting part of the gland. For the sake of identification the writer would designate them *mucous cysts*. The contained fluid is not coagulable by either heat or acid. Its colour is brown, more or less inclining to a greenish hue, and is probably really that of altered milk; it is opalescent, of variable specific gravity—about 1020, rather greasy when rubbed between the fingers, and exhibits an alkaline reaction. When sufficient quantity is collected in a test-tube and allowed to cool, in a few hours the lowermost stratum of the fluid becomes clearer than the upper. The uppermost always remains opalescent. If a little of this last be examined with a microscope, oil-globules are seen, together with the bodies called colostrum cells. The greasy nature of the fluid can be detected by smearing a drop of it on a piece of glass.

The development of these cysts is not very common. We may here remark that they seem to have escaped the observation of surgeons, as no special notice of them occurs in the most recent monographs. Yet they are so distinctly separated from all the other cysts which are formed in the breast, in respect of the composition of their fluid contents, their progress, and their prognosis, that they constitute a marked, distinct, and isolated class. We meet with them in the breasts of single women, in married but sterile females, and in widows between forty and fifty years of age. Usually they are accidentally discovered in any quarter of the gland when about an inch in diameter. Their shape is globular or ovoid; to the touch elasticity is the main feature, and if sufficiently large and superficial, fluctuation may be detected. In some instances the tips of the fingers may be insinuated into a sort of furrow around them. Pain is rarely complained of. The treatment consists in emptying the cyst with a trocar and cannula. After this the fluid does not again form. These cysts usually appear singly and in one breast only, but the writer has seen a patient in whom they were multiple and on both sides.

4. Cysts containing blood—*hematomata*—are very rarely formed in the breast, except in association with some new-growth from which blood, or more often bloody serum, oozes. They may, however, occasionally occur independently of any new-growth.

5. True *entozoa-cysts* are developed in the breast. They are certainly rare, and cannot be distinguished from other cysts until incised. Extirpation by excision is the speediest means of effecting a cure.

6. Sebaceous cysts are occasionally met with in this region.

**B. Solid Tumours.**—We have next to describe the solid tumours. These are essentially new-growths of tissue superadded to the normal gland. Generally, therefore, a

characteristic feature of the existence of such growths is an increased bulk of the affected organ; another, the firmness or resistance to pressure with the fingers when contrasted with the group of tumours before described. The solid tumours have long been classed in regard to their local and constitutional effects, and their results on the life of the individual affected with them, into two groups—the *innocent* or harmless; and the *malignant* or life-destroying.

1. *Adenoma*.—In the first class are placed the growths more or less closely resembling in their tissues those composing the glandular structure of the breast. Various terms have been assigned to these tumours, namely, Chronic Mammary Tumours, Pancreatic Sarcoma, *Tumeur adénoïde*, *Corps fibreux*, *Hypertrophie partielle*, Mammary Glandular Tumour, Fibroma, Adenocoele. Their composition is chiefly fibrous tissue; the cæci or acini of secreting structures, with more or less distinctly marked traces of ducts, being interspersed throughout the mass. *See TUMOURS: Adenoma*.

Adenomata are developed in the breasts of young, unmarried women from the age of puberty upwards; rarely after thirty, but very commonly before that age. They may be intraglandular, occupying the substance or body of the gland, and having the normal gland-tissue investing them. In other instances they seem to be attached by a kind of pedicle either to its surface or margin. In every instance they are placed within the fascial investment of the organ. When attached, as just described, their remarkable mobility, slight lobulation of surface, and firmness, coupled with the youth of the patient, are sufficient indications of the harmlessness of their nature. Usually they occur singly and in one breast; they may be multiple and in both breasts. The only means by which they can be removed is excision. However large the tumour, its removal should be always attempted without cutting away any portion of the normal breast. In most cases this can be done, especially if the patient be youthful and the growth of medium size, even should it be developed in the body of the gland and extend through it to the pectoral muscle. After thirty-five years of age it is expedient to remove the breast as well. For these tumours, especially those which may be called soft adenoids, approach very closely to the cancers (*see TUMOURS*). A section shows a solid, uniform surface, divided into lobes and lobules by fibrous septa, sometimes slightly broken up by fissures or clefts in which there appears a little clear tenacious fluid. The growth is often very succulent, at other times only moist; its hue may be greyish, yellow, or almost white. Its vascularity is scarcely perceptible. The prognosis of these cases is invariably favourable.

2. *Fatty tumour*.—Lipomata or masses of adipose tissue are developed in the breast, or rather in relation with it, to speak with accuracy. They are characterised by the usual indications, and require no special mention.

3. *Nævus*.—It may be questioned whether nævus, or a growth of true trabecular vascular fibre-tissue, is ever developed in the substance of the breast, that is to say, in the gland-tissue, although the integument around the mamilla may certainly be so affected in early life before the development of the gland, and a subcutaneous nævus may exist at the site of the future organ.

4. *Sarcoma*.—Under this heading, as well as that formerly employed of 'fibroplastic tumour,' we include a group of new-growths composed of more or less well developed sarcoma tissue, in which are interspersed somewhat modified acini and ducts of the gland. They are developed in women of middle age, increase rapidly, and after excision are liable to grow again. They usually require complete removal of the breast.

5. *Colloid*.—Colloid growths are occasionally met with in the breast. They are true cancers which have undergone the usual colloid degeneration; and they must be treated on the same lines. They appear in middle life, and are not distinguishable from other solid tumours until after excision.

6. *Carcinoma*.—Carcinoma, commonly called cancer, is developed in the breast in two distinct forms. The first, and most common, is that variety which is due to infiltration of the normal tissues of the gland by the elementary cells of cancer. It constitutes the *scirrhous* variety. The second is that kind which is produced by the development of a mass of *soft, medullary* or *encephaloid* cancer-growth. Both varieties are met with in women after forty years of age, but the first much the most frequently. Previously to that age the disease is rare; from forty to fifty it is most commonly seen; and it becomes relatively less frequent as age advances. A larger number of married women are affected by it than single, and prolific women who have suckled their children are quite as prone to the disease as the sterile or those who have not suckled. For the histological structure *see* CANCER.

*SYMPTOMS*.—*Scirrhous* cancer commences in any region of the mammary gland, although most frequently perhaps in the axillary segment. A small, firm nodule is usually accidentally discovered, without the attention of the patient having been attracted to it by pain. Rarely, the whole organ is simultaneously infiltrated, but most frequently one lobe only is affected. The infiltration is often central in the body of the

gland, especially when the organ is atrophied, and the reverse occurs when the extreme edge of a lobe is affected at its periphery. The infiltration may steadily increase until the whole breast forms a rigid, solid mass; but most frequently the larger part of the organ remains unaffected. The disease gives rise by its contraction to much deformity of the region, to dimpling, corrugation, and irregularities of the otherwise rotund integumental surface. The nipple, just in proportion to the effect of the growth upon the ducts, becomes retracted or drawn towards the tumour. Such are the ordinary objective signs of infiltrating cancer in its early stage. The progress made by the disease is subject to very remarkable variations in different individuals, and the stage above described may be long delayed. In some cases many months or even years may elapse before the growth assumes any grave importance. Sooner or later, however, the integument over the growth becomes adherent to it, infiltrated, and red, and advances towards ulceration. An ulcer now forms, the edges of which are everted, ragged, and attached to the growth beneath. A hole extending into the tumour becomes deeper and deeper; ichorous discharges, more or less profuse, continue without much pain; and the patient becomes at last worn-out, or succumbs to the ravages of a cancerous growth in a vital organ.

The *encephaloid* variety commences in a small, circumscribed, globular nodule in the body of the gland; grows rapidly; separates the lobes of the organ; extends equally in all directions; and becomes adherent to the skin, which commonly sloughs and allows of a protruding, fungating mass.

In both varieties the axillary lymphatic glands sooner or later become involved in the disease, which may also spread to those in the neck and within the thorax.

**TREATMENT.**—Local applications exert little if any influence on the growth of cancer. The vital powers of the patient should be supported as much as possible by hygienic measures, and especially by ferruginous tonics. The removal of the primary growth before the contamination of the lymphatic system is of great importance; and the methodical removal of the axillary glands is to be strongly recommended. If the case have advanced too far for operative interference, and an open sore have formed, suitable antiseptic remedies must be employed.

**C. Mixed Tumours.**—To the group of mixed tumours belong:—1. Those composed of cysts, intracystic growths, and solid interspersed masses of new tissue. 2. *Granulation cysts*—cysts with growths attached to their walls, the elementary tissues of which resemble those of ordinary granulation-growths—whence the term applied to them. 3. Cysts,

so called, often formed upon the surface of cancers, in consequence of the slow exudation of serum from the growth itself.

1. Tumours of the first class belong pathologically to the group of adenoïd formations, and although they differ so remarkably in their external objective appearances, they are, when unalloyed with other growths, perfectly harmless. The sero-cystic disease of Brodie, and the proliferous cysts of Paget belong to this class.

2. The *granulation-cystic* growths constitute a class of themselves. It is only of late years that attention has been attracted to them. They are rarely met with, and when pure are unattended by untoward circumstances.

3. To the third class belongs a group of cases thoroughly cancerous in their nature, and differing only from the ordinary forms of that disease by the accidental formation of cysts. *See* TUMOURS.

**III. Diseases of the Male Breast.**—The male has sometimes a well-developed mammary gland, and the part is subject to the same diseases as the female. But the simple enlargement of the organ is harmless, and should not be interfered with. At the age of puberty the mammary region often becomes painful, owing in part to the pressure of the dress upon the mammilla and the rudimentary organ. Inflammation followed by suppuration has been observed at this time.

**IV. Diseases of the Nipple.**—A *defective formation* of the nipple is of grave importance, and when it exists measures should be adopted to assist its elongation. This is to be done by using an exhausting-glass, such as those employed to empty the gland of milk.

*Inflammation* and its effects produce much suffering, and at the period of suckling frequently excite deep-seated mischief. The small ulcers, called 'cracks,' 'chaps,' &c., which form between the rugæ on the apex and sides of the nipple, may be cured with an application of water-dressing, or by powdering the part with carbonate of magnesium or oxide of zinc, or with a weak solution of carbolic acid. Sir James Paget has described a chronic eczema of the nipple which may assume a malignant character.

*Pendulous cutaneous growths* occur on the nipple, and should be excised.

*Cystic follicular tumours* are sometimes seen within the zone of the areola. *See* NIPPLE, Diseases of.

JOHN BIRKETT.

**BREATH, The.**—The expired air, or what is familiarly termed *the breath*, is important both from an ætiological and a clinical point of view, and the object of the present article is to present a brief summary of the main facts relating to this subject,

with which, for practical purposes, it is necessary to be acquainted.

1. The *ætiological* relations of the expired air will be more appropriately discussed in detail under the general subject of *ætiology* (see *DISEASE, Causes of*), but a few of the more striking examples of the manner in which it affects the health may be given here. It is well known that the expired air, if re-breathed by the same individual without having been purified by a proper admixture with atmospheric air, will produce serious effects upon the economy, and will ultimately lead to death by asphyxia. Again, the breath of a number of persons collected together in an ill-ventilated place may prove injurious to such individuals; the impure atmosphere thus generated tends to lower the general health, to retard the development of the young, to increase the virulence of infectious diseases, and to predispose to pulmonary affections. Indeed, some writers regard re-breathed air as one of the most prominent causes of pulmonary phthisis. Further, undoubtedly the expired air is a most important channel by which the poison of certain infective diseases—for example, that of measles, scarlatina, or diphtheria—is conveyed from one individual to another. It has been affirmed that phthisis can be transmitted directly in this manner, but adequate proof of this statement is entirely wanting.

2. From a *clinical* point of view, the breath may afford useful information in diagnosis; or it may present characters giving important indications for prognosis and treatment. It might be requisite in different cases to submit the expired air to a more or less systematic examination, and the following outline will serve to suggest the particulars to which attention should be directed in this examination, and to point out the practical uses which it may serve.

*a.* The breath has been made use of to distinguish between *real* and *apparent death*. For this purpose a delicate feather or a light is held before the mouth or nostrils, and it is noted whether either of these is disturbed; or a cold mirror is placed before the mouth, when, if breathing is going on, its surface will be clouded by the moisture condensed upon it. These tests are, however, not considered very reliable.

*b.* The *temperature* of the expired air may be important to notice. In some conditions it becomes exceedingly cold, and this may be readily perceptible to the hand, the breath having a chill feel, or it may be visible in consequence of the moisture in the expired air being condensed, even when the surrounding atmosphere is warm. The phenomenon is observed, for instance, in the collapse-stage of cholera. On the other hand, the temperature of the breath may be raised more or less, as in febrile diseases.

*c.* *Chemical examination* of the breath

may prove of service, and it is probable that this might afford useful information, if it were resorted to more frequently than is the custom at present. In the first place, this examination may be employed to determine the proportion of carbonic acid present. In certain affections, as during an attack of asthma, or in cases of extensive bronchitis, the amount of carbonic acid in the expired air is more or less increased; in others, such as in the collapse-stage of cholera, this constituent may be very deficient. Again, chemical examination of the breath may reveal the presence of a poison in the system, introduced from without, for example, hydrocyanic acid. It has also been employed to show the existence of deleterious products generated within the body, especially in cases of renal disease. It is affirmed that ammonia may be detected in the breath in some cases of this kind, by holding a glass rod dipped in hydrochloric acid before the mouth, the ammonia being a product of the decomposition of urea.

*d.* *Microscopical examination* of the expired air has been attempted, but at present no results of practical value have been obtained, although it has been affirmed that tubercle-bacilli have been found in it in cases of pulmonary phthisis.

*e.* The *odour* of the breath is the most important character demanding attention from a practical point of view. It is easily recognised, and the practitioner should always be on the alert to notice the smell of the breath of a patient, as this often affords material aid in diagnosis, and may even reveal certain morbid conditions which otherwise are liable to be entirely overlooked. Besides, patients not uncommonly seek advice on account of 'foulness of breath,' as a symptom for which they require special treatment. The following summary will indicate the principal circumstances under which this clinical phenomenon may prove of service in diagnosis, and in the course of the remarks it will be pointed out in what conditions the breath is particularly offensive. At the outset it must be observed that in some individuals the breath seems to have naturally a more or less disagreeable odour, which cannot be referred to any particular cause, and this amounts occasionally to extreme foulness. In females this may only be noticed at certain periods, and in some instances it seems to pass off in course of time. Again, it must be borne in mind that the breath is frequently unpleasant, either temporarily or constantly, owing to persons eating certain articles of food, or indulging in certain habits, such as excessive smoking, or chewing tobacco.

(i.) The odour of the expired air may aid in recognising poisons in the system. The smell of prussic acid or laudanum, for instance, may be revealed when either of these is present in the stomach. Alcohol, however,

chiefly demands attention in this relationship. In cases of acute alcoholic poisoning, the odour of the alcohol or of its products is at once apparent; and in persons who are found in a state of unconsciousness, the cause of which is not known, the smell of the breath is made use of as one of the diagnostic signs of drunkenness, though it must be taken with great caution. It is in the chronic forms of alcoholism that the breath gives the most valuable information. In very marked cases of chronic alcoholism it has an intensely foul odour, which is quite characteristic; but it gives extremely important indications in less confirmed cases, where the other symptoms of alcoholism are not so apparent; and especially does it enable us to detect dram-drinkers, and to explain the symptoms of which they so frequently complain. These persons, if their habits are inquired into, generally give themselves an excellent character for temperance, and seem entirely to forget that those with whom they come into contact are endowed with the sense of smell. Again, the breath may reveal the presence of certain metallic poisons in the system, of which mercury is the most important example, but lead may also affect its odour. The expired air presents the odour of ammonia in exceptional instances of uræmia, due to the exhalation of carbonate of ammonium, derived from the decomposition of urea. In cases of diabetes, and especially diabetic coma, the breath may have a sweetish, fragrant, or ethereal odour, attributed to acetone.

(ii.) The breath has a peculiar, or more or less disagreeable odour, in connexion with several diseases. That which is associated with the febrile condition is well known. In various disorders of the digestive organs the breath is often very offensive, but it is not practicable to refer any particular odour to particular diseases of either of these organs; it may, however, be affirmed that an unpleasant smell is frequently associated with habitual constipation. In cases of stercoraceous vomiting the breath may have a distinctly faecal odour. In this relation it may be mentioned that in some cases of phthisis the writer has noticed a sickly smell of the breath which is quite characteristic, and which seems to depend upon the state of the stomach. In cases of cerebral disease also, the breath often becomes exceedingly offensive, on account of the condition of the alimentary canal. Local morbid conditions about the mouth, throat, or nasal cavities constitute a most important class of affections which influence the odour of the breath; in many cases it becomes extremely foul, and may be quite peculiar in its characters. Among these conditions should be specially mentioned want of cleanliness of the mouth and teeth; decayed teeth; diseased bone in the mouth or nose; ulceration or gangrene about the mouth, especially cancrum oris, and gan-

grenous ulceration along the gums; suppuration, ulceration, or gangrene in the throat, either of local origin, or associated with syphilis, scarlatina, diphtheria, or other general diseases; ulceration of the nasal mucous membrane, and chronic ozæna; and malignant disease. The smell of the breath is of special value in drawing attention to some of these conditions, for they may exist without giving rise to any local symptoms whatever, and the patient may be quite unconscious that there is anything wrong. Several striking illustrations of this statement have come under the writer's observation. Again, certain conditions of the respiratory organs are liable to affect the odour of the expired air, and may render it unbearably fetid. Among these may be mentioned sloughing ulceration about the larynx, pulmonary gangrene in any form, and the decomposition of retained morbid products in dilated bronchial tubes or in certain cavities. Here, again, the smell of the expired air may reveal what otherwise is liable to be entirely overlooked, and especially when the patient coughs, so as to expel some of the retained air out of the lungs. Lastly, the breath may have a peculiar odour in some special diseases, such as pyæmia and its allies.

TREATMENT.—It is only intended here to offer a few remarks as to the treatment of *foulness of breath*. The first great indication is, of course, to seek out the cause of this symptom, and endeavour to remove or remedy this, by which in a large proportion of cases a cure may be readily effected. The habits should be duly regulated; the mouth and teeth properly cleansed; the alimentary canal maintained in good order; and any special affection requiring treatment attended to. When unpleasant breath depends on the stomach, it may often be improved by taking charcoal powder or biscuits, at the same time remedies being employed suitable for the particular affection present, and calculated to promote the functions of the alimentary canal, the bowels being also kept freely open. When the bad smell depends on local causes, it may be diminished by the use of antiseptic mouth-washes, gargles, sprays, or nasal douches, of Condy's fluid, carbolic acid, or creasote. Antiseptic inhalations are indicated in fœtor from the air-tubes. FREDERICK T. ROBERTS.

**BRIDES-LES-BAINS**, in Savoy.—Thermal alkaline saline waters. See MINERAL WATERS.

**BRIGHT'S DISEASE**.—SYNON.: Fr. *Maladie de Bright*; Ger. *Die Bright'sche Krankheit*.

The term *Bright's Disease* is now recognised as generic, and as including several diseases of the kidney. Many writers recognise at least three varieties,<sup>1</sup> each of

<sup>1</sup> Dr. Quain was the first to describe three forms of Bright's disease: first, the granular or atrophied

which involves chiefly one of the individual structural elements of the organ, and only secondarily, or in a minor degree, affects the others. There are thus morbid processes affecting mainly the uriniferous tubules, the fibrous stroma, and the blood-vessels, particularly those of the Malpighian tufts. That which originates in the tubules is always inflammatory in its character, although the inflammation may be *acute* or *chronic*, and may to a greater or less extent affect the stroma; that which is proper to the stroma is an extremely chronic process, supposed by many to be inflammatory, but as it appears to others, rather of an hypertrophic character; that which commences in the vessels consists in a peculiar degenerative change, the so-called waxy, lardaceous, albuminoid, or amyloid degeneration. In the following article are described:—

I. The *inflammatory affection*, affecting the tubules, or the stroma, or both.

II. The *cirrhotic* or *gouty affection*, originating in the fibrous stroma.

III. The *waxy* or *amyloid affection*, originating in the vessels.

DEFINITIONS.—I. *Inflammatory Bright's disease* is an acute or chronic affection of the kidney; caused by exposure to cold, and by scarlatinal and other blood-poisons; consisting in inflammation of the elements, passing through various stages of transformation, viz. inflammatory enlargement, fatty degeneration, and atrophy; characterised in the earlier stages by diminution of urine, albuminuria, frequently hæmaturia, tubercasts, and dropsy; in the later stages by the same symptoms, in a more or less marked degree, with secondary changes in the heart, blood-vessels, and other organs; terminating frequently in recovery in the early stage, rarely in the later, often in death by dropsy, uræmia, or intercurrent affections.

II. *Cirrhotic Bright's disease* is a chronic affection of the kidney, caused generally by the abuse of alcohol, sometimes by the poison of gout, occasionally by plumbism, and by unknown conditions; consisting in increase of the fibrous stroma, with thickening of the capsule, and ultimate atrophy of the organ; characterised by a very insidious commencement, by the absence of the early symptoms of either of the other forms, by albuminuria, at first slight or only occasionally present, afterwards more pronounced, and by the ultimate appearance of enlargement of the heart, tension of pulse, sclerosis of arteries, polyuria, albuminuric retinitis, œdema of the lungs, and uræmia; resulting ultimately in death, from uræmia, œdema of the lungs, or other intercurrent affections.

III. *Waxy Bright's disease* is a chronic affection of the kidney, caused by phthisis, syphilis, caries, suppuration, and other ex-kidney; second, the enlarged, amyloid kidney; and third, the fatty kidney.—*Lancet*, November, 1845.

hausting conditions; consisting in waxy or amyloid degeneration of the Malpighian bodies, small arteries, and sometimes the basement membrane, with, in many cases, transudation into the tubules; passing through various stages of transformation, viz. simple degeneration, enlargement from transudation, and atrophy; characterised by a large flow of albuminous urine of low specific gravity, and absence of dropsy; often attended by evidences of waxy disease of other organs, particularly the liver, spleen, and intestinal canal; resulting probably in some cases in recovery, usually in death by exhaustion, uræmia, or co-existing affections of the kidneys and other organs.

ÆTIOLOGY.—I. Of the *inflammatory form*. Cold is the commonest cause in the adult. It acts especially on those who have been exposed to its influence whilst perspiring. It frequently contributes towards the production of the disease in persons otherwise predisposed. Various blood-diseases, while they induce temporary albuminuria along with their more ordinary symptoms, have renal inflammation as a common sequela. Among these scarlatina occupies the first place, diphtheria stands next in order, followed by erysipelas, measles, pyæmia, typhus, ague, acute rheumatism, and pneumonia. Many of these maladies being most common in childhood, it follows that in the earlier years of life they are the chief causes of inflammatory Bright's disease. Pregnancy, heart-disease, gout, and malaria contribute towards its production in some cases; and the undue use of cantharides, turpentine, or alcohol may also be reckoned as causes.

II. Of the *cirrhotic form*. The commonest cause is the abuse of alcohol, particularly in the form of ardent spirits. After this, though at a long interval, rank gout and lead-poisoning. Congestion from cardiac disease is also by many authorities, but erroneously, held to be a cause (*see* KIDNEYS, Congestion of). As experience shows that the disease is often met with in people who have neither indulged in alcohol, been exposed to lead, nor suffered from gout, it is obvious that other efficient, though as yet undiscovered, causes must exist. Some authorities believe that true cirrhosis is also frequently a result of acute interstitial inflammation, and that this process is thus referable to cold, scarlatina, and other causes.

III. Of the *waxy form*. Constitutional syphilis, phthisis, prolonged suppuration, caries or necrosis of bone, and other exhausting diseases, such as cancer and chronic rheumatism, induce this degeneration. There is at present no satisfactory evidence as to the precise connexion between these influences and the morbid process.

ANATOMICAL CHARACTERS.—I. Of the *inflammatory form*. When a case of this kind is prolonged, the renal disease passes

through several conditions, which, for convenience of description, may be divided into three stages. (a) *Stage of active inflammation.* In this stage the kidney is enlarged; its capsule strips off readily; its surface appears more or less red, sometimes of a deep purple colour; and occasionally extravasations of blood are present in its substance. On section the cortical substance is found to be relatively increased in bulk. Its vessels, as well as those of the cones, are congested. The structure appears somewhat coarser than natural, while the convoluted tubules often present a swollen, opaque appearance, and occasionally contain blood. On microscopic examination the congestion of the vessels becomes very apparent, and the tubules are found to be dark and opaque, their lumen being frequently occluded. The individual epithelial cells are granular, and in a state of cloudy swelling. In some cases almost all the tubules appear affected, in others comparatively few. The enlargement of the organ is in part due to congestion, in part to exudation into the tubules. As the exudation increases the congestion becomes less marked, so that in the later period of this stage the kidney appears paler and more opaque. Unless recovery or death takes place, this condition passes into (b) *The second stage, that of fatty transformation.* In this stage the organ is still enlarged. Its capsule strips off readily; the surface often presents stellate veins, and its colour is mottled. At this time extravasations are very rarely observed, but there are alternating patches of yellowish opaque sebaceous-looking material, mingled with more natural structure. On section the cortical substance is seen to be relatively increased. There is no congestion of the vessels, and the Malpighian bodies are not prominent. The convoluted tubules are in many parts occupied by the sebum-like material, and sometimes the straight tubules present the same appearance. On microscopic examination the tubules alone are found affected. Many of them present under low powers a black appearance, due to fatty degeneration of the contents of the tubules. It is in the cells alone that this change occurs, and not, so far as the writer has seen, in the free exudation which binds the cells together. Many of the tubules are completely blocked up by this material; and sometimes in making the section of the recent kidney there is such an amount of oil set free that it permeates the whole structure of the organ, and is liable to produce the impression that the fatty degeneration is universal. This condition may be developed within a week or two of the commencement of the inflammation, and it may continue for years. During the whole course of the second stage it must be understood that inflammatory action is going on, although much less acutely than at first, and

less widely diffused. The disease is sometimes recovered from, but, more commonly, it persists; and if the patient survive long enough, it passes into (c) *The third stage, that of atrophy.* The organ is then reduced to or even below the natural size. Its capsule strips off with little difficulty, and without tearing the surface. The surface is uneven; it rarely appears coarsely granular, as in the cirrhotic form, but rather presents a series of depressions, which give it an uneven or finely granular character. Its colour is very similar to that described as occurring in the second stage, but there is less of the sebaceous-looking material. On section the cortical substance is found relatively diminished. The Malpighian bodies are not prominent. The tissue feels more dense, and many of the tubules are occupied by sebaceous-looking material. The blood-vessels, and particularly the small arteries, are sclerosed: the intima and the adventitia are frequently, the middle coat almost invariably, thickened. The fibrous stroma is relatively increased, especially towards the surface of the organ, and the tubules may be traced in different stages of atrophy. On close inspection that atrophy is found to result not so much from shrinking of the fibrous stroma as from molecular absorption of the contents of the tubules.

Besides the typical form of the inflammatory affection just described, a very common variety is the form of post-scarlatinal inflammation first accurately described by Klebs, to which he has given the name *glomerulo-nephritis*, because the glomeruli are especially affected. In this condition an accumulation of round cells (leucocytes) takes place in the glomeruli and neighbouring connective tissue, while blood is frequently extravasated within the glomeruli between the tufts of vessels and the capsule, as well as into the tubules. In the tubules also more or less inflammatory action manifests itself in the epithelium, and the corresponding structures of the Malpighian bodies suffer in a greater degree. At times the accumulation and swelling of these structures is sufficient to compress the vascular tuft. This disease also may be described as passing through three stages—in the first of which there is some enlargement with minute extravasations and the microscopic changes above described; in the second, where a fatty transformation has occurred in the epithelium and the fibrous tissues show thickening; in the third there is more or less atrophy, with shrinking of tubules or morbid increase of the fibrous stroma.

The anatomical changes met with in other organs are described along with the complications.

II. *Of the cirrhotic form.* The course of this affection is very chronic. It consists essentially in an increased growth of the

fibrous stroma, with secondary changes in the tubules and vessels. In an early stage the organ may be found of fully the natural size. The capsule strips off less readily than in health. The surface is somewhat uneven, and may present cysts. On section the cortical substance is relatively enlarged, and this is due merely to an increase of the stroma, not to any change in the vessels or in the tubules. But it is in the more advanced stages that the disease is commonly seen. Then the organ is reduced in bulk, it may be slightly, it may be to one-fourth of its normal size. Its capsule cannot be peeled off without tearing the gland. The surface is uneven and granular, and often of a reddish colour. On section the cortical substance is found relatively diminished, its structure being dense and fibrous. The small arteries are thickened and very prominent, all their coats, but especially the middle, being increased in volume. Many of the tubules are atrophied, but the epithelium of such as are not involved is for the most part natural. Cysts are numerous, and are found in connexion with the tubules, the Malpighian bodies, and the cells.

There are two points worthy of being specially kept in view by those who desire to attain to clear conceptions of Bright's disease—viz. (1) That its different forms are very frequently combined—in particular that the inflammatory affection is found associated sometimes with the waxy disease, sometimes with the cirrhotic; but that the descriptions here given are derived from pure examples of each process; and (2) that atrophy results in all the forms if the disease lasts long enough; that is, that a small, uneven-surfaced kidney may result from either the inflammatory disease of the tubules, or the waxy disease of the vessels, as well as from the increased growth and subsequent contraction of the fibrous stroma in the cirrhotic form.

III. Of the *waxy* or *amyloid* form. This chronic morbid process may also, for convenience of description, be divided into three stages. Of these the first is (a) *The stage of degeneration proper*, in which the organ presents an almost normal appearance. The size is natural; the capsule strips off readily; and the colour is not altered. On section it appears normal, excepting that the Malpighian tufts, without being congested, are prominent, and in certain lights may be seen, even by the naked eye, to present the dim translucency characteristic of waxy degeneration. On applying a little aqueous solution of iodine, the Malpighian tufts and the small arteries assume a peculiar mahogany hue. On examination with the microscope, the stroma and tubules are found to be healthy, the vascular structures being alone affected. The affection is often first seen and is most distinct in the middle coat of the arteries, the swollen transverse fibres taking on the

colour, and producing what has been described as the ipecacuanha-root appearance. How long this condition may last without the tubules becoming affected it is impossible at present to say, for it has only been met with in patients who had died of one or other of the causal complications while the renal malady was still in an early stage. Sooner or later it passes, however, into the condition most commonly met with—(b) *the second stage*, that of *degeneration with secondary changes in the tubules*. In this condition the organ is enlarged. Its capsule strips off readily, the surface is smooth and pale, presenting, in pure examples, little or no mottling. On section the cortical substance appears relatively increased, and looks much paler than the cones. The structure usually is denser than natural. The vessels appear prominent, and the Malpighian tufts resemble minute grains of boiled sago. On the addition of iodine the degenerated parts become characteristically coloured, and stand out prominently from the tubular tissue, which does not exhibit the characteristic reaction. On microscopic examination the vessels are found altered as in the earlier stage, but the change is more advanced. The stroma is normal, but many of the tubules are altered. Some are blocked up by a dimly translucent, wax-like material, which however does not assume the mahogany colour on the addition of iodine. The epithelium in many of the tubules presents a finely granular appearance, and occasionally is somewhat fatty, but the epithelium and the basement membrane very rarely present the characteristic reaction. This change in the tubules is thus secondary to the degeneration proper, which is confined to the vessels; and these secondary changes consist in some alteration of the nutrition of the epithelium, with exudation or transudation of coagulable material into the lumen of the tubes. This condition may last for years, but should the patient live long enough it passes into (c) *the third stage*, that of *atrophy*. The organ is then below the normal size; the capsule strips off readily; the surface presents an uneven granular appearance, and is pale. On section the cortical substance is found relatively diminished. Its small arteries are prominent and thickened; its Malpighian bodies are very conspicuous, and are grouped together in consequence of the atrophy of the intervening structure. The stroma is relatively increased, and many of the tubules are destroyed, while of those which remain not a few present the characters above described as being met with in the second stage. The organ may, in extreme cases, be diminished to less than half its natural size.

**SYMPTOMS.**—1. *Of the inflammatory form.* The leading clinical features of this variety, in addition to the albuminuria which exists in all the forms of Bright's disease, are dimi-

nution in the quantity of urine, and the presence of dropsy.

The onset of the disease may be sudden or gradual. Sometimes it is the diminution and alteration of the urine that attracts attention, sometimes the dropsy, sometimes the gastric derangement and general uneasiness; but whichever symptom may appear first, the others usually speedily follow. The urine is generally diminished in quantity, often somewhat opaque, and smoky or even bloody. It contains much albumin and numerous tube-casts. The casts are granular, being composed mostly of epithelium in a state of cloudy swelling; sometimes bloody; frequently hyaline or fibrinous. The urea is diminished, it may be, to one-half, one-fourth, or even a less proportion of the natural amount. This leads to a corresponding reduction of the specific gravity, unless there be a compensatory diminution of water, or increase of albumin. The dropsy is generally distinct in the face, and swelling of the eyelids is well-marked in the morning. Towards evening, if the patient be out of bed, the legs are chiefly affected. The scrotum and penis are often swollen, and sometimes the abdomen is also dropsical. There is little quickening of pulse or elevation of temperature, but a good deal of general uneasiness is experienced, with debility and pain in the loins; and dyspeptic symptoms are often present, due to gastric catarrh. Such is the usual condition at the commencement of the disease, and during the period which has been already described as the first stage. But sometimes at this stage a much more serious condition is developed—namely, suppression of urine, followed by coma or convulsions leading to a fatal result; or dropsy may increase to such an extent as of itself to cause death. Or, again, without or even with the most unfavourable symptoms, under appropriate treatment the kidneys may begin to act more freely, the urine increasing in amount and improving in characters, with consequent gradual disappearance of the dropsy, and restoration of health. Or, as often happens, the general condition improves, but a chronic albuminuria remains, and the disease passes into the second stage. The urine is then no longer bloody; the quantity is greater, though still below the normal; the specific gravity is low; there is albumin, along with tube-casts, fatty, hyaline, or mixed, partly fatty and partly hyaline. The urea is below the normal standard. The dropsy may continue, and may even gradually increase, or it may pass off and only appear when the patient is fatigued, or when he has caught cold. The general symptoms remain unchanged, except that anæmia comes on, and the patient's debility steadily increases. This course of events may pass on to a fatal result; or there may be complete, or, as is more common, merely partial recovery. In this condition

the patient may linger for many months, occasionally suffering exacerbations, and he may succumb to one of them, or to one of the numerous complications to be presently described. If the patient passes into the third stage, he appears prematurely old. His urine is of natural amount or even somewhat increased in quantity, but of low specific gravity. It contains albumin and a few casts, mostly hyaline, with scattered fatty cells imbedded in them. The urea is still diminished. There is dropsy of the feet and ankles in the evenings, and slight exposure brings on more general attacks. The face is pale and pasty, and the eyelids are often œdematous. The pulse becomes hard and tense; the arteries gradually become thickened from sclerosis and atheroma; while the apex-beat of the heart passes downwards and to the left side, owing to hypertrophy, particularly of the left ventricle. In this condition a fatal result may be induced by an acute exacerbation with general dropsy; by chronic, or, more rarely, acute uræmia; or by intercurrent attacks of inflammatory or other affections of various organs.

Glomerulo-nephritis is perhaps attended with a greater tendency to hæmaturia, and less tendency to dropsy, than other forms of acute nephritis, but the clinical features are not as yet capable of being definitely marked off from those of other maladies. Dr. George Johnson has drawn attention to a peculiar kind of cast, containing numerous leucocytes which characterise this particular lesion.

2. *Of the cirrhotic disease.* The onset of this affection is extremely insidious, and it may exist for a long time without distinctly manifesting itself by symptoms. Its existence may be suspected when a patient suffers from renal inadequacy in respect that the urea-discharge falls below the normal standard, and albumin occasionally appears in the urine, but one is not entitled to establish a diagnosis upon such grounds alone. The condition is often discovered only when dyspepsia, uræmic convulsions, or blindness from retinitis leads the patient to consult a medical man. The earliest symptoms are occasional slight albuminuria, and frequent calls to micturition during the night, the urine however not being excessive, its specific gravity being low, and the urea somewhat diminished. But when the disease has existed for some time the complexion becomes altered; the eye assumes a peculiar appearance, from œdema of the conjunctiva; the patient is subject to dyspeptic attacks; the heart becomes hypertrophied, and the vessels sclerosed and degenerated; while there is little or no dropsy. When the disease is advanced, these changes in the circulatory organs are well-marked, and the cachectic condition becomes distinct. The occurrence of various complications, such as severe gastric catarrh, diarrhœa, anæmia,

dyspnœa, bronchitis, œdema of the lungs, headache, uræmia, and the characteristic retinal affection, render the diagnosis easy. Frequently towards the end there is an increased flow of urine, of low specific gravity. This is in some cases a very prominent symptom. The disease is never recovered from, and the fatal result occurs from uræmia; from some inflammatory complication, such as pleurisy, pericarditis, bronchitis, or pneumonia; or from some result of degenerative change, as hæmorrhage from a mucous surface or into the brain.

3. *Of the waxy disease.* The onset of this affection is gradual and insidious. A patient who has suffered from phthisis, syphilis, or other wasting malady passes an excessive quantity of urine, and finds himself obliged to rise several times during the night for micturition. The urine is pale, of low specific gravity, containing at first no albumin, subsequently only a trace, ultimately a considerable amount. The urea is little, if at all, diminished; the tube-casts are extremely few, and mostly hyaline. There is no dropsy, but evidence of concomitant waxy affections of other organs is frequently afforded. The liver is enlarged, its margin being easily felt and sharply-defined. The spleen is also increased in size. The blood is slightly altered, the white corpuscles being somewhat increased, and the red being rather flabby and ill-defined. These conditions gradually become more distinct, and the strength of the patient diminishes, partly from the disease of the kidneys and other organs, partly from the wasting diseases which have induced the degenerative changes. A case of the kind has been known to go on for nearly ten years, during which time the urine continued of the characters just described, and dropsy never appeared. At length the vital powers of the patient became depressed, head symptoms gradually supervened, and death ensued. It is not often that such an uncomplicated case is met with. More commonly the exhausting disease which led to the degeneration causes death before the waxy change has gone so far. Sometimes also intercurrent complications induce the fatal result. Clinical observation renders it probable that the kidneys, as well as the liver and spleen, may recover from their degeneration, in cases in which the causal malady has been got rid of.

**COMPLICATIONS.**—(a) **Complications connected with the Alimentary System.**

*Gastric affections* are met with in all the forms of Bright's disease. Catarrh of the stomach—acute, sub-acute, and chronic—is common to them all, and is characterised by an unusual tendency to nausea and vomiting. It is especially frequent during the first stage and in acute exacerbations of the inflammatory form, and is often a chief source of suffering during the most advanced

stages. It is not uncommon during the whole course of the waxy form, but is most usually met with in the cirrhotic variety. So close indeed is the relationship between them, that in the management of cases of cirrhosis regard should constantly be had to the state of the stomach, and in no case of chronic gastric catarrh should the physician neglect to inquire into the state of the urine. This affection, when complicating the early stage of the inflammatory form, often owes its origin to the same cause as the kidney-affection itself. When complicating the later stages of the inflammatory, and any of the stages of the cirrhotic disease, the catarrh is probably a result of efforts at elimination of materials retained in the blood by the failure of the action of the kidneys. When occurring in the waxy form, it is frequently due in part to the existence of waxy degeneration of the vessels of the gastric mucous membrane. In the waxy disease we sometimes find blood mingled with the vomited matters, just as we find hæmorrhage in other organs when this degeneration exists.

*Catarrh of the intestine* also occasionally occurs, sometimes producing an exhausting diarrhœa, especially in advanced inflammatory and cirrhotic cases; but it is along with the waxy disease that intestinal symptoms are most common. These are due to waxy degeneration, and consequent ulceration; or to ordinary tubercular disease of the intestine. Both of these affections induce diarrhœa, but there is evidence that not only may it thus occur, but that blood may also be discharged, although there be no ulceration recognisable by the naked eye.

*Hepatic affections.*—Functional derangements of the liver occur in the course of all the forms of Bright's disease. The chief organic changes are fatty degeneration, waxy degeneration, cirrhosis, and syphilitic affections. The first-named is not specially related to any of the forms. The waxy degeneration and the syphilitic affections are of course commonly met with as accompaniments of the waxy disease; whilst cirrhosis is occasionally associated with the cirrhotic kidney, and sometimes with the other forms.

*Ascites* is often seen as a manifestation of general dropsy in the inflammatory form of Bright's disease; and sometimes this is a prominent symptom in mixed forms, when waxy disease of the liver is associated with a waxy and slightly inflammatory condition of the kidneys.

*Peritonitis*, like inflammation of the other serous membranes—one of the most important classes of the complications of Bright's disease, is occasionally the cause of death in all the three forms. It may result from local affections, or from the state of the blood; and may be acute, severe, and therefore obvious, or so insidious as scarcely to attract attention.

### (3) Complications connected with the Blood, or with the Lymphatic- and Blood-glands.

The *spleen* is usually unaffected in cases of inflammatory Bright's disease, except such as prove fatal in the earliest stage, and in which the spleen is affected in common with the kidney. In the waxy and cirrhotic forms corresponding lesions are frequent in this organ.

The *lymphatic glands* are rarely altered excepting in the waxy form, in which they are sometimes the seat of the waxy degeneration, sometimes of tubercular disease, or of strumous inflammation.

The *blood* itself is altered in its chemical composition. In the inflammatory form its density is diminished, the corpuscles and albumen being deficient, while the water is correspondingly increased. The quantity of urea is above the normal. In long-standing cases of waxy disease similar changes are found; and not infrequently there is a slight numerical increase of the white corpuscles, and flabbiness of the red blood-discs when the spleen is affected. In the cirrhotic form like alterations also occur.

*Hæmorrhage* is apt to occur in advanced stages, especially of the cirrhotic form. It may take place from the kidneys, or from the mucous membranes, particularly that of the nostrils. In the inflammatory affection hæmaturia is common in the early stage; in the waxy variety this symptom occasionally occurs, but rarely to a serious extent.

### (4) Complications affecting the Circulatory System.

*Hypertrophy of the heart* is almost always present in cases of advanced cirrhotic disease, and also in the advanced stages of the inflammatory affection. One may trace in patients the gradual development of this hypertrophy, advancing *pari passu* with the progress of the renal affection. It is comparatively rare in the waxy form. *Hydropericardium* is met with in some cases, as a manifestation of general dropsy. *Pericarditis* occurs as an intercurrent affection in all the forms, but especially the inflammatory and the cirrhotic. It is apt to be overlooked, owing to the absence of local pain, or from the pain being referred to the abdomen. *Endocarditis* is also frequently associated with the various forms of Bright's disease.

The *arteries* are sclerosed and atheromatous in the advanced stages of the inflammatory and in the cirrhotic, and to a less extent in the corresponding stage of the waxy disease. In that affection the small vessels in other parts are frequently the seat of waxy degeneration. Thickening of the arteries occurs constantly in the more advanced stages of the inflammatory and cirrhotic diseases, and is due in great part to hypertrophy of their middle coat, in lesser degree to sclerosis of the tunica intima, the tunica adventitia, and perhaps the perivascular lymphatic sheath.

The *pulse* becomes tense and sustained in chronic cases, partly from the hypertrophy of the heart, partly from the changes in the capillaries and smaller arteries.

### (5) Complications connected with the Respiratory System.

*Acute bronchitis* is common, especially in the advanced stages of Bright's disease, and tends to pass into the chronic state. Bronchitis may originate also as a sub-acute or chronic affection. *Edema of the lungs* is very common in advanced stages, and frequently occurs as a manifestation of general dropsy in the early, as well as in the later, stages of Bright's disease. It may be very suddenly developed in cirrhotic cases, and may rapidly prove fatal. *Pneumonia* occurs sometimes as a cause of inflammatory Bright's disease, sometimes as a consequence of exposure to cold during the course of chronic cases. *Phthisis* in its various forms is found causally associated with these renal affections—frequently with the waxy, more rarely with the inflammatory form. It usually proves fatal while the renal malady is yet in its early stage. *Hydrothorax*, acute or chronic, is often seen in dropsical cases. *Pleurisy* occasionally occurs with all the forms of Bright's disease, and may be due to the state of the blood; or, as seems more likely, to increased susceptibility to inflammatory changes, which results from the lowered vitality of the organism. *Dyspnœa* is frequently met with in the inflammatory and cirrhotic forms of the disease, and may be independent of any local lesion, being probably a result of uræmic poisoning. *Edema glottidis* is apt to occur in inflammatory cases, when even a slight laryngitis has from any cause been brought on.

### (6) Complications affecting the Skin and Subcutaneous Tissues.

*Dropsy*, in the form of anasarca, is almost constantly present in the early stage, and during exacerbations of the inflammatory form. It can scarcely be said to occur in uncomplicated waxy and cirrhotic cases. *Eczema* is occasionally troublesome in chronic cases. *Erysipelas* is met with now and then, always constituting a serious addition to the other malady.

### (7) Complications affecting the Urinary Organs.

The chief of these is serofulous disease of the kidney, and more rarely of the bladder and prostate. They occasionally occur along with the waxy affection.

### (8) Complications affecting the Reproductive System.

By far the most important complication is pregnancy, which may coexist with any of the forms of renal disease. Most frequently it is the inflammatory variety that causes danger, and in it dropsy is a prominent symptom, and uræmic convulsions and coma may at any time occur. But in many cases of

this kind the term of utero-gestation is completed without the occurrence of uræmia, and often when uræmia does occur the patient comes safely through the ordeal, the delivery being effected, and the nervous symptoms passing off. Cirrhosis may coexist with pregnancy, and a woman may have successive satisfactory confinements during the continuance of this renal disease. It is rarer to find pregnancy in women suffering from waxy disease, but it may occasionally occur.

Sometimes also there may be strumous disease of the uterus, complicating waxy degeneration of the kidney. Strumous or syphilitic disease of the testicle may be similarly associated.

#### (8) Complications affecting the Nervous System and Special Senses.

*Uræmic blindness* may occur, which is sudden and usually temporary, being unaccompanied by any lesion recognisable by the ophthalmoscope. It is generally met with in advanced cirrhotic and inflammatory cases. *Retinitis albuminurica* is a peculiar and characteristic inflammation of the connective tissue of the retina, leading to the formation of white patches and lines, with fatty degeneration. With it are also frequently associated minute hæmorrhages into the substance of the retina. This occurs by far the most frequently in cirrhosis. It is often also seen in the advanced stages of the inflammatory form, and is rarely recovered from except in the case of pregnant women, in whom it seems apt to occur as a passing condition.

*Uræmia* includes a group of the most striking symptoms of Bright's disease. It may occur at the commencement of the acute inflammatory affection, or in its later stages, or in the chronic forms. The condition is, however, rare in the purely waxy disease, but common in the cirrhotic. There are several types of uræmia, of which the most important are:—(a) Sudden acute convulsions, followed by coma and death; (b) Gradually advancing torpor, passing at last into coma. The clinical features of these and minor varieties are described, and the hypotheses as to their origin discussed, in the article URÆMIA.

*Headache* is frequently complained of by patients suffering from Bright's disease. *Apoplexy* from hæmorrhage into the substance of the brain is common in the later stages of the inflammatory and cirrhotic diseases. It is due partly to the degenerated state of the vessels, and partly to the increased pressure resulting from cardiac hypertrophy.

#### (c) Complications affecting the Locomotory System.

Of these the only ones of importance are *diseases of bones and joints*, which may occur as causal or concomitant complications in waxy cases; and *gouty affections*, which have been already mentioned in connexion with the cirrhotic disease.

**DIAGNOSIS.**—(a) **Of Bright's Disease from other affections.**—From *functional albuminuria* the organic diseases of the kidney are to be distinguished by comparison of the clinical history of the respective conditions. The writer has sought to distinguish four forms of functional albuminuria:—I. *Paroxysmal albuminuria*, a condition often associated with paroxysmal hæmoglobinuria, the discharge of albumen sometimes preceding, sometimes following, and sometimes replacing the hæmoglobin discharge. The attack comes on occasionally, is marked by very distinct albuminuria, and by the presence of numerous tube-casts, and often oxalates, but the symptoms very rapidly again disappear, and are not attended by dropsy. II. *Dietetic albuminuria*, in which the albumen appears only after the ingestion of food, of some particular meal or some special article of diet. III. *Albuminuria from exertion*, in which the discharge of albumen follows muscular effort of a more or less severe kind. In these two varieties (both of which stand related to Pavy's 'cyclical albuminuria' and the 'albuminuria of adolescence') a comparison of the secretion passed at different times of the day and night, enables one to mark the functional off from the organic diseases, for at one time there is no albumen, or almost none; at another it is abundant; and one soon learns to make out in the functional cases the precise conditions which induce its appearance. Other urinary conditions which characterise the functional cases are the normal discharge of urea, the absence of casts, and the occasional occurrence of phosphaturia, of oxalates, of urates or uric acid, of sugar or other chemical abnormalities. Much weight is also to be attached to the absence of the characteristic general features of Bright's disease. IV. *Simple persistent albuminuria*, in which patients continue to discharge albumen with the urine for long periods without giving evidence of organic disease. This is much more difficult to distinguish. The diagnosis depends mainly upon evidence excluding Bright's disease, such as the complete, or almost complete, absence of tube-casts, the discharge of a normal or even excessive proportion of urea, the absence of the complications of Bright's disease, particularly of the various causal, concomitant, and consequent complications which are so generally associated with it. Thus, for example, if a patient has albuminuria for months, but secretes abundance of urea, does not become anæmic, has no increased tension of pulse nor hypertrophy of heart, no tendency to dropsy, or to retinal changes, or any other of the well-known complications, we may conclude that he is suffering from functional disease. But diagnosis and prognosis must always be cautiously expressed in cases of this kind. From *passive congestion of the*

*kidneys* due to cardiac disease these maladies are distinguished by the general condition of the patient; the absence of cardiac disease, and of congestion in other organs; and the characters of the urine. In heart-affections the urine is generally scanty, high-coloured, not of low specific gravity. It may contain albumin, and deposits urates, but rarely blood, renal epithelium, or tube-casts. Hyaline casts may be present, but never in any large quantity. The presence of epithelial and fatty casts, or marked diminution of the amount of urea in any case, proves at least the co-existence of actual inflammation of the kidney. From *paroxysmal hæmoglobinuria*, Bright's disease is distinguished by the abrupt commencement and brief duration of this malady; by the marked nervous symptoms, with gastric catarrh, and sometimes slight jaundice; and by the absence of dropsy. In hæmoglobinuria also the condition of the urine is very distinctive; the dark-red colour being due, not to blood-corpuscles, but to granular pigment, the deposit consisting mostly of this material and of tube-casts. *Hæmaturia*, with tendency to suppression of urine, is distinguished from Bright's disease by the small proportion of epithelial tube-casts, and in some cases by the complete absence of casts. There may be a question whether the case is one of hæmaturia or of commencing acute inflammatory Bright's disease; or, again, whether it is one of a chronic affection, cirrhotic or cystic, in which hæmorrhage has come on. The cases in which Bright's disease simulates hæmaturia are generally the sequelæ of scarlatina or diphtheria; and, therefore, even when these diseases have been overlooked, the presence of desquamation or of paralysis may afford a clue; but the peculiar reddish-brown deposit, rich in cells and in epithelial tube-casts, which occurs in Bright's disease, makes the case clearer even when, as often happens, there is no dropsy. Again, when the question is between simple hæmaturia and hæmaturia with cirrhosis or cystic disease, the evidence afforded by the tube-casts is not important, but the hypertrophy of the heart, the thickening of the arteries, the character of the pulse, the albuminuric retinitis, the low specific gravity of the urine and the small amount of urea which it contains, as well as the tendency to hæmorrhage from other sources, afford evidence of the presence of the chronic organic disease.

Slight *pyelitis*, with or without renal calculus or gravel, may simulate Bright's disease; but the history of pain, the presence of mucus and pus-corpuscles in the urine, of oxalate of lime or uric acid, with the full proportion of urea, and the absence of tube-casts, or their presence only in small quantities, indicate the nature of the case.

(3) **Of the different forms of Bright's disease from each other.**—The discrimi-

nation presents in simple cases little or no difficulty. The points to be attended to are the history of the patient; the amount and characters of the urine; the presence or absence of dropsy; and the nature of the complications. The previous occurrence of exanthematic affections, of chronic wasting disease, or of intemperance, gout, or plumbism, would afford some obvious indications. The mode of origin and progress of the malady is very important. Thus a case commencing acutely with dropsy and diminution of urine is inflammatory; one of less acute character, with polyuria, is waxy; and one commencing insidiously, with no marked symptom until perhaps convulsions or dimness of vision appeared, would be an example of cirrhosis. The leading symptoms of the *inflammatory* variety are diminution of urine; an abundance of albumin and of epithelial tube-casts, with diminution of urea; and marked dropsy. Other indications may be gathered from the complications of each form of Bright's disease. It must be remembered that mixed forms frequently occur, and that in these careful inquiry and patient investigation are essential to the establishing of a correct diagnosis. Of the *waxy* kidney, the prominent features are early and persistent polyuria; waxy degeneration of other organs; and absence of dropsy. In the *cirrhotic* form the insidious commencement; the gradual development of vascular and cardiac changes; with in the later stages, in many cases, polyuria, are the most important phenomena.

**PROGNOSIS.**—The prognosis, though always grave, varies in the different forms of Bright's disease. In the *inflammatory* affection it is least unfavourable, although this affection is the most immediately dangerous. During its first stage we may always hope for complete recovery, especially in cases of post-scarlatinal origin. Of forty-one successive cases treated by the writer in the Royal Infirmary, Edinburgh, twenty-two recovered entirely, while twelve died, and seven passed into the second stage. If this be the proportion in hospital cases, which are generally sent there on account of their severity, and are rarely sent in the earliest stages of the disease, it is obvious that the proportion of recoveries must be much larger in private practice. When the disease reaches the second stage, the prognosis is more grave, complete recovery being rare, and death sometimes taking place from sudden or gradual increase of the symptoms, or from intercurrent affections. But even in this condition complete recovery may be brought about, and in many cases the patient goes on for long periods, presenting few symptoms fitted to attract attention. In the third stage the prognosis is entirely unfavourable, the system becoming steadily more deteriorated, and death occurring, either from the direct

effects of the disease, or from complications. Still, even such cases often go for long periods, if placed under favourable hygienic and therapeutic conditions.

In the *cirrhotic* form the prognosis is very unfavourable, but the progress of the disease is so slow that it is often inadvisable to say anything about it to the patient, as the fatal result may be long deferred. It must, however, be kept in view that the disease is commonly far advanced before its existence is made out.

In the *waxy* form the prognosis must almost always be unfavourable, although the malady is never rapidly fatal. On the contrary, its course is always chronic, in some cases extending over five or even ten years. The fatal result is due to complications more frequently than to the disease itself. Recovery probably sometimes takes place, but only when the cause of the degeneration is removed, and the general surroundings of the patient are favourable. It is certain that the liver and spleen may to a large extent recover from waxy disease, and recovery has been witnessed in cases which presented all the symptoms pointing to implication of the kidneys.

Among the symptoms and complications which are fitted to cause special alarm when they occur in connexion with any of the forms of Bright's disease, we must recognise suppression or great diminution of urine, especially if accompanied by nervous phenomena or general dropsy; uræmia, more particularly its chronic form; and acute inflammations and hemorrhages. Retinitis albuminurica is always a very serious symptom, except when it occurs in pregnant women.

**TREATMENT.**—(1) Of the *inflammatory form*. The objects to be kept in view are to arrest the inflammatory action; to remove the inflammatory products from the kidneys; to supply the deficiency induced by the drain upon the system; and to obviate the deleterious effects upon the system generally of the accumulation of effete materials. One remedy or plan of treatment may meet more than one of these indications. The most useful means of subduing the inflammatory action, or at least the congestion which attends it, are local blood-letting by means of leeches or wet cups; dry-cupping; and the application of hot fomentations, poultices, and counter-irritants. Blood-letting is only serviceable in the early stage of the disease, or when severe exacerbations with suppression of urine occur. Poultices or hot fomentations are of use in the same circumstances. Counter-irritation is helpful in the more chronic conditions. Iodine and croton oil inunction are the best fitted for its induction, while cantharides must be avoided on account of its tendency to irritate the kidneys.

The removal of the inflammatory products which block up the uriniferous tubules is of

the utmost importance, and is in the great majority of cases best effected by means of diuretics. Water and diluent drinks are the safest, and are sometimes found sufficient. The medicinal diuretics must be non-irritating, and the best of all is digitalis, which may be given safely even when the urine is bloody. It may be administered in the form of infusion, tincture, or made up into a pill. Of the infusion from a drachm to an ounce, of the tincture from five to thirty minims, of the powder from half-a-grain to two grains, should be given three times a day. The infusion or the tincture may be combined with spirit of nitrous ether, with acetate of potassium, or with tincture of perchloride of iron. Its action is often favoured by the addition of squill and carbonate of ammonium. Other diuretics, such as caffeine or strophanthus, occasionally act better than digitalis. Sometimes it happens that diuretics do not suit the case, the urine becoming diminished and more bloody under their use; and in other cases the symptoms become so urgent that death might take place before there would be time for diuretics to act. In either of these conditions relief must be obtained by the bowels or skin. The bowels are best acted upon by means of from twenty grains to a drachm of the compound jalap powder, or one-twentieth to half-a-grain of elaterium. The action of the skin may be excited by the use of acetate of ammonia or antimony; but pilocarpine, hot air, vapour-baths, and the wet pack are the most efficient agents. Throughout the whole course of the disease constipation should be avoided, and the action of the skin encouraged. When the disease has become less acute, and certainly when dropsy persists during the second stage, other diuretics are of the utmost value, particularly the acid tartrate of potassium, the oil of juniper, and the decoction of broom. Iron must be assiduously administered, to make up for the waste of the materials of the blood. Gallic acid, ergot, hydrochlorate of rosanilin, and belladonna have all been praised as tending to diminish the discharge of albumin resulting from a persistent chronic inflammation of the tubules. The treatment of special symptoms and complications will be considered after indicating the general management of the other varieties of Bright's disease.

The diet during the earliest stages should be easily assimilable, and not too rich in nitrogenous elements. Milk is, as a rule, well borne. Some practitioners laud skimmed milk as an unfailing remedy in the disease. It is a good diuretic, and, when it suits the stomach, a good article of diet, but possesses no other therapeutical virtue. In the more chronic stages the food should be of a more nourishing kind, and a moderate allowance of stimulants may be needed.

(2) In the *cirrhotic form* it is probable that no remedy we at present possess can

influence the pathological process, although arsenic and alkaline remedies, and particularly iodide of potassium, enjoy a certain reputation. It is the duty of the skilful physician to ward off or minimise the results of the morbid process. It is obviously of great importance to avoid the causes of the disease. Lead-poisoning should be avoided; the gouty tendency kept in check; and the free use of alcohol forbidden.

(3) In the treatment of the *waxy form*, the most important indication is to seek to remove the cause of the degeneration, if still existing. If there be disease of bone or chronic abscess it must, if possible, be cured; constitutional syphilis must be combated by appropriate remedies. The tincture of perchloride of iron, quinine, nux vomica, and combinations of these in the form of phosphates, are useful. The patient must also have good food, and should lead an easy life.

(4) In the management of the *combined forms* of Bright's disease these plans of treatment must be conjoined according to circumstances, but, on the whole, treatment is much less successful than in the simple cases. In the combined waxy and inflammatory affection, for instance, it is not uncommon for dropsy to persist, although the diuretics bring the urine up to or above the natural standard.

(5) With regard to the special *symptoms and complications* of Bright's disease, the gastric catarrh is often benefited by the use of bismuth, soda, and rhubarb; the sickness and vomiting are best relieved by counter-irritation over the stomach, and by giving ice, milk, and hydrocyanic acid internally. These symptoms are, however, often very intractable. Diarrhœa must sometimes be let alone; at other times it must be treated by means of astringents or sedatives, either administered by the mouth or as enema or suppository. Ascites must be treated as a manifestation of dropsy, and occasionally the abdomen requires tapping. Peritonitis must be combated by hot fomentations and opium, but the latter requires great care in its administration. All through the disease in all its forms hæmatic tonics are demanded; iron in some form should be constantly administered. For hæmorrhages the pernitrate of iron, local astringents, ergot and ergotin, gallic acid, or acetate of lead must be tried in various combinations. The best results have followed the use of ergotin in 3- to 5-grain doses injected subcutaneously. The irritating effects sometimes observed after the subcutaneous injection of ergotin may often be obviated by boiling the solution, or by the addition of a minute quantity of salicylic acid. Hydropericardium and pericarditis must be treated in the usual way. In the latter the writer has repeatedly seen much benefit follow the application of leeches, and during the past few years has witnessed a

number of recoveries from this complaint. The vascular and cardiac symptoms are often relieved by the use of the nitrites, and particularly of nitroglycerine cautiously administered. Bronchial catarrh must be carefully attended to, by the avoidance of exposure to cold, by the application of counter-irritation externally, and by the internal administration of expectorants. Œdema of the lungs must be treated by counter-irritants, and by remedies fitted to reduce the general dropsy. Pneumonia, phthisis, and pleurisy must be treated on ordinary principles. Hydrothorax may demand paracentesis. General dropsy is one of the most important complications, and should be combated by means of diuretics, purgatives, and diaphoretics; in many cases the introduction of Southey's tubes or puncture of the œdematous parts is demanded. When these methods have been determined upon, precautions must be taken to avoid inflammation. Eczema and erysipelas, when they occur, should be dealt with according to ordinary principles. Headache is relieved in different cases by iron, by hot or cold applications to the head, by quinine, or by inhalation of a few drops of nitrite of amyl. When uræmia occurs in acute inflammatory conditions, or with suppression of urine, dry-cupping or wet-cupping over the renal regions should be tried, along with free purgation and hot-air baths and, especially in puerperal cases, general blood-letting. But, above all, reliance must be placed in the use of pilocarpine. Generally it is best to inject it subcutaneously, and thereby produce diaphoresis. Bromide of potassium should be given in drachm doses, and if convulsions be severe, the patient must be kept under the influence of chloroform. In the more chronic and gradually advancing form of uræmia, counter-irritation at the back of the neck and over the scalp sometimes appears to be useful. But treatment is not so often of advantage in this as in the other form. In the eye-affections, iodide of potassium enjoys some reputation. Hæmorrhagic apoplexy demands no special discussion in this article.

T. GRAINGER STEWART.

**BRINKENAU**, in Bavaria.—Chalybeate waters. See MINERAL WATERS.

**BROMIDROSIS** (*βρῶμος*, a stench; and *ἰδρῶς*, sweat).—A term for fetid perspiration. See PERSPIRATION, Disorders of.

**BROMISM**.—DEFINITION.—Bromism is the term applied to the morbid effects produced by the administration of the salts of bromine under certain circumstances.

DESCRIPTION.—The effect of the salts of bromine, when administered in medicinal doses, is to reduce nervous activity; and thus, with a certain amount of anæsthetic influence, to promote rest and sleep. When such doses have been long continued, or in

certain idiosyncrasies, or when excessive doses are administered, results are produced which constitute a state of disease, and to this condition the term 'bromism' is applied. These results are manifested on the brain and spinal cord; on the skin; on the mucous membranes and glandular structures; and on the organs of circulation and respiration.

1. *On the Brain and Spinal Cord.*—When the therapeutic action intended to be obtained from the use of a salt of bromine is exceeded, the quiet or sleep becomes more pronounced, and there is more or less constant somnolence; the memory becomes impaired, words being forgotten or misplaced, whilst written and spoken language is confused, the tongue is tremulous, and speech is difficult. The gait becomes feeble and staggering, with inability to control movement, and somewhat resembles the condition observed in locomotor ataxy. The special senses—sight, hearing, taste, and touch—are impaired. Reflex excitability is diminished. This impairment is especially observed in the fauces, where touching does not as usual cause sickness; indeed it may occasionally be present to such a degree as to lead to difficulty in swallowing. Sexual feelings are diminished or altogether suppressed. The general aspect of a case of well-marked bromism much resembles one of senile imbecility.

2. *On the Skin.*—A very frequent result of the internal use of the bromides is a follicular eruption of the skin, closely resembling acne. It is generally situated on the face, chest, and shoulders. When the use of the drug is persisted in, the acne becomes aggravated, and boils appear. A more rare form of skin-disease similarly caused has been described by Dr. Cholmeley, Mr. Hutchinson, M. Voisin, and others. This disease appears as vesicles, which become aggregated into clusters or patches. They proceed to suppuration, and are soon followed by scabbing, their base being slightly raised, hard, sometimes ulcerated, and surrounded by a red areola. In a later stage the eruption presents the appearance of dusky red stains. It has been observed more especially on the limbs and head. Eruptions having more or less the characters of erythema and of eczema have also been described as following the use of these agents.

3. *On the Mucous Membranes and Glandular Structures.*—Dryness of the mouth and tongue is often experienced in bromism; but in some cases there is said to be an increased flow of saliva. Nausea, flatulence, eructations having a saline taste, heat and fulness at the epigastrium, and occasionally gastric catarrh and diarrhoea, have been observed; it is said that acute enteritis and even a typhoid condition have occurred.

4. *On the Organs of Circulation and Respiration.*—The salts of bromine are said to produce contraction of the capillaries.

The skin may present a peculiar dusky hue or pallor, and the extremities are cold. The action of the heart is rendered slower and weaker; and may even cease altogether, under the continued operation of these drugs. The action on the respiratory organs is similar to that upon the heart. It has been observed that bromine—recognised by its peculiar odour in the expired air—is eliminated from the respiratory mucous membrane. Bronchial catarrh occurs, and instances are recorded in which pneumonia is said to have followed and proved fatal.

It must be remembered that, although it has been thought desirable to discuss separately here the effects of the bromides on the several systems, these effects are combined in various degrees. In some cases the affection of the skin is alone noticeable; in others, that of the nervous system; while in a third class there is produced a combination more or less of all the phenomena, constituting what may be called a *cachexia*. In such cases we find loss of flesh and strength, of colour and mental power; paralysis of the muscles; loss of reflex and general sensibility and of the functions of the special senses; complete apathy and general prostration, the countenance having a semi-idiotic expression; coldness of the extremities; and gradual failure of the heart's action.

*PATHOLOGY.*—The condition just described is but the extreme effect of the ordinary physiological action of the salts of bromine. This condition may be due either to idiosyncrasy—that is, to undue susceptibility on the part of the individual—or to the administration of large quantities of the drug, either in medicinal doses for a long period, or in excessive doses administered within a short time. The individual susceptibility may depend upon the want of capability to eliminate the drug; on the general state of health; or on the presence of a disease which resists its action. Under these circumstances, as well as in the presence of certain modifying influences, such as the action of other remedial agents simultaneously administered, it is difficult to fix upon the amount of a bromine salt capable of producing morbid symptoms in any given individual. The writer has seen a nightly dose of ten grains of bromide of potassium, continued for some weeks, produce marked somnolence during the day, and impairment of memory; whilst it has required the enormous doses of 200 or 300 grains a day, which seem to be administered on the Continent, to produce the extreme effects above described. The rapidity with which these effects are produced, constituting the *acute* and *chronic* forms of bromism, will depend on the amount and frequency of the dose, and on the susceptibility of the individual. The effect of a sudden considerable increase in the dose has been observed by the writer in a case which

first directed his attention to the subject of bromism in 1872. This case he saw in consultation with Mr. Alfred Burton. Half-drachm doses of bromide of potassium had been taken twice a day for several weeks, when by mistake the quantity of the drug was doubled; then, after three days, symptoms closely resembling senile imbecility were rapidly developed.

**DIAGNOSIS.**—Recognising the value of the bromides,<sup>1</sup> and the frequency with which they are used, it is extremely important that the peculiar results which they are capable of producing should be borne in mind; for if they are not recognised in time, and if the use of the drugs be persisted in, disastrous effects, which might otherwise be avoided, will follow. Without going into details of diagnosis, it will probably be sufficient to point out the necessity for remembering that the symptoms which have been described above can be produced by the use of bromides; and that when such a combination of symptoms does occur during their use, it is highly probable, in the absence of disease capable of accounting for them, that the symptoms have originated from the operation of these agents.

**TREATMENT.**—This consists in stopping the use of the drug, and hastening its elimination by promoting the action of the kidneys and other excreting organs. It is found that arsenic in combination with these salts acts in some measure as a preventive of the eruptions.

The above description refers more especially to the effects of bromide of potassium; but like effects are produced by other salts of bromine, though to what extent by each has not been so clearly ascertained.

RICHARD QUAIN.

**BRONCHI, Diseases of.**—**SYNON.**: Fr. *Maladies des Bronches*; Ger. *Bronchien-Krankheiten*.—The diseases of the bronchi will be discussed in the following order:—

1. Acute inflammation; 2. Chronic inflammation; 3. Plastic inflammation; 4. Dilatation; 5. Narrowing or obstruction; 6. Cancer.

**1. Acute Inflammation—Acute Bronchitis—Acute Bronchial Catarrh.**

**DEFINITION.**—An acute inflammation or congestion, general or partial, of the bronchial tubes.

**ÆTIOLGY.**—The causes of acute bronchitis may be classed as (a) *predisposing*, and (b) *exciting*.

(a) *Predisposing causes.*—Of these age is one of the most important. The disease is indeed confined to no period of life, but is most frequently met with in the young and the

<sup>1</sup> It is of interest to record here that the use of bromide of potassium in this country dates from May 1857, when its value in the treatment of hysteria was mentioned by Sir Charles Locock at a meeting of the Royal Medical and Chirurgical Society, of which he was then President.

old; and in these subjects it assumes its most serious characters. The imperfect development of the infant, and the diminished vitality of the aged, seem to render them especially liable to attacks of bronchitis, and to make the disease exceptionally fatal in them. Sex appears to have no influence as a predisposing cause. The habits of life have an important influence in the causation of bronchitis. The practice of living in heated rooms, especially where gas is largely consumed, and of breathing the vitiated atmosphere produced by the assemblage of large numbers of persons in apartments, is undoubtedly a fertile predisposing, as well as exciting, cause of the complaint; so also is the practice of keeping children too much within doors on the one hand, or, on the other, of exposing them to inclement weather when insufficiently clad. Temperament can scarcely be considered a predisposing cause, but the state of the general health exercises a powerful influence. A weakly constitution, or one weakened by overwork, improper food, &c., predisposes to bronchitis; whilst such affections as Bright's disease, gout, and diseases of the heart, alike favour its occurrence. Again, certain occupations are favourable to the development of bronchitis. Independently of the fact that living or working in heated and confined rooms predisposes to the disease, such occupations as lead to the inhalation of irritating particles, as those of steel, cotton, &c., give rise to it. The climate most favourable to the production of bronchitis is probably that which is at the same time both cold and damp, and where sudden variations of temperature occur. The seasons of the year in which it prevails most are the late autumn, the winter, and the early spring.

(b) *Exciting causes.*—Although undoubtedly cold directly applied to the surface of the body is in a large number of cases the exciting cause of bronchial inflammation, still the transition from cold to heat—passing from a cold atmosphere to a heated one—is an important factor of the disease. There can be little doubt that bronchitis is often produced directly by the effects of heated and vitiated air on the bronchial membrane, and on the system at large; and that, in the latter instance, the affection is merely a local manifestation of a general influence. Bronchitis may also be caused by the direct action of irritants contained in the air—as irritant vapours, minute particles of steel, cotton, or ipecacuanha, and the emanations (pollen) from flowering plants. Again, morbid conditions of the blood, the result of specific febrile affections, act as exciting causes of the disease; as do also the poison of syphilis, and the altered condition of the blood produced by gout. Bronchitis, moreover, must be regarded as a constant accompaniment of influenza.

**ANATOMICAL CHARACTERS.**—The mucous membrane is mainly affected in acute bronchitis, but morbid changes may be produced in the deeper structures. The mucous membrane is red—the redness being arborescent, streaked, or mottled, but not usually spread uniformly over a large surface. The injected condition of the membrane does not, as a rule, extend into the finer bronchial tubes, but in some cases where there have been frequent attacks of inflammation, these tubes have a red appearance. The membrane is sometimes thickened and soft, but ulceration is very rare. The tubes are generally found more or less filled with secretion, either frothy mucus, muco-pus, or even actual pus. Sometimes the secretion is very abundant, filling all the tubes. Fibrinous masses are occasionally met with, which may form casts of the tubes. Collapse of portions of lung-substance—lobulæ or whole lobules of the lungs—is not infrequently found, as are also patches of lobular pneumonia. The venous system and the right side of the heart are overloaded, and the blood is dark. In many cases fibrinous deposits are found in the cavities and great vessels of the heart.

In speaking of the pathology of bronchitis, it is necessary to refer to the distribution of the bronchial blood-vessels. The bronchial arteries when they have fairly entered the lungs have no accompanying veins. The so-called bronchial veins are some small vessels which return the blood supplied to the structures about the roots of the lungs. The blood which is supplied to the bronchial tubes, when they have commenced their divisions, passes into radicles of pulmonary veins, and is returned directly to the left side of the heart. The question whether there is a communication between the bronchial arteries and the pulmonary artery, is still *sub judice*. If such communication exist, it is only slight. The blood of the bronchial arteries, after supplying the mucous membrane and other structures of the tubes, passes, either wholly or in a very large part, to the left side of the heart, not having circulated through the aërating portion of the lungs. The circumstances of this anatomical arrangement are most important in a practical point of view. Anything which embarrasses the circulation on the left side of the heart—such as mitral regurgitation—must necessarily cause a very loaded condition of the bronchial vessels; and all physicians are familiar with the form of bronchitis which is so common in these cardiac affections. The congested mucous membrane, and the profuse bronchial secretion, are the result of the direct impediment to its circulation which the blood meets with, from passing at once into vessels which go straight to the left side of the heart. The relief often afforded in this form of bronchitis

by the exhibition of digitalis, is explained by the circumstance above referred to.

**SYMPTOMS.**—The symptoms of acute bronchitis vary according as the larger or smaller tubes are affected. The disease attacks first, the larger and medium-sized tubes; and, secondly, the smaller ones. To this latter form of the affection the name of *capillary bronchitis* has been given.

1. Acute bronchitis of the *larger tubes*. The attack is usually ushered in by symptoms of catarrh—sneezing, lachrymation, a sense of fullness about the nose and eyes, with frontal headache; the throat becomes dry and sore, and then increased secretion sets in; the follicles at the back of the pharynx become enlarged; the upper part of the larynx is often involved, there being slight hoarseness; and the affection gradually creeps down into the bronchial tubes. The disease is not ushered in by decided rigors, but chills and sometimes shiverings are experienced; the pulse is not much affected, but its frequency is increased in some cases; there is a general sense of malaise, as well as a want of energy. When the disease has set in fully certain local symptoms are found. More or less pain is felt behind and above the sternum; the sensation is increased by a deep inspiration; the pain shoots at times over the chest in the direction of the larger bronchial tubes; and there is a tickling or unpleasant irritation felt behind the sternum, which gives rise to a cough. Dyspnoea is not a marked feature of this form of bronchitis; it exists, however, sometimes; and in the most severe cases a sense of oppression, weight, and tightness about the chest is experienced. Cough is one of the earliest and most prominent symptoms; it is at first dry, and there is usually at this period some hoarseness. The cough is paroxysmal, and often very violent; it becomes attended with expectoration as the disease progresses. This varies at different stages of the affection; at first watery and frothy, and almost transparent, it becomes, as the disease progresses, more consistent, viscid, and opaque, passing through the stages of mucus to muco-pus and pus; it is sometimes distinctly nummulated. Small streaks of blood are occasionally seen mixed with the sputa. Examined under the microscope the sputa are found in the early stages of the disease to contain epithelial cells from the mucous membrane; and, later, many of the so-called exudation-corpuscles, molecular and granular matter, pus-cells, and occasionally blood-discs.

In the milder cases of this form of bronchitis there is but little general disturbance; and even in the more severe cases the febrile reaction is not usually very great. The pulse rises a little, but does not become very frequent; the temperature rarely becomes high; there is in many cases but little interference with the appetite. A general feeling

of depression, which in some cases is very marked, is usually experienced.

2. Acute bronchitis of the *smaller tubes*—*Capillary bronchitis*. This is a very formidable disease. It attacks the finer bronchial tubes, and probably extends to their smallest ramifications. Its symptoms are very grave. Some of the worst cases of capillary bronchitis are met with in connexion with emphysema of the lungs. It may be an extension of inflammation from the larger tubes; or the capillary tubes may be attacked simultaneously with the larger ones, or alone. The early symptoms are more severe than those of ordinary bronchitis, and rigors are more common. Dyspnoea is marked; it may vary from mere rapid respiration to constant or paroxysmal orthopnoea. The respirations may rise to fifty in a minute. Cough is almost continuous, at times becoming very violent and most distressing. Expectoration is attended with difficulty. The sputa soon become very abundant, and rapidly assume a purulent character; or they are very viscid and ropy.

The general symptoms are very severe. The fever is high—the temperature reaching to 103° Fahr. and upwards; and the pulse is frequent, rising to 120 or 140. The temperature rarely attains the height which characterises acute tuberculosis or pneumonia. There are often profuse perspirations, and in some cases excessive debility is felt. If the disease progresses unfavourably, symptoms of very imperfect aëration of the blood come on. The face becomes turgid and bloated, the lips and ears get livid, the veins are distended, the temperature falls, cold clammy perspirations break out, the pulse becomes very small and rapid, delirium supervenes, the respiration is shallow and catching, and the patient dies of asphyxia, and from the presence of fibrinous clots in the heart and great blood-vessels.

PHYSICAL SIGNS.—The physical signs of both forms of acute bronchitis may be referred to together. Inspection reveals little of practical value in simple bronchitis. The chest-form is not altered. In severe cases the abdominal movements are in excess. The costal movements are frequently those of elevation rather than expansion. In extreme cases the lower end of the sternum and the connected cartilages sink with inspiration; while the expiration-movements are slow, laboured, and inefficient. If the hand is applied to the chest, rhonchal fremitus may be often felt, sometimes over a large area. The percussion-sound may be somewhat exaggerated from over-distension of the lungs, especially in children; not appreciably altered; or deficient in resonance, owing to the accumulation of secretion at the bases of the lungs, to œdema or congestion (as in typhoid fever), or to pulmonary collapse. In young children a sound re-

sembling the cracked-pot sound may be occasionally produced, variable in site. The sounds heard in auscultation vary according to the stage of the disease. The breath-sounds are loud when the tubes are free; when the latter are plugged by secretion, they often become feeble or even totally suppressed, from closure of a tube leading to a portion of the lung. The adventitious sounds of bronchitis include the various rhonchi, dry or moist: the dry rhonchi are heard in the early stages of the disease for the most part, but when once secretion has set in, the moist rhonchi or râles are more or less extensively heard, depending for their character on the size of the tubes which are the seat of inflammation. Thus they are called *mucous* when produced in the large tubes, *sub-mucous* and *sub-crepitant* when produced in the finer ones; the latter term being used to characterise the râles of capillary bronchitis. When the large bronchial tubes are filled with a secretion which is not viscid, the sounds may have a rattling character. The various rhonchi may be heard over different parts of the lungs at the same time, according to the seat and stage of the bronchitis. In capillary bronchitis sub-crepitant râles, accompanying inspiration and expiration, are abundantly heard towards the bases of both lungs especially. As a rule there is no displacement of organs in bronchitis, but the diaphragm is sometimes depressed from great distension of the lungs, and the heart is occasionally displaced towards the right.

DIAGNOSIS.—The diagnosis of acute bronchitis, except in a few instances, presents no great difficulty. In the early stages of whooping-cough it is impossible to decide whether the case is one of simple bronchitis or not, but subsequently the paroxysmal character of the cough settles the point. In some cases of bronchitis occurring in children the breathing may resemble that of croup; but here the presence of catarrh, the wheezing nature of the respiration, the absence of much fever, the characters of the sputa—obtained by wiping the back of the tongue—and their freedom from membranous shreds, and the physical examination of the chest indicating the presence of rhonchi, will be sufficient to establish a diagnosis. From laryngitis the discrimination is not difficult.

Pneumonia may generally be easily diagnosed from capillary bronchitis, with which form it can perhaps be alone confounded. Capillary bronchitis is not ushered in, as pneumonia usually is, by a well-marked and prolonged rigor; the general febrile disturbance is less, and the temperature not so high; moreover the absence of dulness on percussion, and of increased vocal resonance and fremitus will aid in the differentiation. From lobular pneumonia in children the diagnosis is not always easy. In this

disease there is often no dulness to be perceived on percussion; whilst, on the other hand, dulness may exist in bronchitis from pulmonary collapse.

The diagnosis of capillary bronchitis from acute phthisis often presents difficulties. The main points to be relied on, independently of the family history, which may aid, are that in capillary bronchitis the fever is less and the temperature lower; signs of asphyxia soon come on; and there is free expectoration of muco-purulent matter, from which the tubercle-bacilli are absent. In one form of acute phthisis there is evidence of pneumonic consolidation, followed by signs of the formation of cavities. In the miliary tubercular form there are in many cases scarcely any physical signs except râles, most marked at the apices of the lungs. Here, however, the diagnosis may be aided by the examination of the sputa under the microscope, which may reveal the presence of tubercle-bacilli.

**PROGNOSIS, DURATION, TERMINATIONS, AND MORTALITY.**—The prognosis in an ordinary case of bronchitis is favourable, but when the disease occurs in the very young or the aged the prognosis should always be guarded. In the milder forms the affection may last only a few days, or two or three weeks. Severe cases are more protracted. The disease may terminate in perfect recovery, in death, or by passing into the chronic form. It may be the starting-point of emphysema of the lungs, or of certain forms of phthisis. The mortality is much influenced: (1) by age, being greatest in the very young and the very old; (2) by the previous state of health, which, if lowered by any circumstances, will render recovery more doubtful; (3) by the extent of the inflammation, especially when the disease is of the capillary form; (4) by the existence or non-existence of any organic disease of the heart, lungs, or kidneys; (5) by the disease being epidemic in connexion with influenza or otherwise; and, lastly, by the time the case has come under treatment, whether early or late.

**TREATMENT.**—In the treatment of bronchitis regard must be had to the constitutional condition of the patient. Care must be taken to ascertain whether the disease is secondary to some organic affection, or the result of mechanical irritation, or of the presence of gout or rheumatism in the system, or of influenza; or whether it arises idiopathically. The treatment of the disease as a primary affection will be considered first.

In an ordinary case of acute bronchitis it is very desirable to keep the patient confined to his room, and, if the case is at all severe, to his bed. The temperature of the apartment should be maintained at from 60° to 65° Fahr. A higher temperature than this is generally not favourable to the progress of the case. In the early stages of the attack it is well to allow the air of the room

to be more or less saturated with steam. A free action of the skin should be promoted; and for this purpose warm drinks, with or without some form of alcohol or some diaphoretic medicine, may be given; or a hot-air bath may be used in bed. Great relief is often experienced from the application of a large mustard or mustard and linseed-meal poultice to the chest; and it is well, if mustard is applied first, to apply immediately afterwards a large hot linseed-meal poultice, to be renewed every few hours. This constant application of warmth and moisture to the chest is often productive of very great relief to the symptoms.

Cases of acute bronchitis do not require venesection, nor is the application of leeches often, even if ever, called for. Severe counter-irritation is moreover to be prohibited. It is generally desirable to act on the bowels, and a mercurial, followed by a saline purgative, will often be of great service. In the old and debilitated, as also in the young, all lowering treatment must, however, be avoided. In the early stages of the affection, before secretion has commenced, and when the mucous membrane is dry and the cough hard, diaphoretics with ipecacuanha may often be given with advantage; but as soon as secretion is fairly established, carbonate of ammonium, spirit of chloroform, ether, cascarrilla, senega, or such-like drugs should be administered. Indeed in almost every stage of bronchitis carbonate of ammonium is one of the most valuable remedies we possess. Care should be exercised, especially with the aged, that nothing should be given which will so nauseate as to prevent food being taken. In the exhibition of medicines to alleviate the cough, regard must be had to the condition of the patient and the stage of the disease. Opium in all its forms should be given with caution, especially in the young and old. It no doubt often succeeds in checking cough, but in doing so it also checks expectoration, and causes an accumulation in the bronchial tubes, which sometimes becomes very dangerous to life. Chloral hydrate in small doses is often of great use for relieving cough, and it may be combined with oxymel of squill. It has also a good effect in allaying spasm of the tubes, if this exist. In some cases of bronchitis the question of procuring sleep becomes an important one. Opium in its various forms is generally inadmissible, in consequence of its tendency to increase the condition of asphyxia; but chloral hydrate may be given with safety, and the recovery of a patient may sometimes be dated from the sleep which this agent procures.

In reference to the exhibition of alcoholic stimulants, except in the early stages, and in certain cases dependent on a gouty or rheumatic condition, they should usually be given in smaller or larger quantities. They increase

expectorating power, and ward off the tendency to asphyxia. In the old they are especially called for, and, together with carbonate of ammonium, should form the main therapeutic agents to be relied on. In the treatment of capillary bronchitis, ammonia and alcoholic stimulants should be exhibited from the commencement, and the quantity must depend on the symptoms of each case. There is one source of danger in capillary bronchitis which should always be borne in mind, viz. the formation of fibrinous clots in the heart and great blood-vessels. These deposits become the proximate cause of death in many cases, and they are especially liable to form when there is emphysema of the lungs. Their presence may often be diagnosed during life from the respiration becoming very rapid, shallow, and laboured; from the pulse being quick, weak, and small, although the heart may at the same time be felt beating vigorously; from the voice becoming feeble, and the mental faculties seriously impaired. After death a large portion of the cavities of the heart may be found occupied by these deposits, the calibre of the pulmonary artery and the aorta being also materially diminished by them.

In many cases of bronchitis, when the acute symptoms have passed off but the secretion continues profuse, as well as in those cases called bronchorrhœa, the exhibition of iron is often of great service. It seems to give tone to the relaxed capillaries of the mucous membrane, and to diminish the secretion. It may be given in combination with carbonate of ammonium, in the form of the ammonio-citrate; or the tincture of the perchloride with ether or spirit of chloroform may be employed, or the ethereal tincture of the acetate (Ph. Ger.), which is a very valuable preparation in some cases.

Inhalations are useful for allaying cough in the earlier stages of the affection, or for the relief of spasm. In some cases of severe bronchitis where asphyxia has been threatened, recovery has followed the exhibition of large doses—half an ounce—of oil of turpentine. In this dose, however, it sometimes produces alarming symptoms, and it is perhaps better to exhibit it in smaller quantities tentatively. An emetic may be serviceable, especially in children, if the tubes are much loaded. Children suffering from severe attacks of bronchitis should not be allowed to sleep long, for fear of dangerous accumulation in the tubes, and care should be taken that the secretions do not collect about the back of the mouth.

Patients should not be kept on a low diet even at the beginning of an attack, and as the disease progresses the quantity of food allowed may be increased according to the appetite. In the treatment of gouty bronchitis, or bronchitis associated with a tendency to the formation of uric acid in the system,

colchicum and the alkalis must be given, and the general measures used which are applicable to the constitutional condition. If bronchitis depend on a gouty state, it will not yield to the ordinary treatment, but when its cause is recognised and the appropriate remedies are administered, the symptoms usually soon begin to improve.

In the cases of bronchitis which are connected with heart disease, and especially with mitral regurgitation, digitalis is often of great value. By steadying the action of the heart it relieves the overloaded pulmonary veins, and thus directly diminishes the congestion of the mucous membrane, as mentioned in the paragraph relating to the pathology of the disease.

It is impossible in the scope of this article to refer specially to the treatment of bronchitic attacks arising from the various kinds of mechanical irritation. There is, however, one form of bronchitis which may be mentioned, viz. that connected with hay-fever, arising either from the inhalation of pollen, or caused by some peculiar atmospheric influence acting on a peculiar nervous system. It is very difficult of cure. In the writer's experience no remedies seem to have any particular influence over it, and it is usually only to be relieved by removing the patient from the exciting cause of the affection. In the treatment of bronchitis depending on constitutional syphilis, the appropriate measures for that affection must be resorted to.

## 2. Chronic Inflammation—Chronic Bronchitis—Chronic Bronchial Catarrh.

**DEFINITION.**—A chronic inflammation or congestion, more or less extensive, of the bronchial tubes.

**ÆTIOLGY.**—Chronic bronchitis very frequently results from repeated attacks of the acute disease, but it may be chronic from the beginning. Emphysema of the lungs, dilated bronchi, and phthisis are causes of the complaint; as are also various forms of heart-disease, and some blood-affections, such as gout. The inhalation of irritating particles gives rise to chronic bronchitis; and it is also met with in connexion with chronic alcoholism. It is most common amongst the old.

**ANATOMICAL CHARACTERS.**—The bronchial mucous membrane is discoloured, being of a dull-red tint, greyish, or brownish. The discoloration is usually partial, but sometimes general. There is swelling and increased firmness of the mucous membrane, and the sub-mucous tissue in old-standing cases becomes infiltrated and indurated. The fibrous and muscular tissues are hypertrophied; the cartilages in the larger tubes are sometimes calcareous; and there is generally more or less emphysema of the lungs.

**SYMPTOMS.**—The symptoms of chronic bronchitis vary greatly in different cases.

They resemble in kind those of the acute affection. There is cough, expectoration, pain, soreness or uneasiness behind the sternum, with more or less dyspnoea. The constitutional symptoms may be very slight, scarcely any effect on the general health being apparent; or they may be very severe. Three forms of chronic bronchitis are recognised clinically:—1. That which includes the ordinary cases of the disease, varying much in severity; 2. That characterised by excessive secretion—*bronchorrhœa*; 3. That form which is called *dry catarrh*.

1. In the first form of chronic bronchitis the cough is at first slight, perhaps only occurring during the winter, being altogether absent in the summer. After a time the attacks become more frequent, and at last the patient is never free from the affection, which is aggravated at times. The cough in such cases is more or less severe, but usually most so in the morning. It is often paroxysmal, and sometimes very violent. The expectoration, in some cases being scanty, viscid, and difficult to discharge, is in others, especially old-standing cases, copious and easy. The sputa vary much both in appearance and quantity. They may be yellowish-white, muco-purulent matter, or more decidedly purulent, of a greenish-yellow or bright or dark green colour; they are but little aerated, sometimes not at all, so that they sink in water; at times they are nummulated and quite opaque. In some cases the expectoration is fetid, constituting the form of the disease denominated 'fetid bronchitis,' the odour resulting either from sloughs of minute portions of the mucous membrane, or from chemical changes taking place in the sputa. Occasionally streaks of blood are met with. Microscopically the sputa are found to consist of epithelium, pus-cells, and granular matter, with at times blood-corpuscles.

The constitution does not suffer much in mild attacks, but when chronic bronchitis is permanent and general, the system at large sympathises more or less severely: the appetite fails, sleep is disturbed by the cough, emaciation sets in and sometimes becomes marked, but it does not proceed beyond a certain point, unlike that of phthisis, which is usually progressive. In all cases of chronic bronchitis there is great risk of an acute attack coming on, especially amongst the aged. These attacks are very dangerous, in consequence of the rapid extension of the disease throughout the lungs, and its asphyxiating character.

2. The second class of cases is characterised by excessive secretion from the bronchial tubes—*bronchorrhœa*. This form is often met with in the old and feeble, and especially in cases of valvular disease of the heart. The cough is paroxysmal, and attended with the expectoration of a large quantity of thin

watery glairy fluid, or of thick ropy gluey matter, like white of egg. The quantity expectorated is sometimes very large. This form of bronchitis may cause death somewhat suddenly by asphyxia. During the paroxysms of cough there is dyspnoea, but at other times it is absent, except when heart-disease exists. The constitution suffers little, and the flux seems sometimes to be beneficial in cases of obstructive cardiac disease.

3. The third variety, or *catarrhe sec*, is characterised by very troublesome cough, oppression of breathing, tightness of the chest, and sometimes severe dyspnoea. Expectoration is either absent or very scanty, the sputa consisting of small masses of tough, viscid, semi-transparent mucus. There is usually no febrile disturbance. The disease is met with in gouty people, and is often associated with emphysema of the lungs. Pathologically it seems to consist in a congested condition of the bronchial tubes.

PHYSICAL SIGNS.—Inspection reveals nothing abnormal in the form or size of the chest, unless emphysema of the lungs is present. The expansion in long-standing cases is usually deficient; the chest being raised more than in health. Expiration is often prolonged. Rhonchal fremitus may be felt more or less, depending on the state of the bronchial tubes. There is often increased resonance, from the presence of emphysema. The breath-sounds are more or less changed: they may be harsh and loud, and the expiration is prolonged in cases that have existed for a considerable period. The rhonchi vary: they are dry, coarse, moist, or bubbling according to the condition and contents of the tubes. Vocal resonance varies: it may be bronchophonic, normal, or deficient.

DIAGNOSIS.—There is usually but little difficulty in the diagnosis of chronic bronchitis. The affection is most likely to be confounded with phthisis, but the character and degree of the wasting, and the absence of increased temperature, of hæmoptysis, of tubercle-bacilli from the sputa, and of the physical signs of consolidation, will generally enable the practitioner to decide in favour of the less serious disease. The main difficulty lies in the diagnosis of cases where the bronchi are dilated; this will be referred to hereafter.

PROGNOSIS.—Although in itself not a dangerous malady, chronic bronchitis becomes so in consequence of the liability which exists to the occurrence of acute symptoms; when once established in middle or advanced age it is almost incurable. The complaint is further serious from its tendency to produce emphysema and dilatation of the bronchi. *Per se* it can scarcely ever be said to kill.

TREATMENT.—No case of chronic bronchitis can be successfully treated without due regard to the constitutional condition of the

patient. In some cases it is impossible to cure the disease, and all efforts should be directed towards preventing its extension; alleviating the symptoms to which it gives rise; and warding off acute attacks. Attention must be especially paid to the state of the heart and kidneys; the duration of the affection; the age of the patient; the characters of the expectoration; the state of the lungs, as to the existence of emphysema or other morbid conditions; and the presence of gout or rheumatism. Speaking generally, chronic bronchitis must be treated by the use of a generous diet, with more or less stimulants; by the exhibition of expectorants and tonics; and by the avoidance of all depressing measures. The function of the liver must be looked to; and the administration of a few doses of blue pill with a saline aperient often gives great relief, and alters the character of the bronchial secretion. If gout, or a tendency to the formation of uric acid, is present, colchicum with alkalis and other remedies for gout, such as a course of Friedrichshall or Hunyadi or Carlsbad waters, will prove of great service. If cardiac disease exist, whether in the form of valvular incompetence, or of weak, flabby, or dilated heart, digitalis combined with iron frequently produces marked benefit.

In the treatment of ordinary cases of chronic bronchitis, not dependent on any organic disease or constitutional condition, the patient's general health has to be looked to. The affection has a tendency to lower the health and to diminish strength, and therefore the various tonics may often be given with great benefit. Of these the most useful are quinine, the preparations of iron, and those of zinc. Cod-liver oil is also very valuable in some cases where there is much wasting. The cases of bronchitis marked by excessive secretion are generally best treated by tonics; whilst those where the secretion is slight—cases of 'dry catarrh'—being often associated with a gouty condition of the system, are more amenable to the use of colchicum, the alkalis, iodide of potassium, and mineral waters.

In what may be called the symptomatic treatment of the affection, the various expectorants are useful—carbonate of ammonium, ipecacuanha, squill, cascarilla, senega, chloroform, &c., and these may often be beneficially combined with some form of tonic. In many cases of chronic bronchitis the expectorating power is diminished, and stimulating expectorants are of great service. Much caution must be exercised in the administration of opiates and other narcotics or sedatives. When, however, the mucous membrane is very irritable, and when there is but little secretion, with a troublesome cough, these remedies are indicated. Opium is of great value, and chloral hydrate is also very useful, as well as, in some cases, henbane

and hydrocyanic acid, or, whenever spasm is present, stramonium, lobelia, the ethers, and cannabis indica. Inhalations are sometimes very beneficial, as of the vapour of warm water, iodine, creasote, and other substances. The inhalation of creasote is especially valuable if the expectoration is foetid.

Counter-irritation is one of the most important means we possess of relieving chronic bronchitis. The irritation should not be excessive, but should be long-continued. The application of iodine over a large surface of the chest, so as to keep up a constant slight inflammation of the skin, is perhaps the best that can be used; but other irritants may be tried, such as sinapisms, or the various stimulating liniments.

The general management of the patient is most important. A mild climate should, if possible, be chosen in the winter. The patient should live as far as possible in an atmosphere which is mild and dry. Although some cases are benefited by a moist and warm atmosphere, the majority of cases of chronic bronchitis do better in a drier one. The skin must be carefully looked to, its action should be well maintained, and warm clothing always worn. A moderate amount of some alcoholic stimulant is generally desirable; and the food should be nutritious and of easy digestion. Relief will often be found from wearing a respirator.

**3. Plastic Bronchitis.**—This is a rare form of disease, and of its particular causes nothing is known. It is perhaps connected with some peculiar diathesis. The late Sir John Rose Cormack suggested that it may be a variety of diphtheria. It may occur in either sex, and at any period of life, but is most frequent in those who are of a strumous or phthisical constitution. It has been known, however, to attack persons of apparently healthy frame, and in the enjoyment of robust health.

**ANATOMICAL CHARACTERS.**—Plastic bronchitis is anatomically characterised by the formation of concretions in the bronchial tubes. These concretions consist of fibrinous exudation from the mucous membrane; they form casts of the tubes, and are expectorated. These casts are either solid or hollow, and on examination are always found to consist of concentric laminae. They are, for the most part, poured out into the finer bronchial tubes, sometimes, however, into the larger ones, but never into the trachea. The casts are of a whitish colour, but they are often stained with blood. Microscopically they consist of an amorphous or fibrillar material, with exudation-corpuscles, granular matter, and oil-globules.

**SYMPTOMS.**—This disease is essentially chronic, but it has been met with as an acute affection in children. At the times when the casts of the tubes are expelled, exacerbations occur, the patient being attacked

with pain and a sense of constriction across the chest, dyspnoea, and an irritating cough. After a time, varying from some hours to a few days, the dyspnoea becomes very urgent, and the cough very severe; then, after a paroxysm of coughing, it is found that the patient has expectorated some solid material, either with or without blood, usually intermixed with ordinary bronchitic sputa. The dyspnoea and cough now subside, to recur after an interval of a few hours or longer. This disease may last for weeks, months, or even years, marked from time to time by severe accessions, and relieved by the expulsion of further concretions. The matter expelled is often in small masses, but at times casts of bronchial tubes with several ramifications are expectorated. The disease may recur at intervals for many years; the general health in such cases does not seem to suffer, the breathing during the intervals being unaffected. There is, in some instances, an absence of febrile symptoms during the attack, whilst in others the fever is more marked. With the general symptoms are combined the *physical signs*. These are somewhat peculiar. The bronchial tubes being obstructed, portions of lung are deprived of air; the breath-sounds are therefore faint or absent. There may be dulness more or less complete on percussion, from collapse of the lung-substance, or, as occurs in some cases, from localised pneumonia. Asphyxia may be threatened if a large tube is blocked up.

**DIAGNOSIS.**—The diagnosis of this affection turns on the peculiarity of the expectorated matters. Doubtless the disease may be mistaken for ordinary bronchitis or pneumonia; but when once the fibrinous casts of the tubes are observed in the sputa, the nature of the case becomes clear.

**PROGNOSIS, DURATION, TERMINATIONS, AND MORTALITY.**—The prognosis, if the disease is uncomplicated, is favourable; but there is a great liability to recurrence. The complaint may last for many years, and may terminate in complete recovery. A fatal result generally depends on the presence of some other organic disease, such as phthisis or pneumonia.

**TREATMENT.**—But little can be advanced as to the value of any special treatment for this affection. Iodide of potassium is said to have been employed with success. The chief object should be to maintain the general health by hygienic measures, and the exhibition of tonics, such as iron, quinine, and cod-liver oil, especially if there be any tubercular taint. During the exacerbations the administration of ammonia and the use of inhalations should be resorted to, and the general principles on which ordinary bronchitis is treated should be carried out.

4. **Dilatation—Bronchiectasis.**—This is a rare disease, which arises as a secondary

affection. It is often associated with serious pulmonary mischief, and is at times difficult of diagnosis. There are two forms of bronchiectasis, namely, *general or uniform dilatation*; and *saccular or ampullary dilatation*.

(1) The *general* or uniform bronchiectasis consists in a cylindrical dilatation of one or more of the tubes throughout a considerable portion of their extent. The tubes are evenly widened for the most part, and the dilatations end abruptly.

(2) The *saccular* form of bronchiectasis consists of a globular dilatation of a tube at one point, or at several points. The dilatations vary in size, being from half an inch to an inch or more in diameter. On the tracheal side they usually communicate with a slightly enlarged bronchial tube, whilst on the peripheral side the continuity of the tube is almost or entirely lost from narrowing or actual obliteration. Sometimes the cavities communicate with one another.

The two forms of bronchiectasis often coexist.

The walls of the dilatations undergo changes in the course of the disease. The mucous membrane becomes granular, swollen, and congested; while at a later stage it presents a velvety or villous appearance, and in some cases there is even ulceration with superficial necrosis. The muscular and elastic coats become atrophied, and, coincidentally with this, dilatation increases. At times the wasting of these coats is partial; some portion of the walls retaining their natural volume, and forming bands or ridges elevated above the surrounding membrane. The dilated tubes occasionally present an appearance of hypertrophy; the walls are thickened, but the thickening depends on changes which have taken place in the mucous membrane. The cartilages resist the destructive metamorphoses longer than the other structures, but they sometimes partake of them. The contents of the tubes may be either muco-pus, or pus; and casts of the minute bronchi are met with. At times the contents are very fetid. Crystals of margaric acid are occasionally found, and sometimes fragments of pulmonary tissue. It is said that the contents may become calcareous.

Dilatation of the bronchi may be unattended with any change in the surrounding lung-tissue, but, generally speaking, condensation of the latter takes place, as the result either of pressure or of chronic pneumonia. In some instances the tissue forms an abscess, in the centre of which the walls of the bronchus are found, whilst in others the walls of the bronchi and the surrounding tissue are destroyed by gangrenous inflammation. It is generally not difficult to distinguish between a phthisical cavity and a dilated bronchus. The latter is not characterised by the broken irregular surface which

usually exists in the former; its shape is generally more regular; and it is usually continuous with bronchial tubes. The surrounding lung-tissue has no tubercular infiltration.

The *mechanism* of bronchiectasis has occupied much attention. It is probable that the elastic and muscular fibres lose their elasticity and contractility as the result of chronic inflammation, and thus yield to the distending influence of coughing. When once a dilatation is produced, accumulation of the secretions takes place, which tends further to increase the dilatation.

**SYMPTOMS.**—The symptoms of bronchiectasis are those of chronic bronchitis aggravated in some important respects. The cough is frequent and paroxysmal. The expectoration is very abundant, very purulent, and, when the disease has lasted some time, very fetid. The breath also becomes fetid. Hæmoptysis is occasionally met with, even to a considerable extent. There is more wasting than in ordinary bronchitis, and the blood is more imperfectly aerated. Night-sweats are not uncommon. In fact, the general symptoms approach those of phthisis. The digestive functions are usually not much impaired.

**PHYSICAL SIGNS.**—The movement of expansion is diminished in bronchiectasis, while that of expiration is prolonged. Over the affected portions of the lung there may be slight retraction. Vocal fremitus is increased, and rhonchal fremitus is sometimes well marked. The percussion note is altered. If a dilated tube is surrounded by condensed lung-tissue, or is full of secretion, there is dullness on percussion; but if it is situated near the surface and empty, some degree of tubular resonance may exist. Cracked-pot sound may be, at times, elicited. The respiratory sounds are harsh, or loudly bronchial with a more or less blowing character, and they may be distinctly cavernous. Vocal resonance is often greatly increased. The pulse becomes rapid in the later stages. The temperature rarely if ever reaches the height that it does in phthisis with cavities, and the daily oscillations are not so marked.

**DIAGNOSIS.**—The main difficulty as regards diagnosis is in the differentiation of certain cases of phthisis with cavities from bronchiectasis with large globular dilatations. The points to be relied on are that in the latter disease the morbid physical signs are usually met with at the middle and lower parts of the lungs, whilst in ordinary phthisis they are found at the apex; that the temperature differs in the two affections as mentioned above; that emaciation and night-sweats are not so marked in bronchiectasis; and that, if cases are watched, there is usually observed a progressive advance of symptoms in phthisis, whilst in bronchiectasis the symptoms may remain stationary. In phthisis signs of consolidation precede those

of cavities, whilst they follow them in bronchiectasis, and in the latter disease tubercle-bacilli are absent from the sputa. Bronchial dilatations and tuberculous cavities have been found in the same lung. The fetor of the breath and sputa in bronchiectasis may cause a suspicion that gangrene of the lung is present; but the general symptoms will usually enable the practitioner to differentiate between the two affections.

**PROGNOSIS.**—Bronchial dilatation is probably never cured. It may last for years.

**TREATMENT.**—The treatment of bronchiectasis must be that of chronic bronchitis with the use of such measures as are applicable to wasting diseases in general. The fetor of the breath is best relieved by the inhalation of creasote, or some other antiseptic. The disease has, in some cases, been treated successfully by incision and drainage.

**5. Narrowing or Obstruction.**—Narrowing or obstruction of the bronchial tubes is by no means uncommon, and may depend on intrinsic or extrinsic causes. Complete obliteration of a tube is sometimes found in connexion with bronchiectasis, immediately beyond a globular dilatation.

**ÆTIOLOGY.**—The *intrinsic* causes of obstruction are a thickening of the mucous membrane resulting from bronchitis; the retention of viscid secretions; the exudation of plastic material into the interior of the tubes; and the deposition therein of tubercle or cancer.

Amongst the principal *extrinsic* causes are the pressure of adjacent tuberculous or cancerous deposits; the contraction of plastic matter exuded into the tissues surrounding the tubes; solid formations in the pleura; enlarged bronchial glands; and aneurysmal and other thoracic tumours.

Obstruction is most frequently met with in the smaller tubes, but the pressure of thoracic tumours not infrequently causes obstruction, or even obliteration of a main bronchus, which occasionally—as in the case of aneurysms—becomes perforated.

**SYMPTOMS.**—If a large bronchial tube become suddenly and greatly obstructed, dyspnoea of an urgent character sets in, and death from asphyxia may speedily result, unless the obstruction be removed. When the obstruction is on a smaller scale, being confined to the smaller tubes, or when a large tube suffers only from slow, gradually increasing obstruction, the symptoms are by no means urgent for a time, and slight dyspnoea, sometimes accompanied by stridor, is the most marked feature.

**PHYSICAL SIGNS.**—Complete obstruction of a bronchial tube invariably leads to collapse of the portions of the lung to which the tube is distributed, and thus an entire lung may collapse if its main bronchus be obliterated. Where partial collapse is produced, emphysema of the neighbouring lung-tissue

commonly follows, and if one lung become collapsed, the opposite lung becomes enlarged and emphysematous. The existence of collapsed lung gives rise to dulness on percussion over the affected part, unless this be situated away from the chest-walls, or masked by the presence of emphysema. Further, obstruction of the bronchi causes a weakness or deficiency of the respiratory sounds, with a prolonged expiratory murmur, attended at times with sonorous and sibilant rhonchi. Over the collapsed portion of the lung, or over portions of the lung supplied by a tube which has become completely obstructed, the breath-sounds are absent. Deficiency or absence of vocal vibration is another physical sign of obstructed bronchial tubes.

**TREATMENT.**—The treatment of obstruction of the bronchi must depend on the nature of its cause. The chief interest of the affection arises from the means of diagnosis of thoracic tumours which it may afford.

**6. Cancer.**—Cancer of the bronchial tubes occurring independently of cancer in the lungs, or mediastina, is probably never seen; but cancerous matter has been found in the tubes: (1) in cases where the lungs have been infiltrated with a similar deposit; (2) where a cancerous tumour connected with the root of a lung has perforated a tube; and (3) in some cases of cancerous disease of the lung, a tumour of a similar nature being found connected with the mucous membrane of a tube. (4) Cancerous matter has also been found *in transitu* in a tube, having been detached from a cancerous mass.

A. T. H. WATERS.

**BRONCHIAL GLANDS, Diseases of.**—**SYNON.**: Fr. *Adénopathie Trachéo-bronchique*; Ger. *Krankheiten der Bronchialdrüsen*.

**DEFINITION.**—Disease of those lymphatic glands which are situated at the bifurcation of the trachea, between the right and left bronchus, or upon these tubes and their primary divisions.

**GENERAL DESCRIPTION.**—A short reference to the anatomical relation of these glands in connexion with their pathological and clinical history will be useful. Taking the bifurcation of the trachea as a starting-point, we find a group of glands in the space between the right and left bronchus. They are from ten to fifteen in number, and they vary in size from that of a small pea to that of an almond. The glands towards the right bronchus are larger than those towards the left. Glands are also situated upon the tubes; they are few in number and small. The vascular supply of the glands, which is free, is derived from the bronchial arteries, and the blood is returned to the bronchial veins. Afferent lymphatics reach the glands from the lungs, from the pleura, from the neck and other neighbouring parts. Besides

these groups of comparatively large glands, numerous minute lymphatic glands are found in connexion with the primary divisions of the bronchi, chiefly at the back of these tubes, at their bifurcations, and at those of the pulmonary artery. The central group of glands is in relation in front with the pericardium, the arch of the aorta, and the pulmonary artery; behind with the pulmonary plexus of nerves, the œsophagus, the aorta, the vena azygos, &c. The ganglia on the upper, anterior, and posterior surfaces of the right bronchus are four or five in number and smaller than those of the central group. Their situation brings them into relation with the arch of the aorta, the innominate and subclavian arteries, with the brachio-cephalic vein, and with the vena azygos, the pneumogastric nerve, and its recurrent branch. The ganglia on the left bronchus are still smaller than those of the right side. Their position gives them relations with the arch of the aorta, the origin of the left carotid and subclavian arteries, the left branch of the pulmonary artery, with the large veins, with the left pneumogastric nerve, and especially with its recurrent branch. Lastly it should be stated, as a guide in clinical examination, that the bifurcation of the trachea takes place at the back in front of the body of the fifth dorsal vertebra, or between the fourth and fifth, and in front behind the lower end of the first bone of the sternum. The glands, except when diseased, are proportionately larger in children than in adult or aged persons. Knowing that these glands, in common with other lymphatic glands, are liable to such diseases as enlargement, abscess, morbid deposits, growths, and other textural changes, and bearing in mind, as just mentioned, their relations to surrounding organs, we can readily see how important is the study of their diseased conditions. Not only is their study important in reference to the diseased glands themselves, but also by reason of their modifying or masking the symptoms of disease in other organs, as the results of pressure which, when enlarged, they cause on nerves, air-passages, blood-vessels, &c. Throughout English and foreign medical literature numerous cases will be found described, in which there existed, more or less conspicuously, striking disease of the bronchial glands, little notice, however, being taken of less marked, though far more numerous, examples of disease. It is only within a comparatively recent time that the subject has received special attention and been discussed as a disease *per se*.

MM. Rilliet and Barthez, in their well-known *Traité des Maladies des Enfants*, have described the disease in infants, and Dr. West, in his work on *The Diseases of Infancy and Childhood*, has fully and clearly described—under the head of Bronchial

Phthisis—the tubercular diseases of these glands in young subjects. It is, however, to the late M. Noël Guéneau de Mussy, following up and widely extending the investigations of his predecessors, that we are especially indebted for our knowledge of the effects of these lesions, and to his pupil M. Baréty, who has published an exhaustive memoir upon them, under the title *L'Adénopathie Trachéo-bronchique*. The subject of disease of the bronchial glands has attracted the present writer's notice since (or even before) the year 1853, and the notes of more than sixty cases which have fallen under his observation will form the basis of some of the conclusions to be subsequently stated in this article.

**MORBID ANATOMY AND PATHOLOGY.**—The bronchial glands participate in the diseases which affect lymphatic glands generally, and which will be found described in a subsequent article (see **LYMPHATIC SYSTEM**, Diseases of). In this place it will suffice to enumerate the principal morbid changes to which they are liable, and the effects which these changes produce.

a. The bronchial glands are liable to *congestion* with enlargement, as are glands in other situations. *Hypertrophy* will be the result of this last condition becoming chronic. The glands in this situation become after childhood almost invariably studded with black deposits, the quantity of which may be so considerable as to constitute *melanoma*.

b. These glands are liable to *acute and chronic inflammation*. Acute inflammation in this situation, terminating in *abscess*, is rare, but several cases of the kind have been recorded. A case of acute inflammation of these glands associated with double hydrothorax will be found described by the writer's friend the late Dr. Moxon in the *Transactions of the Pathological Society*, vol. xix. p. 572. Chronic inflammation of the glands is by no means uncommon. It may lead to permanent enlargement, to contraction and induration of the glandular textures, with the presence of calcareous particles, or to chronic abscess. The contents of the abscess may be more or less completely absorbed, leaving a partially filled *sac* or *cyst*, containing thick pus, or cheesy or calcareous matter. But these glands, when inflamed and enlarged, may form adhesions with surrounding parts, and the contents of an abscess, if it exist, may be discharged, by an *ulcerative* process, into the substance of a lung, into the mediastinum, into the trachea or œsophagus, or even into a blood-vessel. *General emphysema* has occurred in some cases; whilst the emptied sac has assumed in some instances the character of a *cavity* connected with the lungs. When the matter is discharged into the air-passages, *purulent expectoration* is the result. Two or three examples of the kind were noticed amongst

the writer's cases, and the possibility of their being mistaken for the discharge from a cavity in the lungs, or an empyema, was remarked upon at the time. The abscess may discharge by a sinus in the neck or into the mediastinum. A remarkable instance of this kind is recorded in the case of the late much-lamented Dr. Fuller. A chronic abscess of the bronchial glands had opened into the posterior mediastinum. This led to pyæmia, to the formation of abscesses in the brain, and to the loss of a valuable life. Cases of great pathological interest illustrative of these points will be found recorded in the *Pathological Transactions*—in vol. xxv., p. 29, by Dr. Coupland; in vol. xxx., p. 254, by Dr. Barlow; in vol. xxxv., by Mr. Makins. In vol. xxxvi. Dr. Percy Kidd records two remarkable cases, in one of which sudden death was caused by the impaction of a caseous gland in the trachea, and a second case in which death resulted from profuse hæmorrhage caused by bronchial ulceration perforating the pulmonary artery, due to the pressure of a calcareous gland. In the same volume is recorded a case of suppurating bronchial gland by Mr. John Poland. Dr. Gee has recorded several cases of interest in vol. xiii. of *The Bartholomew's Hospital Reports*. The pressure of enlarged glands may cause dysphagia; and a series of cases illustrative of this point collected by Korne will be found noticed in the *Medical Chronicle*, vol. iii. p. 112. The pressure of enlarged glands on the pneumogastric nerve may have marked effects on the heart's action, causing great rapidity of the pulse or the contrary. See a Memoir by Dr. Marklen, *Bulletin des Hôpitaux de Paris*, tome iii., année 1887.

c. These glands are liable to suffer especially from *tuberculous* or *scrofulous disease*, from various forms of *malignant disease*, and in *secondary* or *tertiary syphilis*. Of the latter form of disease, some striking examples have fallen under the notice of the writer, in which symptoms closely resembling phthisis existed, but which yielded to treatment directed to the specific disease.

**ÆTIOLOGY.**—The causes which give rise to disease of the lymphatic glands being discussed in another article, reference will here be made only to the special circumstances which influence the particular glands; and thus it will be sufficient merely to allude, amongst *predisposing causes*, to hereditary predisposition, to general impairment of health, and the like. With regard to the influence of age and sex, reference has already been made to the works of Rilliet and Barthez, of Dr. West, and of other authors who described the frequency of the disease in childhood. The writer's observations made on young persons and adults show that of 58 cases (of whom 21 were males and 36 females—in one case the sex was not

recorded) 2 were under 10 years of age, 9 were between 10 and 20 years of age, 18 were between 20 and 30, and 26 were over 30 years of age. In three cases the age was not stated. If these observations justify any inference, it is that females are more liable to disease of the bronchial glands than males, and that the disease occurs with increasing frequency after the age of puberty. Amongst the *exciting causes* of disease in the glands we leave to be considered elsewhere those general conditions which give rise to disease in the lymphatic glands generally, such as scrofula, tubercle, malignant disease, &c., and pass on to the consideration of the more immediately *local* exciting causes. *Cold* leads frequently to congestion and enlargement of the bronchial glands. But it is to local irritation or inflammatory disease in organs or tissues with which these glands have a connexion that the source of disease may be frequently traced. As we find the sub-maxillary glands or cervical lymphatic glands enlarged from irritation or disease in the mouth or throat, or the axillary glands or inguinal glands enlarged from irritation or inflammation about the hands or feet, so we may find the bronchial glands enlarged temporarily or permanently from inflammatory disease in parts the lymphatics of which pass to these glands. They have also been observed to be enlarged in the course of or after certain acute specific diseases, such as scarlet fever, measles, and typhoid fever. In whooping-cough this enlargement has been so frequently observed by the late M. Guéneau de Mussy, that he believed this disease to be an exanthem of the bronchial mucous membrane, and that this local condition leads to enlargement of the glands, which, again, by pressure on the pneumogastric and recurrent nerves, gives rise to some of the special phenomena, such as crowing cough, and even to the vomiting so frequently observed in this disease. It is right to remark here that the late Dr. Hugh Ley speaks interrogatively, in his work on *Laryngismus stridulus*, of enlarged bronchial glands being capable of producing a cough like that of pertussis, and he further alludes to some cases of whooping-cough in which the glands by the side of the trachea were enlarged. He asks, 'May it not be that an enlargement of these glands from a specific animal poison, similar to that of the parotid gland in mumps, is after all the cause of whooping-cough?' (Note, p. 440.) The same author gives several striking illustrations of diseased bronchial glands pressing upon the pneumogastric and other nerves producing marked results. The black deposit so often found in the glands is the result of the absorption of carbonaceous or pigmentary matter from the lungs.

**SYMPTOMS.**—The symptoms which have been recorded by the writer as more or less

characteristic of the presence of enlargement of the bronchial glands are as follows:—

1. *Cough*, which is noted as being a prominent symptom in 39 cases. In 21 of these cases it was stated to have been the most troublesome of the symptoms present. In 6 cases it was described as harsh and laryngeal; in 4 cases spasmodic, resembling whooping-cough. In other cases, 5 in number, it was characterised as short and hacking, constant, incessant, and in one case the sound resembled that made by the peculiar cough of a sheep.

2. *Pain* is, in regard to the frequency of its occurrence, the next symptom recorded. It was mentioned as being present in 22 cases. The seat of pain was almost constantly referred to the situation of the fourth and fifth dorsal vertebræ, at one or both sides of the spinal column. The pain was mentioned in a few cases as existing only in front, beneath and at one or both sides of the upper end of the sternum and below the clavicles. The feeling was described in some cases (5) as of distressing tightness, and in one case as a 'spasm.' Tenderness on pressure over the seat of pain was very frequently observed. The persistence of the pain was very varied.

3. *Difficulty of breathing* was a noticeable symptom in several cases. In 13 it was recorded as being specially so; in 4 it had all the characters of spasmodic asthma, occurring at intervals and especially during the night.

4. *Difficulty of swallowing* was noticed in 10 cases; in one of these the difficulty was remarked especially in swallowing liquids.

5. *Hæmoptysis* was present in 10 cases. The amount of blood varied in these cases from marked streaks to copious expectoration, lasting two or three days. No case was recorded as presenting this symptom except on tolerably clear proof that it depended on bronchial gland enlargement, and on no other cause.

6. *Congestion* and puffiness of the face have been mentioned as present in 3 cases.

7. *Expectoration of mucus*, such as results from bronchial catarrh, was frequently present. Copious expectoration of pus was present in 3 cases. In each it resembled the contents of an ordinary glandular abscess, but mixed with air. In one of these the discharge was intermittent. The frequent occurrence of cough without any expectoration was remarked in many cases. Calcareous particles are mentioned also as having been expectorated.

8. *Loss of voice* (4 cases) and hoarseness (2 cases) are recorded as striking symptoms.

9. *Vomiting* is mentioned as having been present twice.

10. Lastly, the *position* assumed with least discomfort by the patient when in bed was noticed in 41 cases. Of these 23 rested on

that side on which the glands were mentioned as being chiefly if not wholly affected. In 15 cases an opposite condition was noticed. In 2 cases the most comfortable position was lying on the back. One patient, unable to lie down, sat when in bed, and stooped forward. One patient, a little boy, could only rest on his face and knees. This case was further remarkable in reference to the clearness with which the disease was recognised, and the successful result of subsequent treatment.

It might be mentioned here incidentally that the glands of the right side were noticed as being chiefly affected in 28 cases, and of the left in 22 cases, in 4 both sides seemed equally affected, and in 4 no record was made. The general or constitutional symptoms connected with the malady under notice need not be discussed here. They are in nowise peculiar, and will be found treated of elsewhere. The symptoms described above have special reference to the bronchial glands. The cough and its peculiar characteristics are, no doubt, in a great measure dependent on pressure or on irritation communicated to the pneumogastric nerves and their branches. So likewise pain and difficulty of breathing, in a great degree, through direct pressure on the air-passages, may also cause or aggravate these symptoms. Aponia especially seems to have relation to the condition of the recurrent nerves. In one of the cases, which the writer saw with Mr. Lennox Browne, paralysis of the left chorda vocalis existed. The diagnosis of glandular disease was clear, a conclusion confirmed by the results of treatment. Vomiting is mentioned in two cases. M. de Mussy wrote that this is a more frequent result when the left pneumogastric nerve is pressed upon. He saw a connexion between the troublesome vomiting which occurs in some cases of tubercular disease of the lungs and like pressure upon nerves. The puffiness of the face and eyes noticed in these cases is due to pressure on the venous trunks, a condition which also accounts not only for some cases of hæmoptysis, but for bleeding from the nose, occasionally present. Copious and sometimes persistent hæmoptysis (profuse, as in the instance previously mentioned) has been traced to the perforation of a vessel by ulceration in connexion with disease of the glands.

**PHYSICAL SIGNS.**—The physical signs noticed in the 58 cases referred to were: 1. *Dulness*. This was present in 47 cases. It was found between the margin of the scapula and the spinal column at one or both sides, on a level with the fourth and fifth dorsal vertebræ. It varied in degree, and was more readily manifested when the muscles of the back were strained by folding the arms across the chest, and was often strikingly distinct when one side was contrasted with the other. Dulness was present in front in 8 cases (whether coinci-

dentally with dulness at the back or not is not clearly stated), beneath the top of the sternum, and at each side below the sterno-clavicular junction. The dulness here was best elicited by the patient expiring deeply and holding the head backwards whilst percussion was being made.

2. *Flattening* of the affected side in front was mentioned in 3 cases. *Diminished mobility* of the affected side, chiefly independent of flattening, was recorded in 4 cases. Prominence in front was not recorded in any case, though, no doubt, it may occur.

3. The *respiratory sounds* were variously modified. Marked tubular breathing was recorded as being present over the seat of disease in 14 cases. In 10 the expiratory murmur was described as being very loud, various modifications of the inspiratory murmur being found at the same time. Feebleness of the respiratory murmur as a whole was noticed in 14 cases. In some this deficiency extended over the whole lung; in others it existed over the upper or lower portion of a lung, behind or in front. The observations made on the voice by the writer were few, but M. de Mussy and M. Lereboullet speak of a peculiar and increased reverberation of both the voice and the cough. Dr. Eustace Smith has described in the cases of children a venous hum, heard at the root of the neck when the head is thrown back, caused by the pressure of the enlarged glands on the venous trunks.

**DIAGNOSIS.**—In the present article those cases are not kept in view in which the bronchial glands, becoming the seat of constitutional disease in association with other glands in the neighbourhood, constitute large and manifest tumours—such will be found described under another head (*see* MEDIASTINUM, Diseases of). Nor is it intended to give prominent consideration to the state of the glands when they enlarge in acute disease—where the state of the glands plays a secondary part. The writer has been anxious to describe and to assist in recognising the presence of a condition in which the disease of the bronchial glands constitutes to some extent a disease *per se*, or gives rise to complications which it is important to discriminate. He believes the symptoms and signs above described will suffice for the purpose, always remembering that in the present and in all similar instances it is necessary to take means for excluding in our investigations diseases which may produce like phenomena. Thus we may find cough, pain, tenderness on pressure, and aponia in a case of hysteria without any evident structural disease. On the other hand, a small tumour—say, a small aneurysm—may produce all the signs of pressure which are above given as the signs of bronchial gland-enlargement. It is the duty of the physician to recognise these differences and

distinctions, to trace them to their origin, and thus establish as far as may be a knowledge of the nature of the disease under investigation.

**PROGNOSIS.**—Prognosis will, as in like instances, so entirely depend on the nature of the disease, on its amount and its condition, on its relation to and effects on surrounding organs and textures, that each case must be regarded independently. It would be impossible to discuss the subject fully here—all that can be said is that the simple enlargements, or those resulting from specific diseases, generally yield to treatment, and within a reasonable period.

**TREATMENT.**—In several cases of morbid gland-enlargement, treatment has proved very effective. These cases would seem to be those of simple chronic enlargement. Many such cases have yielded to the use of iodide of iron in the form of pills or syrup, and to the external application between the shoulders of a solution of iodine, composed of equal parts of the tincture and the liniment of iodine. The same treatment has likewise proved very effective in cases in which a syphilitic origin for the disease could be traced. Symptoms such as cough, difficulty of breathing, pain, and loss of flesh and strength, &c., will all require more or less suitable treatment. The cough and difficulty of breathing may in some cases be relieved by simple expectorants or antispasmodics. A useful application when pain is a prominent symptom, is an embrocation composed of equal parts of chloroform, belladonna liniment, laudanum, and spirit of camphor. A couple of drachms of this composition sprinkled on the surface of impermeable piline, and applied over the painful part for a few minutes, often affords relief. Hypodermic injection of morphine may be required when pain is very severe. Under all circumstances it is necessary to improve the general health by wholesome diet, pure air, and the other conditions which will promote good digestion and a free elimination by the excreting organs.

RICHARD QUAIN.

**BRONCHOCELE** (*βρόγχος*, the throat; and *κήλη*, a tumour).—A synonym for goitre. See GOITRE.

**BRONCHOPHONY** (*βρόγχος*, the throat; and *φωνή*, the voice).—The resonance of the voice, as normally heard on auscultation over those parts of the chest which correspond with the main bronchi, and, in certain morbid conditions, beyond these situations. See PHYSICAL EXAMINATION.

**BRONCHO-PNEUMONIA.**—A synonym for catarrhal pneumonia. See LUNGS, Inflammation of.

**BRONZED SKIN.**—A peculiar discoloration of the skin, frequently associated

with Addison's disease. See ADDISON'S DISEASE.

**BROW-AGUE.**—A synonym for frontal neuralgia, or *tic-douloureux*. See MALARIA; NEURALGIA; and TIC-DOULOUREUX.

**BRUIT** (*bruit*, Fr. a noise).—A word used to designate various abnormal sounds heard on auscultation, in connexion with the heart or vascular system. See PHYSICAL EXAMINATION.

**BUBO** (*Βουβών*, the groin).—SYNON.: *Apostemaignuinis*; *dragoncelus*; Fr. *bubon*; Ger. *Leistenbeule*.—An inflammatory swelling of a lymphatic gland in any part of the body, whether caused by irritation or absorption, as in venereal disease; or referable to some constitutional affection, such as the plague. See BUBO, VENEREAL; and PLAGUE.

**BUBO, VENEREAL.**—DEFINITION.—An affection mentioned in the most ancient medical writings, but not properly distinguished in its several varieties until the present century. The term *bubo* is almost exclusively confined to swelling of the glands of the groin consequent on venereal irritation of the genitals, and will be so employed here.

VARIETIES.—Buboes are divided into:—  
1. *Simple bubo*, known also as *sympathetic bubo*, due to inflammation of a gland through ordinary irritation from an inflamed surface.  
2. *Specific bubo*, the *chancreous* or *virulent bubo*, or an abscess inoculated with the pus of a chancre.  
3. The indolent enlargement of the lymphatic glands accompanying the development of the initial sore of syphilis is, by some, called *syphilitic bubo*, though its formation is not due to active inflammation, and is quite distinct from the two forms just mentioned.

The glands first affected in the sympathetic and in specific bubo are always those in most direct communication with the sore. Further, according to Ricord, when chancreous matter has reached a gland through the absorbent ducts, it never passes beyond that gland into another more remote in the series. On the other hand, the effect of syphilitic absorption is general, and affects the glands of the group which are remote as completely as it does those which are nearest to the inlet of the poison.

Buboes occur most frequently on the side of the body occupied by the source of irritation; still they form not rarely on both sides, and sometimes only on the side opposite to the position of the exciting sore. When bubo forms in both groins, there is usually a sore on each side or one at the mesial line. The crossing of the irritant is due to anastomosis or interlacing of the lymphatic ducts at the mesial line. And when the glandular affection forms on the side opposite to the sore, there is want of communication with the glands of the groin on that side. Bubo, in-

cluding simple and virulent, occurs in about thirty per cent. of chancres; how often with urethritis and other lesser venereal affections is not known.

### 1. Simple Bubo.—*Adenitis.*

**ÆTIOLOGY.**—This variety may be caused by mechanical irritation of the surface, such as erosions or fissures irritated by dirt or by caustic applications; by urethritis, balanoposthitis, or by a chancre when that acts only as a simple local irritant. Simple bubo is also often excited by herpes, erysipelas, boils, or other non-venereal irritants of the genitals. Lastly, though nearly all buboes can be traced to a lesion of the part where their absorbents arise, there are occasionally some observed for which such origin cannot be found. Some are caused by excessive sexual intercourse or excitement; they are met with generally among young lads and girls. Such are analogous to the inflammation of the inguinal glands which sometimes follows excessive straining of the muscles in long walks or rides. But it is also believed by one or two authorities that chancrous poison in rare instances has been absorbed along the lymphatic ducts without producing a sore on the surface. This may be, though extremely rarely, an actual occurrence. It has received the names of *idiopathic bubo* and *bubon d'emblée*.

**SYMPTOMS.**—There are two forms of simple bubo:—1. Slight swelling and tenderness of one or more glands, ending in a few days by resolution. This occurs most frequently in urethritis, balanoposthitis, or simple chafings. In genuine chancre the next and more serious form is most frequent. 2. Swelling, commonly of one, seldom of several glands; branny thickening of the surrounding tissues; redness of the integuments; great tenderness and pain, especially when walking. In a few days this condition terminates either by abscess, the most frequent culmination, by gradual subsidence to the normal state, or by chronic induration of one or more glands. When the bubo suppurates, the matter, instead of pointing at once, may burrow in various directions among the layers of cellular tissue before it breaks through the skin. The pus of this form of bubo is always the ordinary pus of inflammation.

Some cases may terminate by gradual conversion into chronic fistulæ; or the glands may degenerate by tuberculisation with successive formations of abscesses around the tuberculous glands, with undermining of the skin.

**PROGNOSIS.**—This form of bubo is seldom dangerous, but always serious when it causes tuberculisation of the glands and burrowing; it is then often very tedious and exhausting to the patient.

2. **Specific Bubo.**—*Virulent bubo.* This is in reality an enormous chancre, identical

in all but size with the sore whence it was inoculated. This form is never a consequence of gonorrhœa or of constitutional syphilis, but only of the local sore. It may be generated in two ways. 1. By touching the surface of a simple open bubo with chancrous pus. 2. By the absorption of chancrous pus along the lymphatic ducts leading from the chancre to the gland most directly connected with the sore. Two pathological facts prove the reality of this mode of origin. First, it has been observed that occasionally small circumscribed abscesses will form in the course of the lymphatic ducts before they reach the gland. When these little abscesses are opened they present the peculiar characters of the chancre. Secondly, when an efferent duct has brought chancrous pus into a gland, the abscess around the gland retains the ordinary phlegmonous character until the necrosing action going on in the infected gland lays open its interior, then contamination of the abscess takes place, and in a few days the abscess-cavity assumes the aspect of a huge chancre.

The conditions which assist or hinder the conveyance of chancrous pus along the lymphatic vessels are wholly unknown.

The proportional frequency of virulent bubo to the number of chancres is not known. It is far less frequent than simple bubo. It is said that women are less prone to it than men.

When not accidentally inoculated, virulent buboes are almost wholly confined to the groin, and originate in the gland which lies commonly in the centre of the group over the great blood-vessels. Occasionally, when the chancre is on the finger, a gland of the epitrochlear or axillary groups develops into virulent bubo. Hübbenet, of Kiev, experimentally inoculated a soft chancre on the cheek, which was followed by virulent bubo of the gland in front of the tragus of the ear. This bubo is generally unilateral; rarely are both sides of the body affected; in such cases the chancre is at the mesial line, or there are two chancres. Still more rarely the bubo forms at the side of the body opposite to that of the chancre. The time for appearance is generally in the first or second week of the existence of the chancre; but it may occur at any time while the chancre is spreading.

**SYMPTOMS.**—At first the symptoms of specific bubo are those of acute abscess forming rapidly round a single gland. Thus far they differ nowise from those of simple bubo. When the contagious pus reaches the abscess accidentally from without, or from within the gland by ulceration or incision of its capsule, the simple suppurating cavity becomes a spreading ulcer, which rapidly makes itself widely open by destruction of the integuments. The skin, thin as tissue-paper, gives way at several points, and lets

out a large amount of matter, which is thin, yellowish-grey or yellowish-red, with shreds of a chocolate colour floating in it. When perforated, the skin breaks rapidly away until the cavity is widely exposed.

The further progress varies. In the least severe variety the edges of the skin ulcerate irregularly for a short distance, then thicken and begin to granulate; the floor of the ulcer loses its unevenness and rises up to the level of the skin; and cicatrisation follows. But commonly a much greater destruction of tissue is effected. The skin is eaten away into wide gaps; the floor burrows under the skin in long channels, or dives deeply among the great vessels. In other cases the ulceration produces a large shallow sore. The contagious condition of these buboes often continues for weeks or months, so that consecutive inoculation of the skin is not infrequent. The characters of the fully formed virulent bubo are those of the chancre. The surface is greyish-brown, dug-out, 'worm-eaten'; the borders at some parts are thickened, everted, and pared away, at others sharply eroded or undermined and curled in. The matter is thin, shreddy, plentiful, reddish in colour, and inoculable. Again like the chancre, the virulent bubo passes through periods of *extension*, *stagnation*, and *repair*. The virulent, as well as the simple bubo, is liable to erysipelas and inflammatory sloughing.

The *duration* of virulent bubo for the reason stated is wholly indefinite—in a few cases ending in a week, in many cases lasting for months.

**DIAGNOSIS.**—The virulent bubo has in its early stage no distinguishing mark from the simple acute bubo. After it has become inoculated with the contagious matter it is distinguished from every other affection by the characters already described. The earliest signs of virulence are two: the matter which escapes when the bubo is lanced is thin and shreddy, and the edges of the cut become in a day or two eroded and partly covered by adherent sloughs. Subsequently other characters develop and remove all doubt. Nevertheless, in some cases the signs of virulence are so feebly marked throughout that the diagnosis remains uncertain. This difficulty may be increased by the primary sore having healed before the suppuration of the bubo, or by its being hidden in some unusual position (urethra, anus).

**PROGNOSIS.**—This is not always grave. Virulent buboes often reach cicatrisation in a few weeks, and meanwhile cause no serious inconvenience. On the other hand, they are prone to inflammation; or if the patient have suffered from syphilis some years previously, the form of tertiary disease known as phagedæna may attack the ulcers, and thereby permanently cripple the patient, or even destroy life.

**3. Syphilitic Bubo.**—*Indolent multiple bubo; pléiade ganglionnaire.*—This enlargement of the group of lymphatic glands in nearest connexion with the initial sore is the constant, or at least almost constant, symptom of early syphilitic infection. In this it differs much from the bubo of chancre or gonorrhœa, in which affections the bubo is more often wanting than present. Four conditions have been noted to exist in patients in whom the enlargement of the glands could not be detected, namely:—1. Phagedæna of the sore. 2. Obesity; in such persons the lymphatic system as a rule is small. 3. Second infection; *i.e.* a primary sore on a patient who has had constitutional disease before. 4. Scantiness of the induration of the primary sore, the sore itself being ill-developed. But these exceptions are most rare. Fournier gives five instances only of absence of indolent multiple bubo in 265 cases of hard sore, accompanied by well-marked general syphilis; or 2 per cent. In 176 similar cases noted by the writer, three had no apparent inguinal enlargement. One of the patients was a very fat man, and in another the initial sore was only slightly hardened.

The seat of this bubo is, in the great majority of cases, the groin, whither, besides the lymphatic ducts of the external genitals, those of the anus, perinæum, buttocks, and lower part of the abdomen are directed. But the glands of other regions—epitrochlear, axillary, sub-maxillary, buccal, cervical, and nuchal—are all occasionally found primarily enlarged when the syphilitic poison enters the body through the regions whence their absorbent ducts are derived.

In the groin the bubo is generally *double*, that is, the glands are enlarged in both groins, those on the side of the sore being most affected. In a few cases only the glands situated on the same side of the body as that of the sore enlarge, and in rare examples only those of the side opposite to that of the sore. This enlargement affects the group widely, for when the deeply-placed glands can be examined they are found to be similarly enlarged along the iliac vessels and lumbar vertebrae.

The enlargement of the glands is first perceived about eleven days after the induration of the sore, though probably the affection commences at the end of the incubation of the poison. In extremely rare instances enlargement is delayed until the third or fourth week after the induration of the sore.

**SYMPTOMS.**—The distinguishing marks of this bubo are swelling, not inflammatory, and rarely surpassing an almond or a hazel nut in size; gritty hardness; ready isolation and mobility; insensibility to pressure; natural hue and condition of the overlying skin; and absence of fulness of the cellular tissue.

The gland in most direct communication

with the sore is most enlarged; in rare exceptions only a single gland is enlarged. Copious enlargement of the glands does not always attend copious induration of the sore, nor is scanty induration of the sore always accompanied by small swelling of the glands, though commonly this is the case. So also, though absence of tenderness is the rule, the glands may be slightly painful if pressed. Again, though the glands remain distinct in most cases, they have been known to coalesce into a single mass, which becomes fixed to the fascia. In many patients the dorsal lymphatic vessel of the penis becomes indurated sufficiently to be easily distinguished under the skin.

This bubo reaches full development in one or two weeks, and remains without apparent change for several weeks, or even for two or three months. Then it begins to diminish slowly, but is generally still evident in the fifth or sixth month after infection, and now and then even for years afterwards. In rare cases the enlargement vanishes in two or three weeks. The long duration of enlargement renders this bubo a valuable sign of constitutional syphilis when the primary sore has disappeared. Again, the conspicuous enlargement of a group of glands may indicate the place of entry of the syphilitic poison when that is hidden.

**TERMINATIONS.**—In most cases the glands revert to their natural state. Sometimes they degenerate into masses of fibrous tissue. Now and then suppuration takes place, not from the syphilitic change, but from ordinary irritation, and then it produces a simple phlegmonous abscess. A further change is apt to occur in tuberculous persons. The glands enlarge still more, grow soft, and coalesce; presently matter forms in the thickened cellular tissue around the glands, and the further progress becomes that of tubercle.

**DIAGNOSIS.**—This is generally easily drawn from the character of the swelling, aided by the presence of other syphilitic signs (hard sore, rash on the skin, &c.). This bubo may be confounded with chronic inflammatory enlargement, but in such cases the history and attendant symptoms remove doubt.

**PROGNOSIS.**—Apart from its connexion with syphilis, the prognosis is good. The only untoward termination is tuberculous degeneration.

**TREATMENT.**—1. *Simple acute bubo*, from whatever cause it may arise, demands the treatment of acute abscess. In the early stages, antiphlogistics, rest in bed, the constant application of a cream made of equal parts of extract of belladonna and glycerine, warm poultices, fomentations, and baths, sometimes even leeches, are requisite. Caution must be observed in applying leeches if chancre be present, lest the bites be converted by inoculation into chancres. The leeches should be applied at the cir-

cumference of the swelling, so that they may be removed as far as possible from the centre where pointing is most probable. If suppuration arrive more speedily than was expected, and the bubo prove a virulent one, it is best to open it freely and trust to antiseptics, such as iodoform, to prevent the inoculation of the bites.

When active congestion has ceased and matter has not formed, the subsidence of the glands may be aided by pressure with a pad and spica bandage. Stimulating ointments and plasters, iodine, and similar preparations are of doubtful service, and may possibly rekindle the inflammation. When pus has formed, it should be let out by a vertical incision at once. The proper drainage of the cavity should be ensured by making the incision long ( $\frac{3}{4}$  in. usually), and by placing a bit of drainage-tube between the edges of the incision during the first twenty-four hours. Early evacuation checks the undermining of the skin and prevents burrowing. When matter forms at several points, a small incision should be made at each fluctuating point. When free exit has been given to the pus, the groins should be dressed with boracic lint soaked in hot water; and when swelling is gone, the surfaces may be covered with boric acid ointment and overlaid by a thick compress and spica bandage; which, slackly applied at first, may be tightened as the swelling subsides.

2. The *chancreous bubo*, not being distinguishable from ordinary sympathetic bubo during the inflammatory swelling and consequent suppuration, requires the same treatment—sedatives and an early single incision. By early incision the cavity of the abscess, subsequently the chancreous ulcer, is kept as small as possible; by a single incision the number of chancres is restricted. Occasionally, but only when the abscess has already undermined the skin, more than one incision is necessary. Caustics have no advantage over the knife for opening the abscess, while they make a larger gap. When the abscess has been opened and the chancreous nature is suspected, a free cut should be made, and the cavity should be well cleared at the time by injections—of one part of carbolic acid to sixty of water, for example, or of 5–10 grains to the ounce of nitrate of silver, or of tartrated iron, or of some other astringent and disinfectant, and the interior well dusted with iodoform. The injection may be repeated three times in the first twenty-four hours; and constant drainage maintained by a drainage-tube and a compress of Lister's antiseptic gauze or boracic lint. Should these precautions fail to prevent the conversion of the abscess into a chancre, it must then be dressed as a chancre. It must be sedulously washed by injection twice daily, dried by careful mopping with bits of cotton-wool, and well dusted and packed with iodoform in

powder; the whole cavity being loosely filled with pellets of cotton-wool, and compressed gently with a layer of boracic lint and a bandage. Should this fail, as it sometimes will, caustic may be applied. The caustics most suitable are the strongest and most penetrating, such as Vienna paste, acid nitrate of mercury, Ricord's paste (powdered charcoal and the strongest oil of vitriol). To apply the caustic thoroughly the patient should be anaesthetised. If the least part of the surface is left undestroyed, that will re-inoculate the whole. Before cauterising, it is well to scrape the surface thoroughly with Volkmann's sharp spoon. Complete cauterisation is practically very difficult to accomplish, hence caustics should be reserved for the cases where iodoform, which is painless, fails. Overlapping bands of skin should be divided, that the dressing may be fairly applied.

*Phagedæna*.—When the ulceration by its obstinacy deserves this title, plan after plan of treatment must be tried till the destruction of tissue is arrested—caustics being reserved for the last. To the local treatment of phagedæna that for syphilis should be added. The patient should be brought under the influence of mercury as quickly as possible, by giving two or even three grains of blue pill thrice daily, or by the injection into the thick muscles of the trunk of some suitable mercurial solution, such as that of Raggazoni (bichloride of mercury, 1 part; iodide of sodium, 1 part; distilled water, 64 parts: 8 minims being a dose). When the patient's general health is good, the ulceration seldom fails to yield to iodoform, applied thoroughly in the manner directed. While the nocturnal gnawing pain continues, the patient should be narcotised with opium or other narcotic. The cessation of pain is a signal that the eroding action has stopped. When iodoform alone is insufficient, the continuous warm hip-bath must be employed. By it pain is at once arrested and healing soon set in motion. As continual immersion in a bath becomes extremely irksome in many cases, the ulceration may be arrested by keeping the patient in water for sixteen hours every day, and allowing the night to be passed in bed—in this interval the wound should be carefully packed with iodoform. When the ulceration is stopped, the bath may be discontinued and iodoform alone used. If the water-bath fail (and such a result has not yet happened during a considerable employment of this method at the Male Lock Hospital), caustics may then be used.

The strength of the patient, generally much exhausted, should be restored by tonics, good diet, stimulants, and other general means.

3. The *syphilitic bubo* usually causes no trouble, and gets well during the course of treatment which is carried out for the cure of the general disease. If tenderness or aching

occur, rest and a few warm baths are sufficient. If suppuration takes place the abscess must be incised and poulticed. The tuberculous degeneration is best met by remedies against the diathesis—iodide of ammonium or of iron, cod-liver oil, nutritious food, sea-air, and other tonics. Mercury, in  $\frac{1}{10}$ -grain doses of the bichloride, combined with the iodide of sodium or with solution of perchloride of iron, may be added to the treatment. Locally, the abscesses and sinuses must be emptied as fast as they form and cleared by dissecting the diseased glands away and scraping their sites with the sharp spoon.

*Abortive treatment of bubo*.—This once favourite method of treating buboes has fallen into disuse as the varieties of bubo have been better understood. The chronically enlarged glands of syphilis have little or no tendency to suppurate, but subside spontaneously if let alone. Others only suppurate when freely irritated, hence the best abortive treatment is to guard against the increase of irritation, and assuage that which exists by antiphlogistics. In the bubo virulent by absorption suppuration is inevitable. Thus, the sympathetic bubo is the only one which can be acted on by abortive treatment. To antiphlogistics may be added counter-irritants, but these are uncertain in their effect. Those least open to objection are vesicants, and the form most beneficial is repeated 'flying' blisters. By this means a series of small blisters are produced round about, not over the swelling. Any other plan is most uncertain—painting the part with tincture of iodine, or other mild irritant, is simply waste of time.

Compression is of great value for reducing indolent enlargement, or for removing the boggy condition of the groin where several abscesses have formed, with more or less undermining of the skin. It is useless for syphilitic bubo and mischievous for the virulent bubo. The simplest and most effectual method of applying compression is by a graduated compress of lint kept firmly in place by a spica bandage of calico, or of elastic tape. For abscesses, careful packing may be added to the compress. Each focus of pus must be laid open, and bridges or tunnels cut across, so that narrow strips of lint soaked in liquor plumbi subacetatis and lightly pressed between the fingers, to drive out the dripping excess of fluid, may be laid into the hollows and under overhanging borders of skin. The strips must be renewed every day at first, and the cavities well cleared by syringing with water. In a few days, when the discharge is very small, the strips may be left for three or four days unchanged. The first application is painful, but very soon an insensible crust is formed over the ulcerating surface, and fresh applications cause no discomfort. As soon as healthy granulations are formed the plugging should be laid aside.

If the glands do not shrink in size after a fair trial of the methods already described, the patient should be anaesthetised, the glands scraped away by the sharp spoon, and the wound well spread with iodoform.

BERKELEY HILL.

**BULB.**—A synonym for the globe of the eye, and also for the medulla oblongata. Its adjective, **BULBAR**, is commonly restricted in application to the latter, and has for a considerable time been commonly thus employed, as in the terms 'bulbar symptoms,' 'bulbar nerves,' and the like. This use of the adjective has led to a more extensive employment of 'bulb' for the medulla oblongata, because the term 'medulla' is also applied to the spinal cord, the 'medulla spinalis,' as well as in its general sense of 'marrow.'

**BULBAR PARALYSIS.**—A synonym for labio-glosso-laryngeal paralysis; derived from the pathological relation of the disease with the bulb or medulla oblongata. See LABIO-GLOSSO-LARYNGEAL PARALYSIS.

**BULIMIA** (*βov*, a prefix signifying excess; and *λιμός*, hunger).—Excessive or voracious appetite. See APPETITE, Disorders of.

**BULLA** (*bulla*, a bubble).—See BLISTER.

**BURGHERSDORP**, in Cape Colony. See AFRICA, SOUTH.

**BURNS.**—The morbid local effects produced by the direct application of excessive dry heat. See HEAT, Effects of Severe or Extreme.

**BURSÆ MUCOSÆ, Diseases of.**—Bursæ mucosæ are spaces in the connective tissue lubricated with a small amount of serous fluid, and situated at points exposed to repeated pressure or friction. Structurally they are composed of a layer of condensed cellular tissue, fusing externally with the areolar tissue of the part, and lined internally with an imperfect layer of flattened endothelial cells, similar to those found in the pleura or peritoneum. Some bursæ, as that over the patella, that under the deltoid, those about the great trochanter, and many others, are constantly present; but new bursæ, equally perfect in their structure, may form at any part exposed to abnormal pressure and friction, as over the outer malleolus of a tailor, under an old corn, or over the head of the metacarpal bone of the great toe (*bunion*). Like the great serous cavities, bursæ are in direct communication with the lymphatics, and inflammatory products are consequently absorbed from them with great readiness, often giving rise locally to a diffuse inflammation of the surrounding cellular tissue, closely resembling phlegmonous erysipelas in appearance, and always accompanied by high fever.

Bursæ are liable to four kinds of disease:—

1. **Acute inflammation and suppura-**

**tion—Acute bursitis.**—This is usually the result of some more or less violent mechanical injury. It may occur in any bursa, but is most common in the bursa patellæ, those about the hip and over the olecranon, and in the small false bursa formed beneath an old corn, or in a bunion. The symptoms are those of acute inflammation generally, but the redness and swelling often extend a remarkable distance up and down the limb. Thus, a drop of pus beneath a corn may cause œdema and redness to the knee. The febrile disturbance is usually proportionately severe. About the trochanter the abscess may assume a chronic form.

**TREATMENT.**—Hot fomentations, and the application of glycerine and extract of belladonna (equal parts), may be of use. It is very important that pus shall be let out early, or it may burrow extensively, especially about the knee. Antiseptic treatment will be found especially useful in the treatment of suppurating bursæ.

2. **Chronic Bursitis—Dropsy of the bursa.**—This consists of an accumulation of serous fluid distending a bursa more or less tensely. The wall becomes somewhat thickened and opaque, but is otherwise unchanged. The fluid is clear, straw-coloured, and albuminous in character. The cause of the disease is usually repeated slight mechanical injury, but in some cases it may be due to some of those obscure conditions spoken of as 'rheumatism.' The symptoms are merely those of a collection of fluid in the situation of the bursa, perhaps accompanied by a feeling of weakness in the neighbouring joint. There is no pain or tenderness. The commonest form of this disease is the so-called 'housemaid's knee,' but it is not infrequent in the bursa of the popliteal space.

**TREATMENT.**—Avoidance of the mechanical injury, whatever it may be, which has caused the disease, is most important. The swelling may be painted with tincture of iodine twice a day for some weeks, or a series of small blisters applied over it. If this fails, an incision may be made into the swelling under antiseptic precautions, and a small drainage-tube introduced for a week or ten days. But before either of these means is adopted, care must be taken to ascertain that the bursa does not communicate with the neighbouring joint. In the ham it is safer as a rule not to operate, except by means of the aspirator.

3. **Chronic enlargement of the bursa, with fibroid thickening of its walls.**—This affection is most common in the bursa patellæ, but may occur in that situated over the tuberosity of the ischium. The bursa becomes converted into a dense fibroid mass of almost cartilaginous hardness. On section it is found to be composed of concentric layers of dense fibroid tissue. There is usually a small central cavity containing a little fluid. The cause of this change, as of simple dropsy,

is repeated mechanical injury of a slight nature. In some cases it seems to be connected with syphilis. The only treatment is removal by the knife.

4. **Chronic enlargement of the bursa, with the presence in it of the so-called melon-seed bodies.**—In this form of disease, in addition to some thickening of the wall and accumulation of fluid in the bursa, small oval, flattish, smooth bodies of a white colour are found floating freely in its interior. These are similar in nature to those found in some forms of ganglion (*see* GANGLION). This condition is recognised by the peculiar soft crackling feeling perceived on palpation, com-

bined with the ordinary symptoms of an enlarged bursa.

**TREATMENT.**—The affected bursa may be dissected out; or it may be laid open, emptied, and drained. The closure of the cavity may be insured by the application of pure carbolic acid, so as to destroy the lining membrane. MARCUS BECK.

**BUXTON, in Derbyshire.**—Simple thermal waters. *See* MINERAL WATERS.

**BUZIAS, in Hungary.**—Strong muriated chalybeate springs. *See* MINERAL WATERS.

## C

**CACHEXIA** (*κακός*, bad; and *ἕξις*, a habit or constitution of body).—**SYNON.**: Fr. *Cachexie*; Ger. *Kachexie*.

**DEFINITION.**—A chronic state of ill-health associated with impoverished or depraved blood, arising from malnutrition, persistent loss of blood-elements, or the presence of a morbid agent.

In such diseases as tabes mesenterica, lymphadenoma, and the like, the patients become cachectic from direct depravation of the blood, in consequence of disease of organs which are importantly concerned in its nutrition and elaboration. In other diseases persistent loss of blood-elements arising from chronic suppuration or from repeated hæmorrhages (especially observed in some uterine conditions) induces a cachexia which is well described by the term *secondary anæmia*. Again, certain poisons introduced from without, and nurtured within the body, may produce marked cachexia. The syphilitic and malarial poisons are good examples of the first kind. Of cachexiæ produced by the presence of morbid agents which have been generated within the body we may instance those arising from defective elimination, as the uræmic and gouty cachexiæ from inadequate functional activity of the kidneys and liver. In the latter connexion it should be noted that, as has been hinted by Sir James Paget in reference to cancer, the malignant cachexia may arise from blood-contamination with the waste products yielded by the morbid growth in the process of its nutrition; such a growth not only abstracting material from the blood for its nutrition, but also contributing effete material to it.

It must be further observed that in the opinion of some pathologists the term cachexia implies much more than the secondary anæmia consequent upon the ravages of a given local or general disease upon the system. It rather signifies the

morbid constitution which is the disease, and which may precede its local manifestation. Thus we may have the cancerous cachexia, culminating in scirrhus of the breast, the tubercular cachexia in pulmonary tuberculosis, and the like. It is thus evident that with many observers the terms *cachexia* and *constitutional disease* have the same significance.

R. DOUGLAS POWELL.

**CACOPLASTIC** (*κακός*, bad; and *πλάσσω*, I mould or form).—A term applied to products of inflammation which are more or less incapable of organisation.

**CADAVERIC** (*cadaver*, a dead body).—This word signifies 'belonging to the dead body'; and it is applied to the aspect, colour, odour, and other phenomena resembling those of death, which are sometimes observed in the living subject.

**CÆCUM, Diseases of.**—**SYNON.**: Fr. *Maladies du Cæcum*; Ger. *Blinddarmkrankheiten*.—The structural peculiarities and anatomical relations of the cæcum are specially favourable to the occurrence of the diseases to which this part of the large intestine is most liable, viz. (1) *accumulation of solids* (feces, foreign bodies, &c.); (2) *ulcerative inflammation*, typhlitis, &c.; and (3) *dilatation, contraction, and perforation*.

1. **Accumulations.**—Hardened feces, biliary and intestinal concretions, foreign bodies, stones of fruit, balls of worms, and also gases resulting from decomposition, are apt to collect in the cæcum, and cause varying degrees of local disturbance. Sometimes, as in elderly patients of torpid habit, the cæcum is found loaded with feces, without inducing pain or other signs of inflammation. The right iliac region may be full and hard, and in it may be felt a well-defined, almost painless, doughy uneven mass, of the shape of the cæcum. As a rule, however,

sooner or later the accumulation leads to one or more of the following results:—

(a) *Obstruction of the bowels.* This may be partial, as in the various degrees of constipation, or complete. When complete, it may even prove fatal—*e.g.* from perforation or rupture, without the cæcum or peritoneum exhibiting signs of inflammation. On the other hand, general peritonitis supervening, obstruction in the cæcum may be quickly obscured; still, however, the chief pain and tenderness may be found in the right iliac region. (b) *Pressure* on adjacent nerves, vessels, or other structures, producing numbness and œdema of the right leg, retraction of the right testicle and other symptoms. (c) *Inflammation and Ulceration*: stercoral ulcer, cæcitis, typhlitis, peritonitis.

It is of clinical importance to bear in mind that the cæcum, when overloaded or enlarged, may occupy an unusual position, *e.g.* a site between the right and left iliac regions, or may descend somewhat into the pelvis and press on the urinary bladder.

Tympanitic distension of the cæcum is generally associated with some fecal accumulation or obstruction in the colon or other part of the large intestine. See FÆCES, Retention of; and FLATULENCE.

**2. Inflammation.**—SYNON.: Cæcitis.—Until recent years inflammation of the walls of the cæcum has been designated by the term, Typhlitis; but this is no longer applicable, for it has been shown that the symptoms of what is clinically recognised as typhlitis do not depend on inflammation of the cæcum, but on inflammation of the peritoneum investing the cæcum and vermiform appendix (see TYPHLITIS). The mucous membrane of the cæcum is liable to catarrhal inflammation, from the lodgment of feces, indigestible matters, and foreign bodies; but the symptoms induced by it are, as a rule, ill-defined, and generally resemble those of colitis (see COLON, Diseases of). It may, however, lead to ulceration, which may ultimately involve the outer wall of the cæcum and the peritoneum, and thus induce typhlitis.

**3. Dilatation, Contraction, and Perforation.**—The cæcum may be (1) *dilated*, from accumulation within it of solids and gases, or from obstruction in the colon; (2) *contracted*, from growths (polypi, cystic tumour, cancer, &c.), or cicatrisation of ulcers (tubercular, dysenteric); or (3) *perforated*, from ulceration (stercoral, enteric, tubercular, cancerous, dysenteric), from tearing of the wall by over-distension, or from a typhlitic abscess (originating in the appendix vermiformis) opening into it.

GEORGE OLIVER.

#### CALCAREOUS DEGENERATION.

A form of degeneration characterised by the deposit in the tissues of earthy salts, especially those of lime. See DEGENERATION.

**CALCULI** (*calx*, chalk).—DEFINITION. The term calculus is now applied to any kind of concretion formed in the ducts or passages of glandular organs; though older writers limited its employment to the designation of concretions met with in the kidneys and urinary bladder.

VARIETIES.—The following is a concise summary of the principal calculous concretions met with in the human body, given in their alphabetical order.

**1. Biliary Calculi.**—Biliary calculi vary considerably in size, number, form, and composition. In size they range from minute grains about the size of a pin's head to a mass as large as a hen's egg. The smaller they are, generally, the larger is their number. Their form is very various. When solitary they are usually round or oval; when numerous they are generally more or less irregular in shape, their surfaces being flattened and faceted from compression. The colour is usually a blackish-green or brown, less frequently yellow or greyish-white. In consistence, some are soft like wax; others hard, dry, and friable. On section they will be found to differ widely—some being granular, and made up of sub-morphous particles without any apparent nucleus; others crystalline, the glistening white crystals (cholesterin) radiating from a central nucleus, which is frequently found to consist of inspissated bile. Biliary calculi consist of two kinds. (a) Those of the one group are formed of cholesterin and bile-pigments, mixed with a variable proportion of insoluble organic matter, fatty matter, stearate of lime, and traces of the earthy phosphates. To separate the cholesterin, finely powder the gall-stone and thoroughly exhaust with ether; the ethereal solution on evaporation yields amorphous cholesterin. To obtain it in the crystalline form, it must be redissolved in boiling alcohol, which on cooling deposits it in characteristic, glistening, rhombic plates. The pigmentary matters can be obtained by exhausting the residue of the crushed gall-stone, from which the cholesterin has been removed, successively with water, alcohol, and dilute hydrochloric acid. The dried residue is then boiled with pure chloroform for some time, and the chloroform extract is distilled to near dryness, and several volumes of alcohol are added, which throws down bilirubin. Bilirubin thus obtained is an orange-red powder, insoluble in water and ether, slightly soluble in alcohol, but very freely soluble in chloroform. On passing a current of air through an alkaline solution of bilirubin the solution acquires a green colour—biliverdin. According to Städeler, biliverdin is formed from bilirubin by the addition of one atom of water in the presence of oxygen. A brown pigment, bilifuscin, can also be obtained by acting on bilirubin with strong sulphuric acid. (b) Calculi of the other group are composed

solely of one substance: such are the small white pearly calculi, frequently passed by young children and very old persons, and which consist entirely of cholesterin; small black masses like dried tar which consist of pigment; and brownish, irregular fragments which are formed solely of stearate of lime.

**2. Intestinal Calculi.**—Intestinal calculi are rare in man and carnivorous animals, but are not uncommon in herbivorous animals. They consist almost entirely of ammonio-magnesium phosphate, calcium phosphate, and calcium carbonate, deposited round a nucleus, generally a fragment of some indigestible material of the food, such as stones of fruit, husks of grain, or portions of bone. There is a kind of intestinal calculus occasionally met with among Scottish and Lancashire people, who use oatmeal largely as food, which is chiefly composed of the hairs and fragments of the envelopes of the oat, encrusted with calcium phosphate and carbonate. Magnesium carbonate when taken habitually and in bulk is apt to accumulate in the bowels and congregate there; there is now less risk of that danger since the fluid forms of magnesia have come into such general use.

**3. Pancreatic Calculi.**—Calculi in connexion with the pancreas are the rarest of all glandular concretions. When found they are generally numerous, being met with in the main duct, the accessory duct, and even in the smaller radicles. The size varies greatly, the largest that has come under the writer's observation being quite one inch in length. They are generally oval in shape, and their surface has frequently a worm-eaten appearance, of whitish colour, which when rubbed acquires an enamel-like lustre. When broken, the fracture often presents a white, glistening, porcelain appearance. One calculus analysed by the writer gave a percentage composition of organic matter 24, fixed inorganic salts 76. The bulk of the fixed inorganic salts consisted of calcium carbonate, calcium phosphate being present in much smaller proportion.

**4. Prostatic Calculi.**—Prostatic calculi consist essentially of calcium phosphate and calcium carbonate, though incidentally traces of uric acid, calcium oxalate, and ammonio-magnesium phosphate may be found. They occur in three forms, namely (a) small, rough concretions, from the size of a pin's head to a hazel-nut; (b) irregular masses with porcelainous appearance; and (c) large regular concretions. The quantity of earthy matter that may be deposited in the prostate gland is often enormous. When the calculi are of the small variety, fifty or sixty may be present, and a gland may feel like a bag of nuts. The Hunterian Museum contains a specimen showing the enormous size these concretions may attain. See PROSTATE, Diseases of.

**5. Salivary Calculi.**—These are gener-

ally rough externally, irregular in shape, and are usually found near the orifice of the duct, which they obstruct. The nucleus frequently consists of some foreign body which has accidentally found its way into the duct, as a splinter of wood or a fragment of bone. Their chief component is calcium carbonate, of which they contain more than any other kind of concretion, and traces of earthy phosphates.

**6. Urinary Calculi.**—Urinary calculi vary considerably in size, form, colour, and general appearance, according to their composition. The constituents that form these stones are uric acid, urates, cystin, xanthin, calcium oxalate, calcium phosphate, magnesium phosphate, ammonio-magnesium phosphate, calcium carbonate, and also concretions of blood and fatty substances (urostealith). Any of the above-named substances, combined with a varying proportion of organic matter, may constitute the sole ingredient of a calculus; more commonly, however, two or more are associated together. To fully ascertain the composition of the mass of the calculus it must be sawn across, and if made up of different layers, a portion of each layer must be analysed. See the articles on the several kinds of urinary calculi; and URINE, Morbid Conditions of.

**PATHOLOGY.**—The manner in which these concretions are formed, especially renal and urinary calculi, has long been a matter of speculation with physicians, some regarding them as of purely local origin, others endeavouring to show that they are the result of some peculiar diathesis, wherein uric acid, the phosphates, &c., are formed in the body so profusely, and are eliminated in such quantities as to be precipitated in the passages. The researches of Ord and Carter have thrown much light on this obscure subject. They have shown that the particles constituting the bulk of the calculus are not mere accretions, formed in the urinary passages by a process of chemical precipitation, in the presence of blood, mucus, &c.; but consist of structures, designated as 'submorphous'—granules, spheroids, laminae, &c.—and which require considerable time for their formation. They have shown that this modification of form, *i.e.* the change from the crystalline to the submorphous type, can be artificially produced by allowing two saline solutions to intermix slowly through a colloidal medium; as gum, albumen, &c.; the more slowly the mixture is effected and the denser the colloid, the more perfect is the change to the submorphous condition. On the other hand, if the colloid medium is attenuated, and the admixture rapid, the crystalline form is more or less retained. The nature of this colloid medium has not been clearly made out. The fact that calculus rarely accompanies Bright's disease shows that the ordinary effusion of blood or albumen

into the renal tubules does not furnish the necessary medium. Indeed, if simple effusion of fibrin, or increased secretion of mucus, furnished the colloid, calculus, instead of being comparatively a rare disease, would be extremely common. Some authors have regarded the 'entangling mucus' as the product of a specific catarrh. Thus Meckel speaks of a stone-forming catarrh (*steinbildender Katarrh*); and the late Dr. Owen Rees pointed out that among the evils attendant upon gout is 'a tendency of mucous membranes to secrete a viscid mucus, which modifies the ordinary crystalline character of uric acid, causing it to appear in agglutinated masses, which adhere to the sides of the urinary passages.' In speculations as to the origin of stone, too little attention has as yet been paid to the condition of the renal cells. These cells normally eliminate the urinary constituents; and it is not difficult to imagine that under certain conditions of vital impairment these substances may be retained and deposited, instead of being eliminated, the cell itself furnishing the colloid medium. The objection urged against this view is that recent observers have discovered no satisfactory signs of cell-structure in the matrix of calculi; but this objection can hardly be considered fatal, since the accretion of particles within the cell would gradually destroy the cell-structure. Professor Quekett, however, figured (*Med. Times*, vol. xxiv. p. 552, 1851) crystals of calcium oxalate and triple phosphate contained in cells taken from human tubuli uriniferi; and though his observations have not been confirmed by others, still the accuracy of his work has never been questioned, and it may be when the tubuli uriniferi of persons dying of calculous affections come to be more frequently examined by pathologists, cells containing calculous constituents at an early stage of deposition may be observed—that is, before the cell-wall is destroyed. It is a remarkable fact that calculous deposit commencing in the kidney tubules is rarely met with in the convoluted portion of the tubule, but invariably occurs at the apices of the mammillary processes, the extremities of the ducti papillares. Now less blood circulates through this portion of the kidney than through any other part of it, and moreover in the ducti papillares the basement membrane (*tunica propria*) disappears and the wall consists of epithelium alone. May not these anatomical differences render the cells of this part of the tubule more liable to calculous deposit—in short, to undergo calculous degeneration?

C. H. RALFE.

**CALIPERS.**—An instrument employed for measuring diameters, used more especially in clinical medicine for measuring the diameters of the chest. See PHYSICAL EXAMINATION.

**CALVITIES** (*calvus*, bald).—SYNON.: *Alopecia calva*.—A synonym for baldness. See BALDNESS.

**CANARIES, The, in North-east Atlantic Ocean; Teneriffe.**—Mean temperature in winter, 64° to 85°. Warmer, drier, but more variable, than Madeira. East winds from Africa. See CLIMATE, Treatment of Disease by.

**CANCER** (*cancer*, a crab).—SYNON.: Carcinoma; Fr. *Cancer*; Ger. *Krebs*.

**DEFINITION.**—The word cancer is without histological meaning. We find it and its synonym *carcinoma* used as long ago as the time of Hippocrates, and the latter term was then, as is the former at the present day by the vulgar, applied to any new-growth of a malignant character. The name originated in the large ramifying veins and puckered furrows which spread from a cicatrising cancer that is involving the skin. When the broad distinction between the epithelial and connective-tissue type of tumours was established by Virchow and others, it was decided to retain the word *cancer* as the name for the more malignant or epithelial growths; while the equally meaningless but less formidable word *sarcoma* has been, from this time, confined to those tumours which have connective tissue for their type. See TUMOURS.

In this sense of the word the cancers form a class which is, on the whole, easily distinguished by definite microscopical and clinical characters; but at two points, at least, the difference from simpler growths is almost imperceptible. First, as a matter of accident, one sarcoma (the alveolar) resembles a cancer so closely in microscopical structure, that it is impossible to distinguish between them without reference to clinical facts; and, in the second place, as cancers are essentially depraved modifications of epithelial, epidermic, or glandular structures, they may be found to differ so slightly in histological characters from simple hypertrophies, that the fact of ultimate malignancy is often all that can decide between, say, a papilloma and an epithelioma, a glandular cancer of the rectum and a simple polypus, or a scirrhus of the breast and a chronic mammary tumour.

**HISTOLOGICAL STRUCTURE.**—Histologically, cancers are distinguished by consisting partly of cells of an obviously epithelial origin and partly of connective tissue. The connective tissue forms alveolar spaces, and may vary in structure from a loose fibro-cellular material to strong and old fibrous tissue. The alveolar spaces communicate with each other and contain the epithelial cells. These vary much in shape, size, and arrangement, but are always easily separable from the surrounding connective tissue, while they

are never separated from one another by a stroma of any sort.

**CLINICAL CHARACTERS.**—Clinically, cancers are distinguished by the structures in which they originate; by the method of their recurrence and their mode of growth; as well as by a few characteristics apparent to the eye and touch.

*Seat.*—As their nature would *a priori* have rendered almost certain, cancers probably never originate except in connexion with epithelial or epidermic structures—*i.e.* in skin, mucous membrane, or secreting glands; but as the epidermis and epithelium, the original upper and lower layers of the embryo, are widely diffused throughout the body, and often intimately associated with the descendants of the cells of the middle layer, it is not surprising that primary cancers have been described as occurring in organs which have their origin from connective tissue only. Such are the instances of primary cancer of bone and lymphatic glands, the possibility of the occurrence of which may be at present considered undecided.

*Recurrence.*—The first recurrence is almost without exception in the lymphatic glands, which collect their supply of lymph from the seat of the original tumour; when this has occurred the process may be repeated in the next proximal lymphatic glands, or numerous distinct tumours may appear in different parts of the body; but if a single growth occur in another locality without previous glandular enlargement, the case may probably be looked upon as one of double primary development. A soft cancer may break into the abdominal cavity, where its small particles may stick to various parts of the peritoneum and form the starting-points of new-growths (disseminated cancer of the peritoneum); it is probable that a similar seeding may take place into the lungs when an ulcerated epithelioma projects into the trachea. The recurrence is usually of precisely the same structure as the original growth.

*Mode of growth.*—Cancers increase in size by infiltration of the surrounding tissues, and this gives rise to the very important clinical facts that they are not enclosed by a capsule like many simpler growths, and that they have a great tendency to implicate the skin and cause ulceration.

*Naked-eye appearances.*—The contraction of the connective tissue forming the alveoli in its advance towards fibrous tissue gives rise to puckering of the surrounding skin; and the looseness of the connexion between the epithelial and connective-tissue elements causes a milky juice consisting of the former to escape on scraping a recent section. This characteristic was made much of by our predecessors before the word *cancer* had lost its inclusive meaning; we know now that many rapidly growing *sarcomas* yield a similar

juice, but in less abundance than cancers; and thus it has come to pass that a milky juice is now more diagnostic of the malignancy than of the genetic origin of the growth.

This completes the list of the signs by which cancers may be distinguished from other tumours. Tables have been published to show the relative frequency with which cancer attacks different organs; they are not, upon the whole, trustworthy, and this question will be best considered in discussing the subdivisions of the genus.

**CLASSIFICATION.**—The subdivisions of cancers are as follows:—

<i>Hard cancer</i> or <i>Scirrhus</i> .	} GLANDULAR TYPE.
<i>Soft cancer</i> or <i>Encephaloid</i> .	
<i>Lobular Epithelioma</i> .	} EPITHELIAL and EPIDERMIC TYPE.
<i>Cylindrical Epithelioma</i> .	

*Colloid.*

Typical hard and soft cancers stand obviously at opposite ends of one series which is built upon the type of the secreting gland: between the two are an infinitude of intermediate stages. The two forms of epithelioma are, quite as evidently, monstrous growths of skin or mucous membrane. Colloid is probably the result of degeneration of any one of the other forms. Besides these, other varieties are often mentioned which do not justify a more complicated classification; amongst these are tumours, which though of nearly normal glandular structure are nevertheless malignant, and those which have received the names *melanotic*, *telangiectatic*, *osteocancer*, &c.

**DIAGNOSIS.**—The diagnosis depends upon the clinical characters of the several groups. That of an advanced case of cancer is generally easy; in the early stages it is mostly impossible.

**PROGNOSIS.**—The prognosis is always bad, especially in encephaloid cancer, but least so in epithelioma; this suggests the much-debated question of the constitutional nature of the disease. If in its origin a cancer be purely local, early removal ought to effect a permanent cure; but if there be at the bottom a constitutional taint, a reprieve should merely be granted until a suitable fresh irritation arise. There is probably some truth on both sides. The cancerous cachexia is often spoken of; it depends chiefly, if not altogether, on the weakening effects of the discharge after ulceration has taken place; mental worry may have some share in causing it; but it must be remembered that cancerous patients, who, before they are attacked, are frequently amongst the most robust, often retain their health for a remarkably long space of time.

**COURSE.**—The course of a cancer depends upon its seat, and the symptoms must accordingly be sought amongst the articles on

diseases of special regions. If, however, life be not shortened as a result of interference with the functions of the organ attacked, death is caused either by marasmus—the result of prolonged suppuration and pain—or by extensive or repeated hæmorrhages. The rate of progress is more slow as age advances.

**TREATMENT.**—The treatment of cancer in the early stages belongs to the surgeon, the practitioner giving such counsel as will improve the general health and support the strength. In the later stages the physician may be called upon to treat symptoms; but up to the present time all the specifics introduced either by regular practitioners or by charlatans have proved quite inefficient, if not actually harmful.

We shall now discuss the varieties of cancer.

**I. Scirrhus.**—Scirrhus, as its name implies, is amongst the hardest of tumours, if bony growths be excepted. Its hardness, as compared with soft cancer, depends upon the larger proportion which the alveolar stroma bears to the contained cells; and this is probably the consequence of the soil in which the tumours originate, and the rapidity of their growth, rather than of any specific difference between them.

**Seat.**—The female breast is the most common seat for scirrhus, but it also occurs in the stomach, uterus, tongue, œsophagus, and the liver and other glands, and it has been described as primary in the prostate, testicle, skin, and other structures.

**Naked-eye appearances.**—A section through the centre of a matured hard cancer of the breast presents to the naked eye well-marked and constant appearances, which, with the exception of such peculiarities as are due to the situation, will serve as a description of such a tumour occurring elsewhere. These are clearly explained by the microscopical arrangement, and when looked at by the light which it affords, fully account for all the clinical characters. The knife passes through it with a creaking noise, and the cut surfaces are at once hollowed in the centre. There is not a sharp edge to the growth, and the circumference is of a greyish or pinkish-white tint, projecting a little above the surrounding tissues, into which it sends small lobular prolongations; the hollow centre is very hard and of a glistening white colour. Scirrhus is evidently fibrous in structure, and receives from all quarters fibrous bands, which often pass far out into the fat of the breast or the skin, and some of which can nearly always be traced to the principal milk-ducts. Between the centre and the edge the greater part of the tumour is on the whole of a pinkish-yellow colour, but notably pink and soft externally, and yellow and hard internally. The surface yields a milky juice on scraping, and may

show some of the following appearances, which are, however, accidental: round the circumference little masses of healthy fat may be included, though this but rarely happens; cysts containing grumous grey or red fluid may have formed by the breaking down of the new-growth or by hæmorrhage; or such a hæmorrhage may have resulted in patches of yellow or even black pigmentation.

**Microscopical appearances.**—Without discussing the merits of the opposing theories as to the origin of cancer-cells, the following may be taken as the undoubted microscopical appearances of scirrhus. The grey outer layer is made up of indefinite smallish round cells, resembling white blood-corpuscles, infiltrated through the tissue into which the growth is spreading, amongst which are scattered a few which have the appearance of epithelial cells. The next or pink layer represents full development and shows fibro-cellular stroma, enclosing large epithelioid cells, and containing a copious supply of vessels. In the third or yellow layer the stroma has become fibrous, and the cells are undergoing fatty degeneration; and in the inner white centre the cells are replaced by indefinite masses of granular débris, and the stroma consists of firm and old fibrous tissue. See Plate, figs. 14, 16, and 17.

**Clinical features.**—The relation of these appearances to the clinical peculiarities of scirrhus is as follows: the excessive hardness is explained by the great development of fibrous tissue; the peculiar indefiniteness of the edge, and the tendency to involve the skin and ulcerate, by the manner of growth; while the puckering, retraction of the nipple, and indirectly (from the manner in which cutaneous nerves are involved) the pricking and shooting pains, are due to the contraction or cicatrisation of the stroma. To the latter is also due a very important but not generally recognised diagnostic character of an early scirrhus, namely, that long before the skin is involved it is seen to be dimpled when gently moved to and fro over the growth.

A scirrhus which has involved the skin forms a purplish-red, flattened, and shining tumour, covered with small veins and tender to the touch. The ulcer which results from its breaking down is ragged, with a hard base and hard irregular undermined edges, and a dirty surface covered by knobby masses of pseudo-granulations, which have a great tendency to bleed, and often slough. After removal it often returns in the scar. When occurring in the liver it is softer than elsewhere, and the name of scirrho-encephaloid is often given to it. See fig. 13.

**II. Encephaloid.**—Encephaloid, medullary, or soft cancer, so named from its usually brain-like appearance and consistence, is softer and grows more rapidly, and is more frequently observed in internal organs than

scirrhus, often indeed forming enormous intra-abdominal tumours.

*Scat.*—It has hitherto been observed as primary in the salivary and mammary glands, testicle, ovary, and prostate, the thyroid body, and in the mucous membrane of the nose, the liver, and the stomach. It has with some degree of looseness been called the cancer of childhood by those who consider scirrhus as almost peculiar to mature age.

*Naked-eye appearances.*—To the naked eye a fresh section usually presents a convex surface; it is whitish, but generally mottled by coloured patches, the result of old or recent hæmorrhages; and yields very copiously a milky juice on scraping.

*Microscopical appearances.*—Encephaloid cancer differs from scirrhus only in the relative proportion of the two chief factors. The cells are more numerous and are contained in larger spaces; they are sometimes small, but generally much larger than in scirrhus; and the stroma is delicate and fibro-cellular and very small in amount. *See fig. 15.*

Its method of extension is the same as that of other members of the class. It is by far the most malignant form of cancer, because of its rate of growth and recurrence, and the rapidity with which it causes general cachexia.

**III. Lobular Epithelioma.**—Lobular epithelioma, epithelial cancer, or canceroid, develops in connexion with skin and mucous membrane, and though consisting essentially of squamous epithelium, may start from a part which is covered by the cylindrical variety. It occurs near the natural orifices of the mucous tracts—as, for example, on the mouth and tongue, anus, penis, or vulva; but also at other parts of the skin—as on the scrotum (chimney-sweep's cancer) and at the upper end of the œsophagus. The history of a local irritation as its origin is often obtainable, but more frequently nothing of the kind can be discovered.

*Naked-eye appearances.*—The first appearance is that of a pimple, which soon breaks down in the centre, forming a small sore. When fully developed there is an irregular ulcer, with an extensive, hard and nodular, generally inflamed base and circumference; the edges are abrupt or undermined, and the floor grey or reddish, very uneven, discharging a foul pus, and with a great tendency to bleed. As a rule there is considerable pain, and the proximal lymphatic glands are very generally enlarged. A section to the naked eye shows a number of minute cylinders of yellowish-white colour, cut sometimes longitudinally, sometimes transversely, fusing together into an indefinite mass superficially, but more or less discrete below, and infiltrating amongst the subjacent tissues. On squeezing the section little nodules like sebum appear on the surface.

*Microscopical appearances.*—The cylinders or lobes of epithelioma are found to be made up of squamous epithelium, the cells of which generally exhibit in parts a crenated margin (Max Schultze's spine-cells). As in the skin, the deeper—that is the circumferential—layer of cells in each lobe, which are the youngest, are roundish or oblong, with large nuclei which stain readily; further in, the cells are larger and flatter, and in the centre are found the well-known globes or nests. These were at one time considered peculiar to epithelioma, but are now known to occur in warts and corns; they consist of onion-like arrangements of epithelial cells, varying much in size and the number of concentric layers, and containing in the centre sometimes an amorphous mass, sometimes large and irregular cells. The tissues beneath and between the lobules are infiltrated with small cells, and often contain in sections what appear to be isolated masses of epithelium; these are, however, the ends of divided divergent lobules. Opinions differ as to the exact starting-point of an epithelioma, the share which the sweat- and other glands take in it, and also as to the rationale of the formation of the globes. *See fig. 8.*

Epithelioma seems to be more local in its nature than other cancers—that is, a complete and early removal has not infrequently given the patient a long lease of life. It recurs, as a rule, in the lymphatic glands (which inflame and break down or suppurate) or in the scar; and generally proves fatal from the constitutional disturbance which these local manifestations give rise to. Later, but more rarely, it may appear in the internal viscera, bones, &c.

**IV. Cylindrical Epithelioma.**—The cylindrical epithelioma—badly named adenoid or glandular cancer—is specially the cancer of the alimentary mucous membrane, but may occur in the bladder and elsewhere.

*Naked-eye appearances.*—To the naked eye it forms at first a prominent tumour in the interior of a viscus, which has a tendency, like other cancers, to ulcerate and involve surrounding tissues, so that the mass may reach an enormous size, and may even make its appearance through the skin. To the naked eye a section is generally whitish and has a granular appearance, which is given to it by the tubules of which it is made. It frequently causes death by obstruction of the bowel; but if it last sufficiently long, it recurs in the lymphatic glands, and then in the viscera and other parts of the body. It is not unfrequent to find recurrences in the liver with little if any implication of lymphatic glands. The recurrence reproduces precisely the structure of the original tumour.

*Microscopical appearances.*—Cylindrical epithelioma consists essentially of irregular

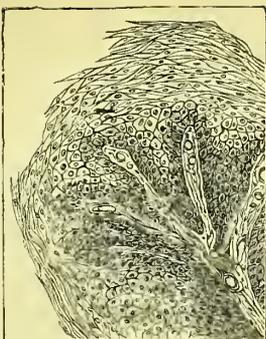


FIG. 7. Papilloma of Soft Palate.

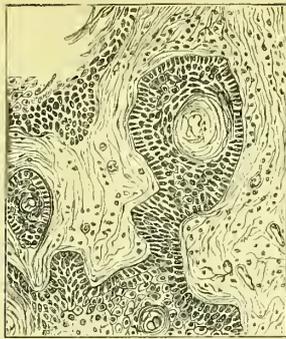


FIG. 8. Epithelioma of Lip.

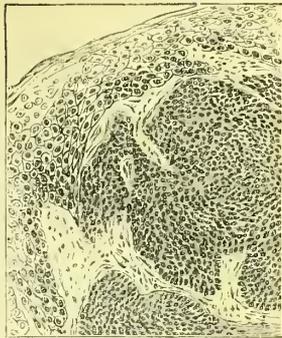


FIG. 9. Edge of Rodent Ulcer.

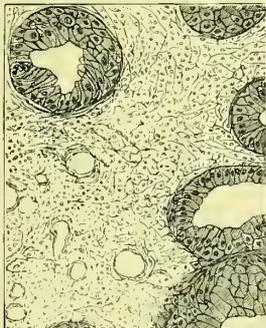


FIG. 10. Simple Polypus of Rectum.

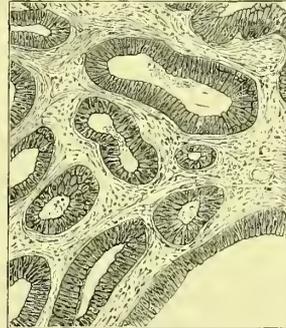


FIG. 11. Columnar Epithelioma of Intestine.

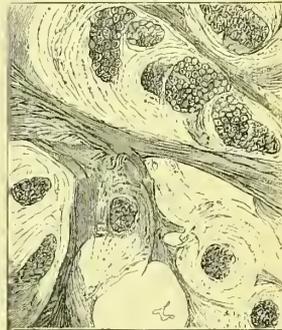


FIG. 12. Colloid of Breast.



FIG. 13. Cancer of Liver (Scirrhus-encephaloid).

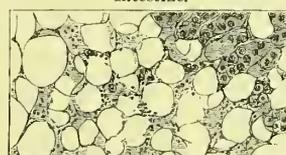


FIG. 14. Scirrhus infiltrating Fat.

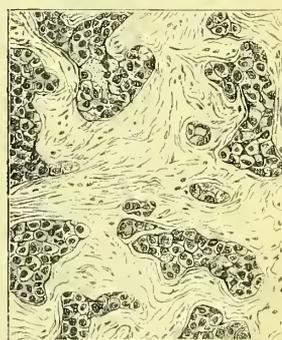


FIG. 17. Scirrhus of Mamma.



FIG. 15. Encephaloid Cancer.



FIG. 16. Cicatrizing Cancer.

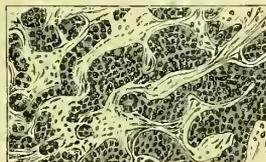


FIG. 18. Adenoid of Upper Jaw (Benign).



FIG. 19. Adenoid of Breast (common type).

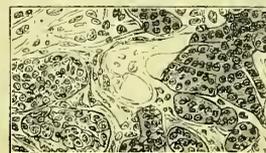


FIG. 20. Ulcerated Adenoid of Parotid (Malignant).

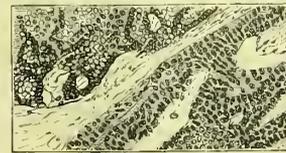


FIG. 21. Adenoid of Breast (epithelial element in excess).

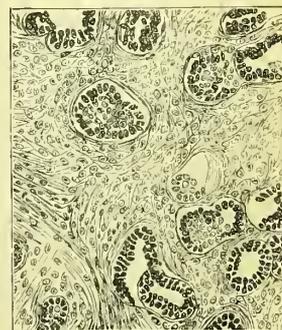


FIG. 22. Adenoid of Breast (Adeno-sarcoma).



tubules lined with columnar epithelium in one or more layers, which are the much overgrown crypts of Lieberkühn, and differs in microscopical structure from simple papilloma of the digestive tract only in the greater irregularity of the cells and in the larger proportion of connective-tissue stroma between the tubes. Compare figs. 10 and 11.

**V. Colloid.**—Colloid, or alveolar cancer, named from its jelly-like appearance, has given rise to much discussion in reference to the question whether it is developed originally in its mature form, or whether it results from the degeneration of one of the varieties of cancer already described. The latter view is that most widely held, though it must be allowed that epithelioma seldom degenerates in this way, and also that the colloid change usually takes place *pari passu* with the growth of the tumour.

**Seat.**—Colloid cancer is found most frequently in the abdominal viscera and peritoneum, but may occur elsewhere, as in the breast. Its malignancy is great, but is shown chiefly by the rapidity with which it involves surrounding tissues; it thus forms primary tumours of enormous size, but as a secondary growth is less common; it does, however, occur in lymphatic glands and other parts. It causes death in most cases by interference with the functions of the organs attacked.

**Naked-eye appearances.**—Colloid cancer consists of the naked eye of a mass of semi-transparent jelly, varying slightly in colour, but mostly pale yellow; this is intersected by delicate white fibrous bands, forming alveolar spaces of different sizes, visible to the naked eye. The consistence of the growth depends upon the relative proportions of these two constituents.

**Microscopical appearances.**—The bands are found to be actually fibrous; the contained jelly is arranged in concentric laminae between which are minute granules, and in the centre of which is a granular mass, sometimes quite indefinite, but often showing clearly that it consists of the remains of altered cells. These cells are seen in the more recent parts of the growth to be the subjects of colloid degeneration. The source of the colloid material must be considered still undecided; that some of it is formed by the cells is certain, but it is not equally clear whether the stroma takes any share in its deposition. See fig. 12.

**CONCLUSION.**—Our knowledge of the pathology of new-growths is undergoing a process of evolution, not now, however, so rapid as in recent years. Not much therefore has required alteration since this article was written nearly ten years ago, and it is hoped that it will still be found fairly to represent the opinions most widely accepted at the present day.

R. J. GODLEE.

**CANCERUM ORIS** (*canerum*, a sore; and *oris*, of the mouth).—**SYNON.**: Gangrenous Stomatitis; Noma; Water-canker; Fr. *Nome*; Ger. *Wasserkrebs*.

**DEFINITION.**—A rapidly progressive gangrenous ulceration of the cheek, gum, and lip.

**ÆTIOLOGY.**—Cancerum oris is usually seen in ill-fed, ill-tended children, living under the most faulty hygienic circumstances. It frequently ensues upon some acute depressing illness, especially measles (110 times in 226 cases—Sannée). Occasionally two or three cases occur together in a school, suggesting the possibility of contagion. Lingard has demonstrated long threads of small bacilli in the spreading edge of noma in man, and in a similar disease of calves; and inoculation on calves of a pure culture from man induced the disease.

**SYMPTOMS.**—Cancerum oris commences on the inner surface of the cheek, often near the angle of the mouth, or upon the gum; and at this stage some superficial ulceration will usually be found here—that is, ulcerative stomatitis. The disease rapidly advances. The cheek becomes much swollen, brawny, shining, and red; a livid spot appears in the centre, and here a spreading black slough forms, while the surrounding parts become purplish or mottled. If the patient can open his mouth sufficiently, the gums will be seen to be red and spongy, ulcerating, or destroyed; even the tongue may show some ulceration. The cheek is lined with a tough, adherent, greyish-yellow or soft purplish slough; the jaw may be laid bare, the teeth loosened, and portions of the alveolus necrosed. The breath is very foul, and the flow of saliva is excessive and fœtid. Fever may be moderate or high. Pain is slight or absent, and the child, though lethargic, is conscious almost to the end. Not more than one in twenty cases recovers, death being generally due to septic broncho-pneumonia.

**DIAGNOSIS.**—Formerly mercurial stomatitis sometimes ran on to gangrene in debilitated patients; this inflammation was general, and affected the tongue markedly. No hard line can be drawn between ulcerative stomatitis and noma.

**TREATMENT.**—This consists in complete and careful excision by knife and sharp spoon of the diseased area, regardless of the deformity which must result. To parts which cannot be excised, nitric or pure carbolic acid should be freely applied. Iodoform gauze is the best dressing. The mouth must be frequently and carefully syringed and sponged out with an antiseptic lotion, especially before food is taken. As far as possible, the child should lie so that foul saliva, &c. may dribble from the mouth and not down the throat. Dr. Goodhart thinks that even tracheotomy might be done to prevent inhalation of septic material into the lungs. Fluid food, especially pancreatized beef-tea or milk-gruel, should be given

regularly and frequently, by mouth, nasal tube, or enemata. Ammonia and bark or other tonics should be exhibited.

W. FAIRLIE CLARKE. STANLEY BOYD.

**CANITIES** (*canus*, hoary or greyhaired). Whiteness or greyness of the hair. *See* HAIR, Diseases of.

**CANNES**, in France, on the Mediterranean coast. A dry, bracing, fairly mild winter climate. Exposed to N.W. Abundant accommodation, both near and at some distance from the sea. *See* CLIMATE, Treatment of Disease by.

**CANTHARIDES, Poisoning by.**—*SYNON.*: Fr. *Empoisonnement par la Cantharide*; Ger. *Cantharidenvergiftung*.

Cantharides, the so-called Spanish Fly, owes its poisonous properties to the presence of *cantharidin*, a non-alkaloidal body. All the preparations of the drug are highly poisonous.

**SYMPTOMS.**—Soon after taking a poisonous dose of cantharides, the patient is seized with burning pain in the pharynx, and a sense of constriction in the œsophagus. The pain soon extends to the abdomen, and vomiting ensues; the abdominal pain becomes aggravated; and usually purging sets in. The stools are numerous, often scanty, passed with great pain and straining; they are at first mucous, and finally bloody. If carefully examined, little iridescent specks—portions of the elytra of the beetle—may be observed in the feces and vomited matters. These are of course only observable when the powdered insect has been taken; and they frequently escape observation. Up to this period of the case, should portions of the beetle not have been detected, there is nothing to distinguish the case from one of ordinary irritant poisoning; except, perhaps, that salivation and swelling of the salivary glands are usually prominent symptoms. The gastro-intestinal inflammation may be so intensely and rapidly developed, that death may occur from collapse before strangury, the diagnostic symptom, is set up. Generally, however, the course is somewhat different, genito-urinary irritation and inflammation setting in; the symptoms of which are aching pains in the lumbar region, frequent desire to micturate, with violent tenesmus of the bladder, till eventually a few drops of albuminous or bloody urine only can be passed, or none at all. Priapism, erotic excitement, and swelling of the genitals are of frequent, though not of constant, occurrence. Delirium, tetanic convulsions, or paraplegia, may be noted in some cases. Eventually the intolerable agony gives way to collapse, stupor, coma, and death. Abortion not infrequently occurs in pregnant women, the drug being formerly much used as an abortifacient.

**DIAGNOSIS.**—The intense strangury, the swelling of the genitals, and the bloody stools, will leave little or no doubt as to the nature of the case; and the presence of particles of the drug in the ejecta will be conclusive.

**Fatal dose.**—Less than half a drachm of the powder, and an ounce of the tincture, have alike proved fatal.

**TREATMENT.**—Evacuation of the stomach by the use of the stomach-pump, syphon-tube, or an emetic is the first indication in poisoning by cantharides. It is best to thoroughly wash out the stomach. Mucilaginous drinks, white of egg (not the yolk), and demulcents, may be freely given; but oil in any form is to be avoided, as tending to dissolve cantharidin. Opium by the mouth or rectum, or the hypodermic injection of one-third of a grain of morphine, is advisable. Leeches to the region of the bladder, warm fomentations, and warm sitz-baths may afford relief. Chloral should also be given, or the patient kept cautiously under the influence of chloroform. Collapse may be met by ammonia and other stimulants. The hypodermic injection of a few drops of ether is useful. There is no known antidote for cantharidin.

THOMAS STEVENSON.

**CAPE COLONY**, in South Africa. *See* AFRICA, SOUTH.

**CAPE OF GOOD HOPE.**—A warm, generally dry climate, but very variable, and liable to sudden storms. *See* AFRICA, SOUTH; and CLIMATE, Treatment of Disease by.

**CAPILLARIES, Diseases of.**—*SYNON.*: Fr. *Maladies de Vaisseaux Capillaires*; Ger. *Krankheiten der Capillargefässen*.—The morbid conditions of the capillaries may be described in the following order:—1. Fatty Degeneration. 2. Calcareous Degeneration. 3. Albuminoid Degeneration. 4. Pigmentation. 5. Changes in Inflammation. 6. Dilatation. 7. Narrowing and Obliteration. 8. Thrombosis. 9. Embolism. 10. Rupture. 11. The New Formation of Capillaries. 12. Capillaries in New-Growths and Tubercle. 13. Changes in the Perivascular Space and Sheath. 14. Teleangiectasis.

1. **Fatty Degeneration.**—This is the most common disease of the capillary-wall, and is frequently associated with fatty degeneration of the surrounding tissues. The cause of this change in the protoplasm of the capillary is, as elsewhere, interference with nutrition, and especially with oxidation. It is accordingly found in morbid conditions of the blood; in interference with the blood-supply; and in lesions of the nervous system. The microscopical characters of the early stages of fatty degeneration when it affects the capillaries are not peculiar; in advanced stages the diseased vessels may present the appearance of opaque granular cords; and

the lymphatic sheaths of the cerebral capillaries are sometimes found, under such circumstances, filled with oil-globules and fatty cells. A frequent termination of the disease is rupture and hæmorrhage. Fatty degeneration of the capillaries occurs most frequently in the nervous centres, where it can be readily demonstrated, in the kidneys, in certain tumours, and in the products of infarction and inflammation.

**2. Calcareous Degeneration.**—This disease is rare in capillaries.

**3. Albuminoid Degeneration.**—Albuminoid degeneration affects the Malpighian glomeruli in the early stage of this affection of the kidneys. In other parts of the body the capillaries are less subject to albuminoid change than the small arteries.

**4. Pigmentation.**—Pigmentary granules may sometimes be found in the walls of capillaries, but they more frequently occupy the perivascular space. In either situation pigmentation is the result of chronic congestion or inflammation, or of hæmorrhage.

**5. Changes in Inflammation.**—The changes of the capillaries of an inflamed part constitute an important factor of the process of inflammation. See INFLAMMATION.

**6. Dilatation.**—Dilatation of capillaries, which is one of the changes in passive venous congestion and inflammation, may become permanent if the cause persist.

Changes in the nutrition of the capillary-wall, combined with disturbances of the circulation, such as increased pressure, produce local dilatation or **Aneurysm** of the vessels, and subsequent rupture. This is one form of *miliary aneurysm* as it occurs in the brain. See BRAIN, Vessels of, Diseases of.

**7. Narrowing and Obliteration.**—Narrowing of capillaries may be temporary, as in inflammation; or permanent, from external pressure, or from interference with the blood-supply. Narrowing may proceed to complete obliteration.

**8. Thrombosis.**—Thrombosis rarely occurs in capillaries. See THROMBOSIS.

**9. Embolism.**—The phenomena of ordinary embolism in a great measure affect the capillaries corresponding with the obstructed artery. But, besides this change, capillaries are themselves subject to embolism, or impaction of particles within them. The products of inflammation or degeneration, pigment-particles, oil or fat-drops from the marrow of fractured bones, organisms, and various substances artificially introduced into the circulation, have been discovered obstructing the capillaries in different instances. All the possible results of embolism in large vessels may follow, according to circumstances, but anastomosis being very free, they are comparatively insignificant functionally, unless the obstruction be extensive; and in the case of the cerebral vessels definite symptoms are believed by some to result, such as

delirium and choreic movements. See CHOREA.

**10. Rupture.**—Three circumstances specially determine the occurrence of this lesion of capillaries, namely, disease of the vessel-wall, increase of the blood-pressure, and a 'terminal' distribution of the branches of the artery that supplies them. The most common diseases of the wall are fatty degeneration and aneurysm. The blood-pressure rises within the capillaries of any part in ventricular hypertrophy, in increased tension of the arteries of other parts, and in venous obstruction. When an artery is 'terminal,' that is, unprovided with other anastomoses than through its capillaries, no lateral relief can be afforded in sudden and excessive rises in the force of the circulation. For these several reasons, rupture of capillaries is most frequent when the vessel-walls have been weakened in the fatty degeneration of senile decay, in septicæmia, inflammation, purpura, fever, and scurvy; in chronic Bright's disease with increased blood-pressure; and in such organs as the corpus striatum, retina, spleen, kidney, villi, and skin. Disturbances in the pressure of the air within the chest powerfully influence the occurrence of capillary-rupture in the respiratory tract. When a capillary-wall gives way, the blood is extravasated either on a free surface, constituting hæmorrhage; into the substance of the tissues around; or along the lymphatic sheath of the ruptured vessel, where it gives rise to the appearance that has been described by some authorities as *dissecting capillary aneurysm*.

**11. New Formation of Capillaries.** Capillaries grow or develop in nearly all forms of new-growths, whether inflammatory or otherwise. The young capillaries are derived either from cellular buds upon previous capillaries, which become hollowed by the blood-pressure; from anastomosing exudation cells, or connective-tissue corpuscles; or, in some cases, from the parallel disposition of exudation-cells.

**12. Capillaries in New-Growths and Tubercle.**—The capillary-walls are believed to play an important part in the production of certain forms of new-growths. See TUMOURS; and TUBERCLE.

**13. Changes in the Perivascular Space and Sheath.**—The perivascular or lymphatic sheath, which probably envelops all capillaries, is liable to certain morbid conditions, which are chiefly secondary to changes in the vessel within it. Thus the space may become filled with blood from escape of the corpuscles by rupture or otherwise; with leucocytes in inflammation; with oil-globules and fatty corpuscles in degeneration of the wall; with pigment-particles; or with serum in disturbances of the circulation. The calibre of the perivascular canal, which varies inversely with that of the contained capillary,

may thus be increased, and present uniform or irregular dilatation.

Changes in the *outer* sheath, or wall of the lymphatic space, have also been observed, including fatty degeneration of the lining cells, and hyaline thickening.

14. **Teleangiectasis.**—At least one form of vascular tumour consists of a local overgrowth of capillaries, which are both enlarged and multiplied. *See* TUMOURS.

J. MITCHELL BRUCE.

**CAPILLARY BRONCHITIS.**—Inflammation involving the minute bronchial tubes. *See* BRONCHI, Diseases of.

**CARBOLIC ACID, Poisoning by.**  
*See* POISONS.

**CARBONIC ACID, Poisoning by.** The inhalation of carbonic acid causes injurious or fatal results, according to the length of time and degree of concentration. Carbonic acid accumulates in large quantities, almost undiluted, in pits, cellars, wells, mines (especially after explosions, constituting what is called choke-damp), volcanic grottoes, fermenting vats, lime-kilns, &c. A continuous contamination of the atmospheric air with carbonic acid goes on from the respiration of animals and the combustion of fuel. The generation of  $\text{CO}_2$  by a human being averages about 0.6 cubic foot per hour; that of an ordinary gas-burner 2 cubic feet of  $\text{CO}_2$  for every one of gas burnt; that of an oil lamp relatively more. The gradual exhaustion of oxygen and proportionate accumulation of carbonic acid in ill-ventilated apartments is one of the factors of the evil results of bad ventilation, but not the only one, as other animal exhalations contribute largely to the result.

As a rule excess of carbonic acid means corresponding deficiency of oxygen in the atmosphere, and the proportion cannot exceed 10 per cent. without rapidly fatal results ensuing; but much less than this causes injurious, and even fatal consequences if long inhaled; and less than 2 per cent. cannot be breathed for any length of time with impunity.

If the amount of oxygen be not correspondingly diminished, carbonic acid if present in sufficient quantity in the atmosphere respired will still act fatally. Thus Bernard found that a bird died instantaneously in an atmosphere of equal parts of oxygen and carbonic acid; and Snow found that 20 per cent. of carbonic acid in an atmosphere containing the normal proportion of oxygen soon proved fatal to small animals, and that even 12 per cent. might cause death after a longer interval.

**SYMPTOMS.**—Undiluted carbonic acid is not readily inhaled, as it tends to induce spasm of the glottis, but immersion in such an atmosphere is rapidly fatal. It seems to act as a narcotic. The patient falls down prostrate and insensible, and death occurs almost immediately. This effect is seen occasionally

when labourers incautiously descend an old well, or when miners enter a region filled with choke-damp. Not infrequently several fall victims, as one goes to see what has happened to the other and meets the same fate.

When the carbonic acid is more diluted, the symptoms are headache, giddiness, and sense of oppression, followed by drowsiness, and singing in the ears. Ultimately a condition of insensibility ensues, with stertorous breathing and muscular prostration; death usually occurring quietly and without convulsions. If the excess of carbonic acid corresponds with deficiency of oxygen, we have in addition to the essentially narcotic effects of carbonic acid, the dyspnoea and other symptoms of asphyxia. *See* ASPHYXIA.

**POST-MORTEM APPEARANCES.**—These are largely those of asphyxia, namely, a general engorgement of the venous system. This is generally seen in the brain more frequently than in asphyxia pure and simple. The blood is dark and fluid. The hæmoglobin is completely reduced. Animal heat is said to be retained long after death, and rigidity is well-marked and enduring.

**PATHOLOGY.**—As has been already said, carbonic acid does not act merely as a negative asphyxiant by taking the place of oxygen, but has a distinctly toxic narcotic effect. Very frequently in cases of poisoning by carbonic acid there is a combination of asphyxia, essentially due to deficiency of oxygen, with the narcotic symptoms due to carbonic acid.

**TREATMENT.**—1. *Prophylactic.*—Caution should be exercised in exploring wells, mines, &c., where there is any likelihood of the accumulation of carbonic acid. The introduction of a lighted candle is a rough and ready test of considerable value. The mere fact of a candle continuing to burn in an atmosphere is no test of its being respirable with impunity, for a candle will burn in an atmosphere containing 10 per cent. of carbonic acid if the oxygen is present in the normal amount, and the presence of an amount of carbonic oxide sufficient to cause death will not materially affect the flame. If carbonic acid reaches the proportion of 16 per cent. the candle will, however, be extinguished. If a candle is extinguished, then certainly the atmosphere cannot be breathed, and therefore the test is of sound practical value. If carbonic acid is present, it should be expelled by creating a draught of some kind. Thus wells may be swept by some such contrivance as an inverted umbrella, and a stream of air or steam can be directed into enclosed spaces.

2. *Restorative.*—Artificial respiration and its various accessories are needed to restore a person actually in a state of coma from carbonic acid. This treatment, of course, is subsequent to instant removal from the impure atmosphere. Pure oxygen should also be administered if at hand. *See* ARTIFICIAL RESPIRATION; and RESUSCITATION. D. FERRIER.

**CARBONIC OXIDE, Poisoning by.**

Carbonic oxide is a much more dangerous agent than carbonic acid, and to it are due many of the effects sometimes ascribed to the latter. Pure carbonic oxide is rarely generated out of the chemical laboratory, but mixed with other gases carbonic oxide is not uncommon. This is especially the case in the fumes of burning charcoal. The carbonic acid of the burning charcoal while passing over the heated embers loses an atom of oxygen, or takes up an atom of carbon, and is converted into carbonic oxide, which burns with a bluish flame at the top. The toxic action of charcoal vapours is essentially dependent on the carbonic oxide they contain. Usually charcoal fumes contain from 2 to 3 per cent. of carbonic oxide, to 25 of carbonic acid, along with some heavy carburetted hydrogen. The vapours, however, are still as effective after being passed through lime-water, which fixes the carbonic acid. Poisoning by charcoal vapour is not an uncommon form of suicide, more particularly abroad, and many cases have occurred accidentally in this country, from sleeping in rooms in which there was no flue for the escape of the fumes of burning charcoal, or into which there has been leakage from stove pipes, &c. Carbonic oxide is always found in the air of rooms where cast-iron stoves are used, from some of the carbon of the cast iron itself being formed into CO, and the iron when heated allowing a large quantity to pass through the metal.

Carbonic oxide also exists in coal gas, and in still larger quantities (44 per cent.) in the new water-gas, constituting their main danger. It is likewise found in the emanations from brick-kilns.

Carbonic oxide is an extremely active poison. Letheby found that .5 per cent. of carbonic oxide in the respiratory medium killed small birds in three minutes, and that 2 per cent. killed a guinea-pig in two minutes. Many similar experiments have been performed with similar results. The animals soon become insensible, and die generally without exhibiting convulsive phenomena beyond a few tremors or flutterings.

**SYMPTOMS.**—In man inhalation of carbonic oxide for a short time, as Sir H. Davy and others have proved on themselves, causes headache, pulsation in the temples, giddiness, nausea, and great prostration, tending to drowsiness and insensibility. Continued inhalation causes profound coma. Usually death occurs quietly, but signs of vomiting are frequently observed near those who have been poisoned by charcoal fumes.

**POST-MORTEM APPEARANCES.**—The specially characteristic appearance of death from carbonic oxide is the cherry-red colour of the blood and internal organs. The post-mortem hypostasis exhibits a similar bright red tint. Frequently in those poisoned with carbonic oxide the face retains a ruddy hue.

The red tint of the blood is due to the compound which carbonic oxide forms with hæmoglobin. Carbonic oxide displaces the oxygen and forms a very stable compound with the hæmoglobin, not readily broken up, and hence the oxygen-carrying power of the corpuscles is paralysed.

In the spectroscope carbonic-oxide blood exhibits two absorption-bands very similar to those of ordinary blood-colouring matter or oxyhæmoglobin, but a difference in the exact breadth and position of the bands can be made out by means of the microspectroscope, when the two are compared together. Carbonic-oxide-hæmoglobin resists reduction in the usual manner, and here again differs from normal blood-colouring matter. Hoppe-Seyler gives as an additional test the action of caustic potash on carbonic-oxide- and ordinary hæmoglobin respectively. With the latter it causes a green colour on a porcelain plate, while with the former the colour continues red.

**PATHOLOGY.**—Carbonic oxide acts by paralysing the blood-corpuscles, as Bernard expresses it, and rendering them unable to take up oxygen. Hence internal respiration is prevented, and death ensues from asphyxia.

**TREATMENT.**—As carbonic-oxide-hæmoglobin is a very stable compound, and offers a very considerable resistance to displacement by oxygen, though not absolute as was at one time considered, artificial respiration is not likely to be successful by itself. The best treatment is venesection and transfusion of fresh blood. This method of treatment has proved successful in one or two instances in which it has been employed.

D. FERRIER.

**CARBUNCLE** (*carbunculus*, a small coal—*carbo*).—**SYNON.**: Anthrax; Fr. *Anthrax*; Ger. *Karbunkel*.

**DEFINITION.**—A specific local inflammation of the subcutaneous areolar tissue, rapidly leading to sloughing of the deeper and more central parts, followed by destruction of the skin; the whole of the dead tissues finally separating in the form of a slough.

**ÆTIOLGY.**—Carbuncle is a constitutional affection, dependent upon conditions of general debility or of plethora, and often associated with gouty or diabetic tendencies. It is more commonly seen in men than in women; is rarely met with under the age of twenty; and it attacks all ranks of life.

**SYMPTOMS.**—The most common seat of carbuncle is the back of the trunk or neck, but it may occasionally be found in other situations. The affection usually begins as a painful, hard, slightly elevated, and ill-defined swelling, which gradually increases in extent and assumes a dusky red tint. A vesicle containing bloody serum soon forms over the most prominent part, and on rupturing discloses several small apertures in the sub-

jacent skin, which give exit to a glutinous purulent discharge. This sieve-like condition of the undermined integument often persists throughout the course of the disease. Occasionally, however, owing to the destruction of the intervening skin, the several apertures merge into a single, large, ragged opening, and thus expose the characteristic ash-grey, slimy slough, which separates slowly by supuration, leaving an irregular cavity with deeply undermined edges. The cicatrix left after healing is usually uneven and may be permanently discoloured.

In the early stage of the disease, while the inflammatory œdema is still extending, the patient generally complains of a burning, throbbing sensation in the part, which may become intensely painful; but on the full exposure of the slough, the pain diminishes, and in the later stages it may cease entirely.

When the carbuncle is large, or involves a portion of the scalp, there is usually considerable constitutional disturbance of an asthenic type. Death may then occur from exhaustion, which is sometimes aggravated by free hæmorrhage resulting from incisions; but the most frequent cause of a fatal termination is pyæmia.

**DIAGNOSIS.**—Carbuncle is distinguished from boil by the size and extent of the swelling, and by its tendency to spread; by the livid tint of the skin, and the early formation in it of more than one aperture; by the character of the slough, by the severity of the pain, and the marked constitutional disturbance; and, finally, by the fact that carbuncle, unlike boil, usually occurs singly.

**PROGNOSIS.**—This will depend chiefly upon the age of the patient, and upon the seat and extent of the disease, which proves most dangerous to life when situated or encroaching on the scalp, especially in a person over fifty. The coexistence of albuminuria or chronic saccharine diabetes is always a grave complication.

**TREATMENT.**—The *constitutional* treatment and the management of patients with carbuncle are best conducted on general principles. In ordinary cases the diet should be of good quality and sufficient in quantity, with a moderate allowance of stimulants, proportionate to previous habits. Should the patient's strength and the situation of the carbuncle allow him to move about, he need not be confined to his room, and may even be allowed exercise in the open air.

In the more severe forms of the disease, the frequent administration of dietetic stimulants and good nourishment in an easily assimilable form is usually necessary. The bowels, if they require it, should be cleared out by some non-irritating aperient, and the patient put on a course of quinine or bark and the mineral acids. Opium or other sedatives may be required in the earlier stages to relieve the intense sufferings of

some patients; while in the after-course of the disease, it may be sometimes needed to procure sleep.

For *local* treatment see BOILS.

For *carbuncle of the face*, an affection distinct from the Malignant Pustule described by Continental surgeons, see PUSTULE, MALIGNANT; and BOILS.

WILLIAM A. MEREDITH.

**CARCINOMA.** See CANCER.

**CARDIAC DISEASES.** See HEART, Diseases of.

**CARDIALGIA** (*καρδία*, the heart; and *ἄλγος*, pain).—A synonym for heartburn, originating in a popular impression that this painful sensation, which starts from the epigastrium, is connected with the heart. See HEARTBURN.

**CARDIOGRAPH**, The (*καρδία*, the heart; and *γράφω*, I write).—This is an instrument for registering graphically the form of the heart's movements. It really records a tracing of the cardiac impulse, the curves of the trace representing the changes of pressure exerted by the apex of the heart on the chest-wall. We owe the invention of the cardiograph to Marey, who, in his physiological researches on the circulation of the blood, obtained by the following means the form of movement of each cavity of the heart. He introduced into the auricles and ventricles of a horse, hollow sounds terminating in elastic ampullæ filled with air. The air communicated through the sounds and elastic tubes with terminal ampullæ, or tympana covered with elastic membrane, on each of which rested a light lever. The movements communicated by the heart to the closed column of air were amplified by the levers and recorded by them on a revolving cylinder. In this way tracings of the forms of movement of each cavity were obtained, and by comparison with a tracing taken simultaneously of the exposed apex-movement, an explanation of the complex apex-tracing was rendered possible. The cardiograph used for clinical research is a modification of the above, and consists of a hollow cup containing a small spring which can be depressed by means of a screw so as to rest firmly on the chest-wall where the impulse is felt. The cup communicates by means of an elastic tube with a tympanum covered with elastic membrane, carrying on its surface a lever. When applied to the chest the cup hermetically seals the air column, which transmits as waves the motion received by the spring to the lever resting on the tympanum. The movements are recorded by the end of the lever, either on the plate of a sphygmograph or on a revolving cylinder.

By means of this apparatus a very perfect representation of the cardiac movements can

be obtained, the auricular and ventricular elements traced, and the duration of each measured. The transmission of the motion through an elastic medium like air has been objected to, as liable to modify the tracing by (1) the production of secondary oscillations in the air column; and by (2) gradual change in the form of motion caused by the elasticity of the medium. Practically, however, these objections are not valid, as is shown by the fact that the last of a series of cardiac pulsations is often an exact reproduction of the first, and also by the close resemblance between the tracings obtained in this way and those registered by the sphygmograph, or an enlarged modification of it, such as Galabin's cardiograph, applied over the apex-beat. POND'S sphygmograph may also be used as a cardiograph.

A cardiogram collected on a healthy person by the instrument described is here given.



FIG. 23.  
Cardiogram.

One cardiac revolution is figured. The several waves may be interpreted as follows. The wave *a*, in the line of ascent, corresponds with the early part of the ventricular diastole. The wave *b* corresponds with the true auricular systole; from *b* to *d* the line marks the true impulse caused by the ventricular contraction, the rounding of the heart, and its pressure against the chest-wall. The wave *c*, at the summit of the curve, immediately follows the closure of the auriculo-ventricular valves, which does not always form the summit of the curve, but occasionally in slower cardiac contractions is marked by a wave below the summit. The waves between *c* and *d* are oscillations produced by the closure of the auriculo-ventricular valves. The length of the summit *c* to *d* is important; it is long and sustained in hypertrophy, narrow and pointed in dilatation. Its length is a measure of systolic strength. The closure of the aortic valves is marked by *d*; while *e* marks the closure of the pulmonary valves, separated by an interval of .05 to .09 second. If the pressure in the aorta falls and that in the pulmonary artery rises (pulmonary second sound accentuated), *d* and *e* approach and may no longer be distinct. In the above cardiogram the period of ventricular contraction is measured by the space between the commencement of the line of ascent after the wave *b*, to the point *d*, which marks the termination of the systole; the rest of the curve is diastolic. The wave *d* is prominent in hypertrophy of the left ventricle.

The clinical value of the cardiogram has yet to be fully established. It has hitherto been useful in showing the relation of præ-systolic murmur and thrills to the ventricular and auricular contractions; in demonstrating modifications of the form of impulse in ad-

herent pericardium; in recording a considerable increase in the wave *a*, and a sudden rising of the trace after *a* to *b*, as signs of aortic insufficiency; and in the recognition of the relation between reduplication of the heart-sounds and respiratory influences. The wave *a* is larger the more sudden the relaxation of the ventricle, and thus it is increased by the influx of the blood in cases of aortic and mitral regurgitation. Waves following *a* usually indicate vibrations and thrill due to mitral stenosis, while prominence of wave *b* indicates auricular hypertrophy.

The cardiograph is also of great value in registering the form of movement of pulsating tumours and aneurysms. In its application, it is sufficient to hold the instrument firmly over the apex-beat, and to record the pulsations at the end of expiration, the breath being stopped for a brief interval. When the influence of respiration is to be observed, this precaution is of course unnecessary; but it must be borne in mind that the movement of the chest-wall modifies the tracing.

(BALTHAZAR) WALTER FOSTER.

**CARDITIS** (*kapdia*, the heart).—Inflammation of the substance of the heart. *See* HEART, Inflammation of.

**CARIES** (*caries*, rottenness).—A destructive inflammatory disease of bone, analogous to ulceration of soft tissues. *See* BONE, Diseases of.

**CARLSBAD**, in Bohemia. Thermal alkaline sulphated waters. *See* MINERAL WATERS.

**CARMINATIVES** (*carmino*, I card, or cleanse).

**DEFINITION.**—Substances that aid the expulsion of flatus from the stomach and intestines, and relieve griping.

**ENUMERATION.**—The principal carminative remedies are—the essential Aromatic Oils, for example, Peppermint and Cloves; Chloroform; Charcoal; Ethers; and Camphors, and substances containing them.

**USES.**—The uses of carminatives are sufficiently indicated in the preceding definition. They are extensively administered in cases of flatulent dyspepsia, especially when it is associated either with disease or disorder of the heart, or with a nervous or hysterical state of the system. A combination of several different carminatives is usually more successful than the exhibition of a single drug, one of the best being a mixture containing Spirit of Ether, Aromatic Spirit of Ammonia, and Tincture of Orange. Given along with antacids they are useful in correcting acidity, as in the favourite Soda-mints; and they are frequently prescribed with purgatives to prevent pain—an application illustrated by the Compound Rhubarb Pill.

T. LAUDER BRUNTON.

**CARNIFICATION** (*caro*, flesh; and *fit*, I become).—A condition of the lung in which it resembles flesh. The term was formerly applied to the transformation of any tissue into a flesh-like substance. See LUNGS, Collapse of.

**CARPHOLEGY** } (*κάρφος*, chaff; and  
**CARPHOLOGY** } *λέγω*, I collect).—The movements of the hands and fingers observed in certain delirious patients, as if they were searching for or gathering imaginary objects. A familiar illustration of the act is 'picking of the bedclothes.' See TYPHOID FEVER.

**CARTILAGE, Diseases of.**—SYNON.: Fr. *Maladies du Cartilage*; Ger. *Knorpelkrankheiten*.—For a due appreciation of the abnormal conditions to which cartilage is subject, a brief description of this tissue in its healthy state is necessary.

The *temporary* cartilage, which forms the early skeleton, gradually undergoes conversion into bone, leaving at the joint surfaces a thin layer, the *articular* cartilage, which never becomes ossified. Certain other portions of the skeleton also retain their cartilaginous condition throughout life; these are known as the *permanent* cartilages, and as examples the cartilages of the ribs, ears, and nose may be named. The extremities of the long bones, or epiphyses, remain separate from the shaft for a varying period after birth, and so long as the bone continues to grow, they are attached to it by a thin but important layer of cartilage, called the *epiphysial* cartilage. There are also the *fibro-cartilages*, in which the fibrous and cartilaginous elements are found in varying proportions, according as the tenacity of the one or the elasticity of the other material is required.

Cartilage is altogether destitute of nerves, and therefore of sensibility; and it is equally devoid of blood-vessels, being nourished by imbibition from the vessels of the neighbouring parts. All cartilages, except the articular and the fibro-cartilages, are covered by a fibrous membrane, the *perichondrium*, which is similar to, and subserves the same purposes as the periosteum. When cartilage has been destroyed, it may be replaced by fibrous tissue or by bone, but it is never reproduced.

Under the microscope a section of cartilage presents a transparent, structureless matrix, studded with nucleated cells; these cells are flattened and arranged parallel with the free surface of the cartilage, whilst more deeply they are elongated and grouped vertically. The nutrient materials are absorbed from the neighbouring blood-vessels, and transmitted throughout the cartilage by means of these cells. In those cartilages where tenacity or flexibility is needed, this

hyaline substance is denser and more distinctly fibrillated than in the others.

**SUMMARY OF DISEASES.**—Cartilage being non-vascular, its inflammation is of a modified type, and it may undergo degenerative changes as the result of impaired nutrition. In uncomplicated disease of cartilage there is no inflammatory exudation, and when lymph or pus is found in a joint, it is obvious that other structures have become inflamed.

1. The ensiform and costal cartilages, with those of the trachea and larynx, show a great tendency to *ossification*, as the result of senile changes; they are also liable to *necrosis*. The articular cartilages never ossify, but large or small portions of them may perish and be detached, in consequence of some interference with their supply of nutriment from the subjacent bone. In pyæmic joint-effusions the cartilage softens, and is rubbed off the most prominent portions of the articular surfaces in a manner quite characteristic. In osteo-arthritis, one of the earliest of the pathological changes occurs in the articular cartilage. The cells proliferate, the primary capsules enlarge and fill with secondary capsules. The matrix fibrillates and softens, and by degrees the cartilage is worn off the central part of the articulation, exposing the bone underneath; laterally, where the pressure and friction are less, the cartilage cells proliferate and form a tuberculated margin, which subsequently ossifies.

2. The cartilages of the epiglottis, ears, nose, eyelids, and Eustachian tube have little disposition to ossify, but they are liable to *ulceration*, especially of the syphilitic variety; in these cases the diseased action commences in the skin or mucous membrane, and spreads to the cartilage by contiguity.

3. The cartilage of the external ear is often the seat of *chalk-stones* in gouty persons, and similar deposits of urate of sodium are found in the articular cartilages.

4. The epiphysial cartilage may be the seat of *inflammatory changes*, generally very acute, which lead to separation of the shaft from the epiphysis, a condition which, whether the result of disease or accident, is of great moment, inasmuch as the destruction of this layer of cartilage checks further growth at this end of the bone.

Primary epiphysitis is rare: the changes in the epiphysis usually spread to it from the extremity of the diaphysis. Syphilis, especially of the inherited form, is the most common cause of this disease. Injury, particularly in strumous children, is also a factor in its production. The acute form is often associated with general septicæmia, and is followed by destructive changes in the adjacent joint.

5. Cartilage is not primarily attacked by *cancer*, but it may become involved by the spread of a malignant tumour. The *epithelial* form of cancer not infrequently

extends from the mucous or cutaneous surface, in which it originated, to the subjacent cartilage.

6. The articular cartilages are liable to certain structural changes as the result of disturbed nutrition; and the fibro-cartilages are also subject to the same abnormal conditions.

*Ulceration, absorption, degeneration of cartilage*, are terms used to denote a series of destructive changes which take place in the substance of articular cartilage, and lead to its partial or complete removal. These changes may originate in the cartilage itself, or they may be secondary to disease of the bone or synovial membrane; however this may be, the morbid action is the same, and consists in increased cell-development, with disintegration of the hyaline substance.

According to the observations of Goodsir and Redfern, the cartilage-cells become enlarged, filled with nucleated corpuscles, and arranged irregularly; the distended cells then burst, and set free their contents upon the surface, or in the midst of the altered hyaline substance. Whilst the cell-changes are taking place, the matrix softens: in acute cases it rapidly disintegrates and is discharged; but when the disease is more chronic, it splits up into fibres which remain attached by one end to the cartilage, and at the other project loosely into the interior of the joint, giving a villous appearance to the affected spot. The remains of the matrix and the granular contents of the cells together form a fibro-nucleated membrane, which ultimately is converted into fibrous tissue, and constitutes the sole medium of repair when a cure is effected. When this membrane is recent it has an indistinct granular appearance, from the presence of nuclei amongst the fibres, and according to Rainey these nuclei are often converted into fat-globules; when the membrane is of older date it is distinctly fibrous; and no doubt the several appearances which the membrane presents under different circumstances have led to the several terms *fibrous, fatty, and granular degeneration* being applied to this disease, in the belief that they were really distinct pathological conditions.

Changes of this kind may be primary, as in osteo-arthritis, but they are commonly secondary to disease in the articular ends of the bones, the result of syphilis, tubercle, or injury.

W. MAC CORMAC.

**CASEOUS DEGENERATION.**—A form of degeneration in which the products present the appearance of cheese. See DEGENERATIONS.

**CASTS** (derived from the Middle English word *casten*, which is adapted from the Old Norse *kasta*, signifying *to cast or throw*). SYNON.: Fr. *Cylindres*; Ger. *Cylindern*.

**DEFINITION.**—A term applied to moulds of

gland-tubules and hollow viscera, thrown off in certain states of disease.

**CLASSIFICATION.**—The varieties of casts met with may be thus grouped:—

A.—CASTS OF GLAND-TUBULES.

I. Of the Uriniferous Tubules.

- Blood-casts.
- Pus-casts.
- Hyaline casts.
- Granular casts.
- Epithelial casts.
- Fatty casts.

II. Of the Seminal Tubules.

III. Of the Gastric Tubules.

IV. Of the Bile-ducts.

V. Of the Cutaneous Glands.

B.—CASTS OF HOLLOW VISCERA AND PASSAGES.

I. Of the Alimentary Canal.

II. Of the Urinary Bladder.

III. Of the Female Genital Passages.

IV. Of the Respiratory Passages.

A.—CASTS OF GLAND-TUBULES.

I. Of the Uriniferous Tubules.—Dr. F. Simon, of Berlin, is usually credited with having been the first to describe these bodies in his work on *Medical Chemistry*, published in 1842; but it appears that before then they had been noticed and described by Vogla in 1837 and 1838, by Rayer in 1838, and by Nasse of Marburg in 1842. These observers, however, do not seem to have entered on the question of the origin, structure, or significance of these bodies, and for years they were looked upon rather as curiosities, and by some writers were wholly disregarded. Henle, in 1844, appears to have been the first to refer their origin to the coagulable matter of the blood, but it was some time after that date before their value in the diagnosis and prognosis of renal diseases came to be appreciated. This result has been mainly effected in this country by the labours of Basham, Lionel Beale, George Johnson, William Roberts, Dickinson, and Grainger Stewart.

Casts may be formed in any part of the kidney. They have been found in the convoluted tubules even up to the Malpighian capsules, and also in the straight tubules. Various modifications in shape and contour probably occur in the course of their passage along the tubes, even to the inclusion of a small cast in one of larger size.

**CHARACTERS.**—The urinary casts are mostly cylindrical in shape, frequently somewhat coiled and bent, and occasionally forked. Their length, depending very much on accidental circumstances, varies considerably. In sections of the kidney they may be occasionally traced for some distance in the tubules; but they become broken up into smaller pieces after leaving the gland. In

diameter casts range between  $\frac{1}{1000}$  and  $\frac{1}{500}$  of an inch—the former being known as ‘small,’ the latter as ‘large’ casts. The greater number are of a ‘medium’ size of  $\frac{1}{700}$  of an inch. The diameter of the casts is in part determined by the calibre of the tubule in which they are first formed, and in part by any subsequent additions they may receive in their passage outwards. Dr. Beale has suggested that after their formation the casts may probably shrink. In tubules that have become abnormally dilated or contracted, casts beyond the limits above mentioned may be found.

The appearance of renal casts varies considerably, not only in different kidney-diseases, but also in various stages of the same affection. In all cases the cast consists of a solid cylinder of a transparent or very faintly granular substance, which is rarely fibrillated. It was formerly accepted that these bodies were produced by a coagulation of fibrin due to an escape of blood-plasma into the tubules, and hence they were known as ‘exudation-cylinders’—a term still often employed. It is easy to understand the formation of casts in this manner, and it is certain that such *blood-casts* do occur, whether as shreds of fibrin with abundance of blood-corpuses in its meshes, or as casts consisting of little more than pure fibrin with its characteristic fibrillated appearance. In a similar manner *pus-casts*, so-called, may be produced.

In most cases of renal disease, however, in which casts are found in the urine, these bodies usually appear as transparent and

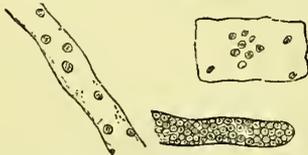


FIG. 24.—Blood-Casts.

faintly granular, tolerably uniform cylinders, frequently somewhat rounded at the extremities, and often overlooked unless searched for with care. In them no sign of fibrillation is to be discerned, and they do not cor-

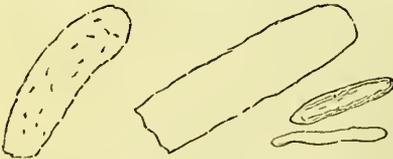


FIG. 25.—Hyaline Casts.

respond in their chemical behaviour to fibrin. These are the *hyaline*, *transparent*, or *waxy* casts, which may be large, small, or medium in diameter; the largest and smallest casts met with belong to this variety. Several views, supported by experimental evidence,

prevail as to the nature and source of these bodies; and it is not improbable that, differing in their composition, they may originate in either of the following ways:—

(a) The coagulation of some proteid material, which has transuded from the blood-plasma into the tubules, the epithelial cells being unaffected, and, so far as is known, taking no share in the formation of the cast. Such may be called ‘*transudation cylinders*,’ and masses of the coagulated substance are occasionally to be seen between the epithelial cells and the *membrana propria* of the tubule. The exact nature of the material is unknown; as stated above, it is not ordinary fibrin, but is probably some proteid derivative which has been made to coagulate—perhaps, as has been suggested, by the acid urine, or by the gland-cells.

(b) In contrast to the foregoing, it has been demonstrated that the living epithelium may, under certain conditions of irritation, as from cantharides, swell up, and, undergoing a form of colloid or mucoid degeneration, become transformed into clear transparent casts of the tubes, sometimes exhibiting a partial segmentation, indicative of their origin from several coalesced portions, or involving the remains of cells which have been only partially metamorphosed. To these the term ‘*fusion cylinders*’ has been applied. Or the cells, without being entirely converted into colloid matter, may appear to secrete drops of such material, which are moulded in the tubules into hyaline casts—the so-called ‘*secretion cylinders*.’ Here, again, the actual nature of the cast-substance is unknown, though described as mucoid or colloid; and from the great variability in its behaviour to staining fluids and other reagents, it most probably differs much in composition. Occasionally some of the large waxy casts will give the characteristic reactions, with iodine or with methyl-violet, of lardacein; even, it is asserted, when the kidney itself offers no evidence of lardaceous change.

Whatever may be the true explanation of their formation, they present themselves as semi-solid and somewhat viscid cylinders, readily entangling adjacent matter. Due to this property is much of the variety they offer; thus, should the epithelium of the tubules be loosened, as in nephritis, the



FIG. 26.—Epithelial Casts.

cells will adhere to the cast which has been formed in the lumen of the tube, and an *epithelial cast* will be voided. Such casts may present but one or two cells on their

surface, or may appear as if consisting only of coherent epithelial cells. Should the cells

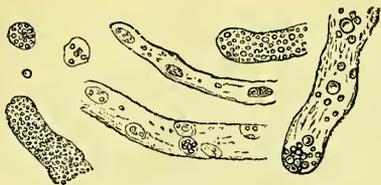


FIG. 27.—Fatty Casts.

have undergone fatty degeneration, the cast will be pervaded with oil-globules of all sizes, more or less escaped from epithelial cells, according to the extent of the degeneration; this constitutes a *fatty cast*. Very frequently the casts are finely or coarsely *granular*, this appearance being produced by the involve-

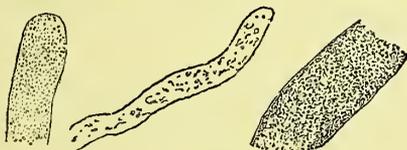


FIG. 28.—Granular Casts.

ment in the base-substance of granular matter derived from broken-down epithelial cells or blood-corpuscles, molecular fatty matter, amorphous urinary salts, or micro-organisms. Granular casts may exhibit a faint yellowish

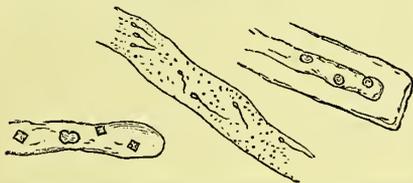


FIG. 29.—Casts enclosing crystals; and a smaller cast; also of seminal tubule with spermatozoa.

or brownish tint depending on their constitution. In a similar way casts may be found containing *crystals* of oxalates, triple phosphates, hematoidin, salts of fatty acids, &c.

It is very commonly the case that more than one variety of cast occurs in the same urine—epithelial and hyaline, or granular and fatty, often co-existing. Corresponding to the casts in the urine, free epithelial cells, blood-corpuscles, fat-globules, and salts, amorphous or crystalline, are always found, and almost always albumin, though not of necessity is there any correspondence in the quantity of this substance and the number of casts. The epithelial cells, whether free or on the cast, are rarely quite normal in appearance. The pathological changes associated with their desquamation have at the same time altered them more or less. Not infrequently the cells of an epithelial cast present all the microscopic characters of leu-

ocytes produced by an abnormal proliferation of the renal epithelium.

*Method of examination.*—Samples of urine (three or four ounces) suspected to contain casts should be allowed to stand at least three hours (and longer if the urine be of very low specific gravity) in perfectly clean conical glasses, and a few drops should be removed from the bottom with a pipette, and covered in the usual way on a glass slide. They may be so abundant as to form a visible sediment in the glass. For all practical purposes a  $\frac{1}{4}$ -in. objective, giving with the eye-piece a magnifying power of about 350 diameters, is sufficient. The hyaline casts are often so transparent as to escape any but the most careful observation, and then a little dilute staining fluid, introduced beneath the cover-glass, much facilitates their detection; cutting off some of the transmitted light has a similar effect. It is to be remembered that casts retain their characteristics far better in acid than in alkaline urine; in the latter they are very apt to break up and disappear. As a rule there is no mistaking a renal tubecast, but occasionally a transparent or granular streak may be noticed, the nature of which cannot be positively stated; shreds of mucus, especially when mixed up with granular matter, are the commonest objects which simulate casts, and are not infrequent in disease of the bladder or prostate; their disappearance on the application of liquor potassæ and a little heat to the slide determines their character.

*CLINICAL SIGNIFICANCE.*—Valuable as is the evidence of the changes in the renal epithelium afforded by the detection of these bodies in the urine, their recognition and comprehension is nevertheless but one of the means to be employed in the study of kidney diseases. Of themselves they afford information, rarely, if ever, conclusive when taken alone. Without doubt certain renal diseases may exist, and may continue throughout their course, either to recovery or death, without the occurrence of casts in the urine; and, on the contrary, casts may be found entirely independent of renal disease. But for all practical purposes it may be accepted that when casts do occur, they generally indicate the existence of a disease of the kidneys which is possibly incurable, certainly serious. With our present knowledge of the conditions under which they are found, it would appear that their presence in the urine does not necessarily imply the existence of an inflammatory state of the kidney, for they may accompany simple congestion, the gland substance being intact; but doubtless in the majority of cases they are evidence of a nephritis, acute or chronic, and the subsequent associated degeneration. Besides the value of casts in determining the existence of kidney-disease, they are further most important aids in helping to distinguish what

variety or stage of disease it may be, and also in making out the actual condition of the kidney, thus furnishing valuable data on which to form a prognosis, and to suggest a plan of treatment. Little is to be concluded, however, from one examination. This should be performed frequently, as in that way alone can the morbid progress in the kidney be recognised.

From what has been said of the nature of casts, it should be expected that several varieties of these bodies occur at the same time in the urine. It is rare for any variety to exist singly, at least for any time. In such cases their significance is ascertained by careful study of coincident circumstances, and especially by a frequent comparison, in order to determine which variety is in excess.

The fact that casts are very abundant in any sample of urine is not in itself necessarily a serious sign. Thus in 'granular kidney'—one of the most serious of all renal affections—the casts may be, and usually are very few, and require careful looking for; whilst in the convalescence from acute nephritis they may be extremely numerous. In chronic nephritis, however, the number becomes an important element in the consideration.

*Blood-casts* are diagnostic of hæmorrhage into the tubules, whether that be due to wounds of the organ, or to intense arterial hyperæmia or venous congestion—causing escape of blood from the capillaries of the glomeruli.

*Pus-casts* may indicate the bursting of a renal abscess into the tubes, and coagulation of the escaped pus; or a diffuse suppuration of the gland substance.

*Hyaline casts.*—The large forms of this variety chiefly occur in chronic nephritis, and are therefore usually a grave sign. Produced in tubules which have been denuded of their epithelium, or in others that have become dilated from contractions in the intertubular substance, they indicate an advanced condition of disease. Exceptions to this do occur, and large hyaline casts may be found in acute and curable cases.

Small hyaline casts are formed in both acute and chronic forms of renal disease. They therefore become valuable as means of diagnosis only in conjunction with other signs, such as the history of the case, the character of the other urinary sediments, &c. They are frequently seen in acute nephritis, particularly in the later stages of the disease, and are then formed in tubes which have not been stripped of their epithelial lining. In simple congestion of the kidney they may be formed from a coagulation of the fibrin of the effused plasma. When associated with the large variety they usually indicate a chronic and advanced stage, being then found in tubules that have become contracted. These two varieties of hyaline casts are common in

the lardaceous kidney, and similar casts have been met with in the tubules in cases of diphtheria.

*Granular casts.*—The significance of these is very variable. As has been said, the granular casts differ much in nature, and no positive diagnosis can be made upon them alone. They may occur in conjunction with blood-casts where the corpuscles have broken down, and will then generally indicate a commencing recovery from an acute stage. A similar interpretation may sometimes be put on casts whose granulation is due to fat-molecules resulting from the degeneration of inflammatory products. Large, dark, and coarsely granular casts are more particularly noticed in 'granular kidney,' where indeed they may be the only casts found. In such circumstances they become a very serious sign. In the later stages of chronic nephritis the epithelial cells disintegrate and produce granular casts.

Casts consisting of micro-organisms entangled in a basic substance have been met with in septic affections of the kidney. They are for the most part distinguishable from other 'granular casts' by their resistance to such reagents as nitric acid and liquor potassæ, which dissolve up mineral granular matter.

*Epithelial casts.*—These are more especially met with in the earlier stages of nephritis during active proliferation, and their significance much depends on the character of the epithelium cells, which may differ but slightly from the normal, or may be so altered as to resemble leucocytes in appearance, or they may be loaded with fat. They may be very abundant at first; later on, when the hyaline variety appears in the urine, becoming less numerous.

*Fatty casts.*—Probably no casts are so general in their occurrence as the fatty. Their presence may be of the gravest import, or they may betoken commencing recovery, and more than any, therefore, must they be considered in connexion with other circumstances. The casts that are found in the later stages of acute nephritis during convalescence are in part fatty. The inflammatory products undergoing this degeneration are those thrown off. Hyaline casts, both large and small, frequently present a few oil-globules on their surface. In certain forms of chronic nephritis the casts may appear as if made up of oil-globules only. In such case an advanced stage of fatty degeneration of the gland is distinctly indicated, and the persistence of fatty casts is generally taken to signify the same; though such casts have been known to continue in numbers for some weeks, and to be followed by recovery.

*Casts in non-albuminous urine.*—The occurrence of renal tube-casts in such urine is now well recognised, being chiefly met with

in cases of prolonged jaundice, lithuria, oxaluria, diabetes, and more rarely after very severe exercise. They are of the hyaline variety, and probably owe their origin to the epithelial cells rather than to the escape of any material from the blood, but their pathogenesis cannot be regarded as being understood. Their entire independence of albuminuria should not be too readily assumed, for, apart from the fact that this condition may only exist to such a very slight degree as might be overlooked, it may be that the presence of albumen is intermittent, the casts being voided in the intervals.

*Cylindroids* is a term given to certain spurious casts of a long, flattened, filamentous character which have been found in the urine of scarlet fever and cholera patients, also, it is said, more frequently in children. Whether they are renal in origin is very uncertain, and albuminuria does not always accompany them.

**II. Of the Seminal Tubules.**—Dr. Beale has pointed out the occasional existence in urine of casts containing spermatozoa (see fig. 29). The base-substance of these bodies is a viscid tenacious mucus, and they are usually much larger than the casts derived from the uriniferous tubules. They have not been found associated with inflammatory conditions of the testicle, and do not correspond pathologically to the renal casts just described.

**III. Of the Gastric Tubules.**—In inflammation of the gastric mucous membrane, especially in scarlet fever, a desquamation of the epithelial coat involving the glands has been noticed. The casts of the follicles have been found in the vomit, and more abundantly in the contents of the stomach *post mortem*. Their length is variable, and in width they range from  $\frac{1}{600}$  to  $\frac{1}{100}$  of an inch. The base-substance is described as fibrinous, and is covered more or less completely by altered epithelial cells and granular *débris*.

**IV. Of the Biliary Ducts.**—In the centre of gall-stones there have been found long, hair-like threads, often branched, and generally under  $\frac{1}{100}$  of an inch in diameter, consisting of mucus, inspissated bile, and epithelium, which have been regarded as casts of the smaller biliary tubes.

**V. Of the Cutaneous Glands.**—In the various skin-affectations which are associated with desquamation of the cuticle, casts of varying length, coming from the sweat and sebaceous glands, are thrown off as part of the general shedding of the epidermis. Such bodies are hollow tubes, and bear no resemblance to the renal casts in nephritis.

#### B.—CASTS OF HOLLOW VISCERA AND PASSAGES.

From time to time, more or less perfect casts of these organs are met with. The conditions which determine their occurrence are

but imperfectly understood; they are in most cases associated with inflammation of the surfaces from which they are thrown off, especially with those forms of inflammation accompanied by the formation of false membrane, croupous or diphtheritic. A mere catarrhal inflammation, provided the resulting mucus be sufficiently viscid and coherent, may entangle the proliferated epithelium, and be detached as more or less complete casts of the surface.

**I. Of the Alimentary Canal.**—Solid casts of the tonsillar crypts and branches consisting of inflammatory products, are occasionally expectorated in follicular tonsillitis and diphtheria.

Muco-cellular casts of several inches in length, and more or less perfect, have been met with from the œsophagus, and more rarely pieces have been detected in vomit as coming from the stomach. Casts of portions of the gullet formed of dense masses of aphthæ have also been described. More frequently seen are intestinal casts—indeed, small flocculent pieces from the surface of the colon are far from uncommon, especially in association with chronic constipation. These also consist of viscid mucus and epithelial cells, matted into tough, coherent pseudo-membranes, often forming complete hollow moulds of the tube, large or small, and varying in length from a few inches to several feet; they have been observed to be solid cylinders of jelly-like mucus and embedded cells. Occasionally they retain on their surface the impression of the follicles, sacculations, and other inequalities of the intestinal surface. See *Path. Soc. Trans.*, vols. ix. and xxiii.; *Brit. Med. Journ.* 1887, pp. 617 and 832.

**II. Of the Urinary Bladder.**—A complete exfoliation of the mucous membrane of the bladder has been occasionally observed in puerperal women. It does not appear to be always the result of inflammation, and though the detachment may be complete, perfect recovery may follow. In structure such bodies consist of epithelial cells in varying stages of degeneration, felted together by mucus and fine granular material. The surface is frequently thickly coated with urinary salts. The conditions giving rise to their formation are quite unknown, though retention of urine is associated with their occurrence.

**III. Of the Female Genital Passages.** Casts of the uterus and vagina have been frequently noticed. The latter occur as hollow moulds of the canal, more or less complete, and consisting of layers of epithelium cohering by a tough mucus. They have been known to follow astringent injections. Partial casts of the vagina formed of diphtheritic membrane have also been seen.

Uterine casts are of commoner occurrence, and are of various kinds. Exclusive of solid moulds of the cavity formed of coagulated blood, the states of pregnancy and menstrua-

tion are each liable to give rise to the formation of membranous casts of the surface, complete or partial, and usually accompanied by much pain in their extrusion (membranous dysmenorrhœa). The menstrual cast is rarely entire, and is usually about one and a-half inches long, and one-twelfth of an inch thick, of a translucent appearance, and consists of the decidua ordinarily shed at the period, but of greater consistency than normal. It may be that the cast passed at any one time is the product of a previous menstrual period. The cast expelled in extra-uterine pregnancy is much larger and thicker, appearing as a triangular bag with orifices at the angles corresponding to the openings of the Fallopian tubes and cervix; externally the bag is rough and shaggy, but on the inside it is smooth, and shows the orifices of the uterine glands.

**IV. Of the Respiratory Passages.**—The mucous membrane of the respiratory tract is remarkably prone to those forms of inflammation which are distinguished by the production of considerable quantities of new material highly coagulable in character, to which the terms croupous and diphtheritic are applied. Of such material are the different forms of casts of the air-passages composed, the products of simple catarrhal inflammation never being sufficiently coherent to retain the shape of the surface from which they are derived. In these conditions the epithelium of the mucous membrane comes to be replaced by a layer of material derived from the metamorphosed epithelial cells, with a variable amount of coagulated fibrin formed from the effused blood-plasma. Such false membrane appears under the microscope to be made up of interlacing fibres of a clear homogeneous-looking substance, felted together in all directions, and containing in the meshes leucocytes, altered epithelial cells, blood-corpuscles, and a small quantity of serum. It is easy to understand that the extent to which the materials constituting this membrane infiltrate the deeper layers of the mucous membrane must vary considerably; yet on the extent of infiltration, and consequent adherence, depends in great part the distinction between croupous and diphtheritic exudation. More important is the difference in the changes which occur in the epithelial cells in these two conditions. In the diphtheritic they are far more profoundly altered, undergoing a process of necrosis and fusion into a mass of coagulated tissue, filaments of fibrin and leucocytes infiltrating the subjacent parts. In the croupous state the epithelium, though degenerating, is far less changed, and the cells desquamate more freely. False membranes thus formed are loosened by the effusion of serum beneath them, and subsequently expelled in shreds or as entire casts.

In the larynx and trachea, the new material forms more or less complete layers, covering the vocal cords, dipping into the ventricles,

and even blocking up the laryngeal cavity completely. Occasionally a cast of the whole length of the trachea, and part of a main bronchus, may be expectorated.

When this inflammatory product originates in the bronchial tubes, it forms the so-called bronchial polypi, so characteristic of plastic bronchitis. It is rare for more than very limited areas of the air-passages to be so affected, but within these areas perfect casts of the entire extent from trachea to alveoli may be obtained. Expectorated as irregular, rolled-up, and twisted masses, they are capable of being shaken out in water into ramifying whitish or pinky white moulds of the tubes, varying in length from one and a half to six or seven inches, and of a maximum diameter of half an inch, often with swollen ends corresponding to the infundibula. They are either hollow and membranous, or solid, and in the latter case frequently present indications of being made up of concentric layers. Sometimes only one of these casts may be expectorated, especially if a large one; at other times they may be re-formed again and again, and large quantities may be coughed up daily. Microscopically they consist of altered epithelial cells, leucocytes, red corpuscles, fat-globules, and very rarely octahedral crystals, entangled in a fibrillar or granular base-substance. Similar casts of the smallest tubes are exceptionally found in the expectoration of acute croupous pneumonia, but are commonly to be found *in situ* on post-mortem examination.

W. H. ALLCHIN.

**CATALEPSY** (*κατάληψις*, a seizure).—**SYNON.**: Fr. *Catalepsie*; Ger. *Starrsucht*.

**DEFINITION.**—A disease of the nervous system, characterised by attacks of powerlessness, commonly with loss of consciousness, and accompanied by a peculiar form of muscular rigidity, in which the limbs remain for a time in the position in which they are placed by passive movement. On account of the trance-like state which exists in the attack, the name is sometimes applied to simple trance, but should be restricted to the condition in which there is the peculiar state of the muscular system. The origin of the name is connected with the old spiritualistic pathology, which referred such sudden states to the action of an immaterial agency, and recognised as such only some immaterial agent of personal nature.

**ETIOLOGY.**—Catalepsy may occur at all ages between six and sixty years, and in both sexes, but it is incomparably more frequent in the female sex and in early adult life, at or soon after puberty. It is, in the majority of cases, associated with distinct evidence of hysteria. In other cases, in which no hysterical symptoms have preceded it, the affection may be traced to such exciting causes as give rise to the hysterical paroxysm. Nervous exhaustion is the common predisponent; and

emotional disturbance, especially religious excitement, or sudden alarm, or blows on the head and back, are frequent immediate causes. It occasionally appears in the course of mental affections, especially melancholia, and is said to occur as an early symptom of epilepsy, but it is much more likely that the fits thought to be epileptic were really hysteroid. In an imperfect form it has appeared to be due, in some cases, to paludal poisoning or to other toxæmic states, as chloroform-narcosis. In a few instances meningitis, and other organic cerebral or spinal diseases, have caused a cataleptoid condition; but these cases are too rare and diverse to allow of any inference from them.

**SYMPTOMS.**—Catalepsy is usually paroxysmal, attacks lasting hours or days being separated by periods of freedom; less commonly a single prolonged attack constitutes the whole affection. In some cases headache, giddiness, or hiccough has preceded the attack. The onset of the special symptoms is usually sudden, commonly with loss of consciousness. The whole or part of the muscular system passes into a state of rigidity. The limbs remain in the position they occupied at the onset, as if petrified. The muscular rigidity is at first considerable, and movement is resisted; but after a short time the limbs can be moved, and then remain in the position in which they may be placed. The resistance to passive movement is peculiar: it is as if the limbs were made of wax, and hence the condition has been termed *flexibilitas cerea*. The rigidity commonly yields slowly to gravitation. The countenance is usually expressionless. The respiratory movements and heart's action are weakened. Substances placed in the back of the mouth are swallowed, but slowly. The state of sensibility varies; in profound conditions of catalepsy it is lost to touch, pain, and electricity, and no reflex movements can be induced even by touching the conjunctiva, a state of mental trance being associated. In other cases partial sensibility remains, and reflex phenomena may be excited. In rare instances hyperæsthesia is present. In induced catalepsy there is often a peculiar local exaltation of reflex action, so that stroking the skin over a superficial muscle causes an increased contraction of it. This involves a change of posture, in consequence of a definite sensory impression, and an analogous effect may be produced through the special senses, especially hearing; if a ticking watch be brought near the subject's ear, the posture is changed with a start, the head turned towards the object, and the eyes, if previously closed, may be opened, although the cataleptic condition continues as before. Consciousness, although usually lost, may remain, but it is seldom intact, being generally in an obscured condition. The temperature is commonly lowered

in a long seizure. The attack may last a few minutes or several hours. Recovery is gradual or sudden; sometimes the patient at first is unable to speak. A sudden respiratory stimulus—such as blowing on the face—will often terminate an attack of induced catalepsy. Sometimes a strange periodicity may be observed in the occurrence of the paroxysms. In the intervals between the attacks, headache, giddiness, or hysterical manifestations may be present, or the patient may feel and seem perfectly well.

**PATHOLOGY.**—Concerning the nature of the disease there has been much speculation, but little definite knowledge. It may probably be placed between epilepsy and hysteria in the scale of maladies, but nearer the latter than the former, and as regards the nature of its chief feature, it may be regarded as essentially one of the motor. But there is also distinct interference with the intellectual processes, and interruption of the connexion between the will and the motor centres.

To say this is only to express obvious facts in general terms, and affords no explanation of them. A careful consideration of their nature may give us at least the material for thought on the subject. The wax-like rigidity is an excess of the action by which muscles are constantly adapted to posture, *i.e.* to the distance between their attachments, and yet preserve the same degree of physiological tone. This is effected by a reflex process, the afferent impulse for which probably comes from the afferent muscle nerves, and is generated by the sum of the related tension and compression of the interstitial tissue in which these nerves begin. The motor cells (and therefore the muscular fibres) are active in inverse proportion to these impulses, and so the opponents of a passive movement relax, the muscles shorten that correspond to it in their action; they are thus adapted to posture, and, if this process is in excess, the cataleptic rigidity must of necessity ensue. But it is probable, on many grounds, that the afferent impulses from the muscles have a double action—a lower on the motor centres of the spinal cord, and a higher, through the cerebellum, on the motor centres of the cortex cerebri. The phenomena suggest that the action is similar on each; since the cortical cells govern the spinal cells, their functional states must be in related correspondence. Thus the afferent impulses that regulate adaptation to posture must do this through both cortical and spinal centres simultaneously. The excessive action of catalepsy may be in the cortical and not in the spinal mechanism. This would enable us to understand the otherwise strange fact, that the cataleptic state may be induced by influences acting on the brain, as in the induced catalepsy of hysteria. The motor cortical centres, whence the pyramidal fibres proceed, are probably the lowest of the centres

of the cortex; the induction of catalepsy may consist simply in the inhibition of higher centres that act on and through them and normally restrain them. The over-action of these entails the over-action of the spinal centres, and the wax-like rigidity results. Thus, too, we can understand that consciousness should be lost or strangely changed during the attack, and its relation to emotion as a cause. So, too, it is clear that every forced change of posture will leave the condition of the centres essentially unchanged, although there will be a slightly different distribution of activity. Arrest the inhibition which prevents the higher cortical centre from acting on the lower, and the resumed control involves restraint of the over-action of the latter and of the spinal centre, and the cataleptic state is at an end.

**DIAGNOSIS.**—The peculiar rigidity of catalepsy is characteristic, invariable, and renders the diagnosis a simple matter. Hysteria with tonic spasm has been erroneously termed catalepsy, but is distinguished by the local position of the spasm, and the fact that it does not yield to passive force. The malady most likely to be confounded with catalepsy is tetany, since in it there are attacks of tonic spasm involving all the limbs. But in this the rigidity is fixed; it cannot be overcome, and the posture of the limbs changed; while the hands are in a characteristic posture, with the fingers straightened at the middle joints, and their points and that of the thumb brought together. The condition is sometimes simulated: in true catalepsy the rigid limb slowly yields to the influence of gravitation, and more rapidly if a weight be attached to it; in the feigned form the limb and weight are held firm.

**PROGNOSIS.**—The prognosis is favourable in simple catalepsy, in proportion to the freedom of the intervals from affections of sensibility or motion. In pronounced hysteria and psychical affections the condition is often obstinate; and, by interfering with the due nourishment of the system, may cause grave inanition.

**TREATMENT.**—During the attack itself little can be done save an attempt, which may be repeated at intervals, to rouse consciousness by external stimulation. The ordinary applications—ammonia to the nostrils, cold douches, &c.—often fail in the spontaneous form, but this does not hold true of most cases of induced catalepsy, in which any stimulus that excites a respiratory effort commonly arouses the patient and cuts short the attack. A pinch of snuff will, however, often succeed in both varieties. Another effectual stimulant is faradisation. It may be applied to a limb or to the cervical spine. The current should be gentle at first, and gradually increased. Emetics are also useful in arresting an attack. Injections of tartar emetic into the veins have been used

with success by Calvi, but can hardly be recommended. The writer has found subcutaneous injection of apomorphine,  $\frac{1}{2}$  to  $\frac{1}{12}$  of a grain, an efficient remedy for similar paroxysmal conditions; with the onset of nausea, about five minutes after the injection, consciousness is regained, and all spasm ceases. In the intervals between the attacks the treatment is that of hysteria. Iron, antispasmodics, especially valerian, aloetic aperients, and cold baths, are the most effectual measures. Firm moral treatment is also indispensable. Removal from home influences is often necessary to effect a cure.

W. R. GOWERS.

**CATAMENIA, Disorders of.**—*See* MENSTRUATION, Disorders of.

**CATAPLASM** (κατὰ, down; and πλάσσω, I mould or smear).—A synonym for a poultice. *See* POULTICE.

**CATARACT.**—**DERIVATION AND SYNONYMS.**—Earlier Greek: γλαύκωσις (Hippoc.), γλαύκωμα (Aristot.); Later: ὑπόχυμα and ὑπόχυσσις; Latin: *Suffusio* (Celsus); Arabic: *clmâ ennâzil* (descended water or humour); Mediæval Latin translators, *Aqua in oculum descendens*; *Descensus Aquæ*; also *Cataracta* (allied to Greek *καταράκτης*), used at first in the sense of an obstruction or obstacle, like a floodgate, a portcullis, an obscuration such as that of the sun by a cloud. Fr. *Cataracte*; Ger. *Staar*.

**DEFINITION.**—Cataract is an opacity, partial or complete, of the substance of the crystalline lens, or of its capsule. The former is called *lenticular*, the latter *capsular*. Either may exist separately, or the two may occur together, to form *capsulo-lenticular*. From a surgical point of view it is sometimes necessary to embrace within the definition of cataract some extra-capsular opacities, such as inflammatory deposits, or the remains of embryonic structures within the pupillary area of the anterior or of the posterior layer of the capsule. Such opacities are called *false cataracts*.

**ANATOMY AND PHYSIOLOGY.**—The nature and origin of cataract can only be made intelligible by reference to the anatomy, embryology, and physiology of the crystalline lens.

**Anatomy.**—The lens consists of three parts: first, the substance of the lens, which is made up of fine, flattened fibres; second, a layer of endothelium lining the anterior part of the capsule; and, third, the capsule, which, as a bag, surrounds and encloses the lens-substance and the endothelium. The fibrous portion of the lens consists of a harder, central part, or *nucleus*, and a softer external portion, or *cortex*. The cortex is disposed around the nucleus in thin, concentric layers, each of which is subdivided into sectors. Within the sectors the fibres lie with their

ends at the edges of the sectors, and each sector has two of its edges towards the anterior, and two towards the posterior surfaces of the lens. The apposition of the edges of the adjoining sectors leaves fine radiating fissures, which make up the rays of the stella existing at the anterior as well as at the posterior surface of the lens. The fibres of the lens are softest at their ends, where they abut on the rays of the stella. The endothelium consists of a single layer of nucleated and nucleolated cells, and is usually limited to the anterior capsule. Towards the margin or *equator* of the lens these cells accumulate, elongate, and become gradually transformed into lens-fibres. Fine channels, occupied by lymph and other fluids, exist between the endothelium and the capsule, and between the endothelium and the anterior layers of the cortex, passing thence through the rays of the stella into the substance of the lens and between its several layers. The capsule, which is elastic and transparent, consists of two layers, a delicate inner one and a stronger outer one, which is further strengthened by prolongations of the suspensory ligament—the zonula of Zinn.

*Embryology.*—The ancestry and mode of origin of the several parts of the lens determine the peculiarities not only of the normal nutrition of the lens, but also of its pathological departures. The lens-fibres and the endothelium are descended from the external layer of the blastoderm—the epiblast, and are, therefore, immediately related to the cuticular tissues, including the enamel of the teeth and the epithelium of the sense-organs, and more remotely to the cerebro-spinal system. As will be seen later, this common origin from a primitive stock of epiblastic elements throws light upon many of the pathological associations of congenital and other cataracts. According to recent researches, the capsule of the lens seems to have a double origin, the inner layer being derived by a process of cuticularisation from the lens-substance, while the outer is the offspring of the mesoblastic elements from which the foetal capsulo-pupillary sac, the vitreous, choroid, and intra-ocular blood-vessels are derived. Hereby the capsule is in direct histogenic relations with the bones, muscles, blood-vessels, and other fibro-vascular tissues derived from the middle layer of the blastoderm. These relations may also determine pathologicæ manifestations in the capsule, and through its intermedium within the lens-substance itself.

*Growth.*—The foetal crystalline lens is somewhat globular in shape, and its fibres have an antero-posterior disposition. But during later intra-uterine life, and still more after birth, the lens alters its shape, becoming broader and relatively flatter. It grows by the apposition of lens-fibres upon the nucleus, chiefly about the equator. The fibres themselves are derived from the endothelial cells

lining the anterior capsule. These cells proliferate and gradually shift towards the equator, where they accumulate, and, as already stated, become transformed into fibres. The fibres bend round the equator, and spread out in front and behind, so as gradually to encase and push towards the centre the older underlying layers. The growth of the lens continues throughout life, and does not, in health, cease with the attainment of adult life. Priestley Smith has found that the volume of the lens increases through life at the rate of about 1.5 cubic mm. each year (*Trans. Ophthalm. Soc.*, vol. iii. p. 79).

*Nutrition.*—The crystalline lens, being a non-vascular body, is nourished by imbibition from surrounding fluids, which are chiefly furnished by the ciliary body, though this source is supplemented by contributions from the choroid, the iris, and perhaps the retina. It is probable that these fluids are modified by their passage through the vitreous. It is not known exactly how or where these fluids gain access to the lens-substance, but it is evident that they pass through the capsule, and that in the passage they undergo some alteration; for if the anterior or the posterior capsule be ruptured, the lens becomes opaque. The surrounding fluids then come into immediate contact with the fibres of the lens, and being unmodified by the dialytic properties of the capsule, or the elaborating influences of the endothelium, they are unsuited for the purposes of normal nutrition. In health, after the nutritive fluids have made their way through the capsule, they pass from the periphery to the centre through the stellate clefts and other channels which exist throughout the lens. In this respect the lens differs from other cuticular structures, in which waste products are shed from the surface; while in the lens waste products are apparently driven inwards to the centre, to undergo elimination in a manner not yet fully understood.

The nutritive activity of the lens, as manifested by the number of nuclei, is apparently greatest at the equator, and it is at this point, between the layers of the suspensory ligament, that the capsule is in most immediate contact with the ambient fluids. Many authorities believe that the nutritive fluids enter the lens chiefly, if not entirely, at this place, and that this region is the principal seat of cell-growth. There are, however, reasons for thinking that neither endosmosis nor cell-proliferation is most active here. In some diseases of the choroid or of the retina on the one hand, and of the iris or of the cornea on the other, opacities of the lens first manifest themselves not at the equator, but at the posterior or the anterior surface respectively, that is at that part of the lens which is *nearest* to the diseased structures, and which bears the brunt of the morbidly

altered fluids. A typical example is the formation of posterior polar or stellar cataract in retinitis pigmentosa. Moreover, microscopical examinations of cataractous lenses often disclose proliferation and accumulation of nuclei behind the anterior capsule, and even within the posterior capsule, without corresponding increase at the equator.

Beyond increasing in size, the lens does not, in health, undergo much change till after middle life. Thenceforth the fibres gradually assume a yellowish or brownish tint, and the rate of formation of new fibres diminishes. As age advances the fibres become drier, harder, and less elastic, and the capsule thickens. If a senile lens be ophthalmoscopically examined with a strong magnifying-glass, semi-translucent lines, flaws, spaces, and vacuoles, and fine, dusty films, may often be discovered, even when there are no coarse physical opacities. These phenomena are due to shrinking of the lens-fibres and loosening of the lamellæ from one another, with accumulation of fluid and lens-débris in the spaces thus formed. Still later, separation of the cortex may take place, both from the nucleus and from the capsule. The separation from the capsule is, as Becker has shown, especially liable to occur at that part of the capsule which is most acted upon by the suspensory ligament during accommodation. Increase and extension of the changes eventuate in opacity, and the occurrence of senile cataract. These senile changes have their analogues in the skin and its appendages. The skin of old people is wrinkled, the cuticle becomes dry and harsh and shrivelled, and the hair thin and white.

**PATHOLOGY.**—A lens of good ancestry and of good constitution continues to be transparent so long as the conditions and processes of nutrition are normal; but any fault in the original constitution of the lens, or any defect in the embryonic process, or any alteration in its nutrition at any subsequent period, will, sooner or later, and in proportion to the degree of divergence from the healthy standard, induce opacity of some portion of the lens or of its capsule.

The chief *embryonic* abnormalities are (1) inherent faults of the epiblastic cells which constitute the rudimentary lens. The fibro-cellular portions of the lens fail to undergo proper evolution, and either do not become transparent, or, if they do clear, their potentiality is low, and they soon retrograde. The lens is often undergrown as well as opaque, and is not infrequently malposed. Sometimes the eyeball itself is small (*microphthalmia*). Even if the lens do attain its natural proportions, the new cortical substance is generally of inferior quality, because the endothelium, which furnishes the cortical substance, shares the same faults as the nucleus itself. Hence the whole lens is either opaque from the beginning (*congenital cata-*

*ract*), or becomes opaque within a few months or years (*infantile* or *juvenile cataract*). (2) Owing to causes which may be peculiar to the lens, or which may affect the whole of the growing organism, or which may operate indirectly through so-called maternal influences, a temporary or permanent disturbance may occur during intra-uterine life to thwart the development of a lens. If the interference be violent its effects may be permanent, so that the whole of the lens becomes opaque, even though it may continue to increase in bulk. If the check be of a temporary character, only a thin layer of opacity may be produced: natural development re-asserts itself, and transparent cortical fibres are produced in the ordinary way. The nucleus of the lens, though not unaltered, is usually translucent and the cortex transparent; but the intermediate layers—that is, the sub-cortical or perinuclear—are more or less opaque. This form of opacity is called *lamellar* cataract, because in sagittal section it is seen to be disposed in layers around the nucleus; or *zonular* cataract, because the opaque portions, when looked at *en face*, are bordered by a transparent zone of clear cortex. It would seem that the layers which are opaque are either those which were immediately sub-capsular at the time when the disturbance took place, or those which were produced by the endothelial cells during the period of disturbance. On this assumption the position of the opacity may be regarded as indicating the time at which the interruption took place. The earlier the interference the nearer will the opacity be to the nucleus of the lens, and *vice versâ*. In some rare cases there are two or more concentric layers of opacity, with intervening layers of clear substance, as if there had been several disturbances at different epochs, followed by intervals of normal growth. Lamellar cataracts may be found in eyes otherwise healthy, but they are sometimes associated with developmental defects of the eye and of the general organism, an almost invariable concomitant being deficiency of enamel of the permanent teeth, especially the first molars, the incisors, and the canines (Hutchinson), and not infrequently there is a clinical history of 'fits,' or other evidences of a neurosis. (3) As the result of inflammation of the cornea, uveal tract, or vitreous during intra-uterine life, single or multiple opacities may form in the capsule and sub-capsular layer. These are called *congenital capsular* cataracts, and are most common at the anterior pole. They may, however, be scattered about the anterior surface, or lie about the posterior pole of the lens. These opacities consist of proliferation of the endothelium and granular disintegration of the superficial cortical layers, similar to those changes which produce capsular and sub-capsular opacities after birth. It is,

however, not improbable that congenital anterior polar cataracts may sometimes be due, not to inflammation, but to retarded separation of the involuted lens-vesicle from the cornea, and delayed closure of the vesicle. This retardation may permit of the intrusion of mesoblastic elements within the vesicle, and these would still further increase the opacity about the capsule. This may conceivably cause an axial opacity, the so-called *spindle-shaped* cataract. (4) *Congenital false* cataracts may be due to the presence of extra-capsular opacities upon the anterior or the posterior pupillary areas. These opacities may be the results of inflammatory exudations, but more commonly they are due to an arrest of development in its final stage. The development and growth of the lens may have been normal up to the stage at which the removal of the embryonic capsulo-pupillary sac should have taken place, but, having ceased immediately before the removal was complete, portions of the foetal structures remain. When these are on the anterior surface, they are connected with the edge of the iris, or more frequently with its anterior surface; when on the posterior surface of the lens, the remnant may be connected with a fine thread which is itself the remains of the hyaloid blood-vessels. This state of incompleteness may be designated by the Aristotelian term *Ateleia* (*Physic*, viii. 9; *De Animal. Gen.* iii. 9).

The alterations in nutrition which, *after birth*, lead to the formation of cataract may be classified as follows:—

(1) *Diminution in the quantity* of the nutrient elements, without material alteration in their quality. This diminution may be due to many causes: (a) mechanical obstacles impeding due access of the fluids; (b) damage to the secretory apparatus consequent upon chronic atrophy of the ciliary body and choroid; (c) general malnutrition, cachexia, anæmia, cardiac, renal, or other visceral disease; or (d) simple old age.

Where diminution of the quantity of nutritive elements operates slowly on the nutrition of the lens, and the opacity which results manifests itself in or about the nucleus—that is to say, the parts most remote from the sources of the nutrient fluids. The cortex seems to receive enough pabulum to keep it clear, but not enough to spare for the support of the deeper layers.

(2) *Alterations in the quality* of the nutrient elements. These may be due to: (a) congestion, inflammation, or any other morbid state of the secretory organs of the nutritive fluids—namely, the ciliary body, iris, choroid, and retina; (b) changes in the vitreous or in the aqueous humour; (c) morbid states of the blood and tissues, as in many general and diathetic diseases—gout, glycosuria, albuminuria, &c.; (d) changes in the physical, chemical, or vital properties of

the capsule or of its endothelium, in consequence of which the fluids that pass through the capsule are not properly prepared for the due nutrition of the lens; or (e) impeded elimination of waste products, whether from the lens itself or from the whole eyeball.

Alterations in the quality of the nutrient elements are more or less prompt—and if the alteration be very great, as, say, in rupture of the capsule—they are very prompt in producing opacities. The opacities which result affect primarily the sub-capsular and the cortical layers, more particularly at the ends of the fibres adjoining the rays of the crystalline stellæ where the endosmotic processes are most active, and where the effect of the altered quality of the fluids is first apparent. The affected parts are those with which the altered fluids first come into contact, and which lie therefore nearest to the sources of the altered fluid. If the alteration be intense, but of brief duration, as in the case of perforating ulcer of the cornea in purulent ophthalmia of children, the change may be limited to a circumscribed area of the capsule and sub-capsular layers, giving rise to an *anterior polar* cataract, or, if this opacity assume a conical shape, *anterior pyramidal* cataract. In alterations of less intensity, but of continuous duration, due to chronic morbid states in the posterior segment of the globe, such as retinitis pigmentosa, slowly forming opacities may occur in the posterior sub-capsular layers of the lens, beginning at the pole of the lens, and extending thence in a radial direction along the rays of the posterior stella, giving rise to *posterior polar* or *stellar* cataracts.

(3) *Alterations of quantity and quality combined*. This co-operation obtains in most of the cases of cataract; diminution of quantity being the chief factor in some, alterations in quality predominating in others. Where the departure from the normal approaches most closely to mere diminution in quantity, the onset and progress of the cataract is slower, and the opacity tends to affect at first the parts nearer the centre of the lens; but where the alteration in quality is pronounced, the onset and progress are more rapid, and the opacity begins in the cortex. It will easily be understood that the great majority of cataracts are cortical, because there are comparatively very few cases, even in apparently healthy eyes, where the alteration in nutrition does not exhibit some difference in the quality of the nutrient elements, as well as falling off in quantity.

MORBID ANATOMY.—The changes which take place in cataract are not the same in all cases. There is, for example, a great difference between the changes which produce some embryonic cataracts and those which give rise to acquired cataracts, and between those which produce lenticular cataract and

those which give rise to capsular cataract. Indeed capsular opacities are often secondary to lenticular. Even in true lenticular cataract the morbid changes are many and various. In senile cataracts, and in most slowly forming cataracts, the opacity of the lens-fibres is merely an extension of physiological senile changes, and is usually preceded by the formation of spaces and vacuoles between the fibres and the lamellæ due to shrinkage and sclerosis, as already described. Priestley Smith's investigations have shown that a diminished rate of growth of the lens precedes cataract (*op. cit.*). According to Otto Becker the initial cause of senile cataract is the drying of the nucleus and its separation from the cortex, the gap between them being filled by a fluid which is probably derived from the fibres. In more rapidly forming cortical cataracts it may often be observed that the opacity begins in the sub-capsular layers and about the borders of the rays of the anterior stella. Instead of shrinkage and sclerosis, the fibres may swell and become softer. In some cases the rays of the stella are broadened, but whether they are or not, a hazy line may often be traced along one or both of the edges of the ray, in the ends of the cortical fibres. From the ends of the fibres the opacity extends along their lengths towards the equator, until the whole of the cortex is implicated. The endothelium also undergoes changes, giving rise to triangular patches of glistening opacities which may present a mother-of-pearl appearance. When once the opacifying process begins in any part of the lens it tends to progress till the whole of the lens is involved. The cataract is then said to be *ripe* or *mature*.

If a ripe cataract be allowed to remain within the eye indefinitely, it may undergo further degenerative changes and become what is called *over-ripe*. In some instances the lens-substance shrinks and becomes hard and calcareous, and the capsule thickens and grows more opaque. In other cases the cortex liquefies, allowing the harder nucleus to move within it (*Morgagnian* cataract). In calcification of the lens the process begins in the external cortical layers, and it may ultimately affect the entire thickness of the lens. Calcification is more liable to occur in cataracts due to gross disease of the eye, and especially when there is an inflammatory exudation upon the capsule. Short of general calcification, minute white dots, lines, and patches may appear in the capsular endothelium of mature cataracts. Over-ripe cataracts may also become loosened, in consequence of softening or rupture of the suspensory ligament. Fluidity of the surrounding vitreous may likewise occur, and then on slight provocation the loosened lens may fall into the vitreous.

A peculiar form of degeneration sometimes occurs in the lens, producing what is called

*black* cataract. This is commonly thought to be due to the staining of the lens by blood-pigments, and in some cases this may be so; but, according to a careful spectroscopic examination by Dr. McMunn, no blood-pigments were found, but the lens was infiltrated with a pigment analogous to melanin, which is the pigment of hair and feathers (*Trans. Ophth. Soc.* vol. ii. p. 10).

**ÆTIOLOGY.**—Besides the causes and conditions already described, there are other circumstances which may induce cataract, or at least predispose to it. The most important of these are as follows:—

**Age.**—Cataracts occur more frequently in infancy and in old age than in early adult and middle life, except when due to disease of the eye or to injuries. **Sex.**—The two sexes are about equally affected, except in respect of traumatic cataract, in which males greatly preponderate. **Heredity.**—Hereditary influences may determine the occurrence of cataract in several ways. There may be the inheritance of actual (congenital) cataract or of a disposition to cataract. In either case the descent seems to pass more through the male than the female line. **Occupation.**—Certain occupations predispose to cataract, especially such as necessitate close application of the eyes to near work, or involve stooping positions or exposure to bright light, more particularly if light be combined with heat (as in the occupation of smiths, cooks, stokers, &c.), and occupations in which the eyes are exposed to irritating fumes and vapours. Uncorrected *errors of refraction* and accommodation may favour the production of cataract by overstraining the accommodation, and by the frequent and successive changes which the capsule has to undergo. These strains not only increase the wear and tear of the lens, but facilitate the loosening of the cortex of the lens from the capsule. Hypermetropia and astigmatism may predispose to cataract in another way. The incessant strain upon the ciliary muscle required to correct hypermetropia involves constant congestion of the ciliary body and the base of the iris. The nutritive fluids secreted during this state are not perfectly normal; they are in some measure altered in quality and will therefore sooner or later make their influence felt on the transparency of the lens. **Injuries.**—Wounds of the capsule are generally soon followed by opacity, which occurs more quickly in proportion to the size of the rent and the youth of the patient. Perforating wounds of the cornea, especially when followed by suppuration, may induce cataract even though the capsule have not been perforated. Blows on the head, face, or brow, shocks and jars of the body, may also be followed by cataract, either from causing flaws or cracks or rents in the capsule or by rupture of the suspensory ligament. Other conditions which accompany and apparently pre-

dispose to cataract are degenerative changes in the blood-vessels, especially of those of the head and neck and of the eye; diseases of the orbit, or changes in the orbital vessels; cardiac disease, renal disease, gout, and diabetes. It is probable, however, that the influence of diabetes in the production of cataract has been overrated. The number of diabetic persons who get cataract is comparatively small, and when cataract coexists with glycosuria there is no necessary causal relationship between the two. It has been stated that ergotism often leads to cataract (J. Meyer).

CLASSIFICATION. — Cataracts have been classified in many ways, as (1) according to *age*—congenital, juvenile, senile; (2) to *stage*—incipient, progressive, stationary, immature, mature; (3) *consistency*—fluid, soft, hard, and mixed.

All these plans possess some clinical utility, but they are not sufficiently comprehensive. Following the indications afforded by the consideration of the pathology and causation of lenticular opacities, cataracts may be arranged into five main groups according (1) as they are due to developmental abnormalities—*embryonic*; (2) as they occur independently of obvious disease of the eye—*idiopathic*; (3) as they are associated with obvious disease of the eye—*sympathetic*; (4) as they follow wounds or injuries of the eye—*traumatic*; and lastly (5) as they are due to the opacification of lens-matter remaining within or on the capsule at the time of operation for the removal of cataract, or of lens-matter which may have grown subsequently and then become opaque—*deutero-pathic* or subsequent (*δευτεροπαθία*, I suffer later.—Galen, *De Loc. Affect.* i., iii. ed. Kühn viii. 31). Within one or other of these groups all the varieties of cataract may be arranged according as they implicate (1) the nucleus; (2) the perinuclear region (lamellar); (3) the axis (spindle-shaped); (4) the cortex, anterior or posterior; (5) the equator; (6) the capsule, anterior or posterior; (7) the external surface of the capsule—extra-capsular—which may be congenital or acquired.

SYMPTOMS.—The chief subjective symptom of senile and other acquired cataracts is impairment and 'fogginess' of sight, especially for distant objects. As the cataract advances, this dimness increases, until the appreciation of form and colour may be lost; but, in eyes otherwise healthy, the power of discerning light always remains, even though the lens be quite opaque. In ordinary daylight the eye may still be able to 'count fingers' at a distance of six to twelve inches, or see the movement of, say, a hand at a distance of twelve to eighteen inches (hand-movement); and in a darkened room the eye should be able not only to see a lighted candle at a distance of ten to fifteen feet, but should be able also quickly to indicate its position in various parts of the field of vision without

movement of the eyes or head. This would indicate that there is good *perception* of light, good *projection*, and a good *field of vision*. In the early stages of cataract, especially when the opacity occupies the pupillary area, patients may see better when the eyes are shaded, the pupil being thereby made to dilate; a bright light embarrasses and distresses them. *Muscæ volitantes* are also often seen; these may be due to the congestion of the ciliary body and choroid which may have preceded cataract, or they may be the results of the straining efforts to see through a hazy lens. Another common symptom is the multiplying or splitting of objects, especially of lights (mon-ophthalmic polyopia). The amount of impairment of sight is not always in proportion to the amount of appreciable opacity; diffused haziness about the nucleus or about the pupillary area is more hampering than well-defined striæ or sectors with clear interspaces. There may be dense opacities at the equator with perfect acuteness of vision. An occasional early symptom of cataract is a change in the refraction of the eye, leading to what some persons describe as getting 'the second sight.' Eyes that have required strong convex glasses for near work prefer weaker ones, or none at all; and those who have needed convex glasses for distance may find that they see better without them, or even with concave glasses. This is a passing state, though it may last for weeks and even for months; it is due either to the swelling of the lens or to some change in its refractive index. After a time this condition passes off and is followed by progressive failure of sight. Many children with zonular cataract are thought to be merely short-sighted, and often they are short-sighted, but the true cause of the defect is frequently not discovered till the 'optician' has failed to get good vision by means of spectacles.

DIAGNOSIS.—In all cases of failing sight, whether opacities are to be seen or not by means of direct light or by so-called focal or oblique illumination, the media of the eye should be examined with the ophthalmoscope. Owing to thickening of the lens-capsule, the pupils of old persons are sometimes grey instead of black, and the lenses may seem to be opaque when they are not really so; and in glaucomatous eyes the change of colour which takes place in the cornea and in the lens not infrequently suggests cataract even when the media are translucent. Diagnosis of advanced cataract is easy, but in slighter cases careful examination of all parts of the lens may be needed. The aims of diagnosis are to recognise the presence of the cataract, to ascertain its seat, extent, consistence, and its relations or complications, and to discriminate it from other morbid states which may be mistaken for it. Examination of the anterior part of the

lens, the capsule, anterior cortex, and the nucleus may be made with 'focal illumination,' that is concentrating light upon the eye by means of a magnifying-glass. The equator of the lens and the posterior cortex require the ophthalmoscope for their examination. Opacities which seem white or greyish by focal illumination appear black against the red ground of the fundus-reflex when looked at with the ophthalmoscope. The different parts of the lens should be examined methodically, beginning with the anterior capsule and passing through the thickness of the lens to the posterior capsule.

Anterior extra-capsular opacities, whether inflammatory deposits or remains of the capsulo-pupillary membrane, project beyond the level of the capsule. The latter have usually attachments to the anterior surface of the iris (*circulus minor*), whereby they are distinguished from inflammatory adhesions (posterior synechiæ) which are connected with the edge and posterior surface of the iris. Capsular cataracts may be single or multiple. Anterior polar cataract occurs as a small circumscribed dot in and behind the centre of the capsule; when viewed in profile the dot can be seen to project slightly beyond the general level of the capsule; occasionally it projects considerably and is conical (pyramidal cataract). Both these cataracts are generally associated with nebula or leucoma of the cornea. Multiple capsular opacities may be scattered irregularly or arrayed in a circle. Cortical opacities assume many shapes—striae, wedges, or irregular patches. When the opacities are in the anterior cortex, they are convex in their arrangement; when in the posterior they are concave. Equatorial opacities may be partial or complete, continuous or interrupted, limited as a corona to the periphery, or send tapering offshoots towards the anterior and posterior cortices. Perinuclear opacities may be circumscribed or diffused. The circumscribed (lamellar or zonular cataracts) are best seen with the ophthalmoscope when the pupil has been dilated by a weak solution of atropine or other mydriatic. Lamellar cataract appears as a greyish disk surrounded by a clear zone. If the nucleus be translucent the disk is most opaque at its edge; but if the nucleus be itself opaque, then the opacity is densest in the centre. The edge of the disk may be sharply defined, or indented or broken by centrifugal striae. Nuclear opacities may be dense and diffuse, as in some congenital cataracts, or they may appear as a brownish or yellowish undefined blur or cloud, as in early senile cataract. Posterior polar cataract can be properly seen only with the ophthalmoscope. It may assume the form of a smaller or larger dot, or of several dots, or of radiating streaks. The position of the opacity is determined partly by its remoteness from the pupil, and partly by the small excursions it makes in the

various movements of the eye, the posterior pole of the lens being immediately in front of the centre of rotation of the globe. Posterior polar cataract may be congenital, but more commonly it is pathological and associated with opacities in the vitreous or with disease of the choroid or retina.

Whenever practicable, it is desirable in all forms of cataract to examine the state of the optic nerve and fundus of the eye. The data thereby obtained may influence the prognosis and the later treatment. When the cataract is mature the pupil is uniformly white or greyish, and the opacity comes up to and seems to touch the pupillary edge of the iris, and no reflex can be obtained by means of the ophthalmoscope. If the cortical layers are not opaque, a shadow of the iris can be thrown into the lens by means of oblique illumination, giving the appearance of a gap between the opacity and the edge of the iris. For surgical purposes it is not necessary that the whole of the lens-substance should be opaque; it is enough that the cortical layers be opaque, for then the connexion between the cortex and the capsule is broken, and the endothelial cells have lost their power of growing fresh lens-fibres. It is not always possible to estimate the consistency of a cataract; but, as a rule, cataracts which begin in the cortex, or occur in persons under the age of thirty years, or are rapidly formed, are usually 'soft'; such cataracts are generally whiter and more opaque than harder ones, and the opacity is often patchy or diffused. Nuclear cataracts, and those which occur in old persons, or are of slow formation, or yellowish in colour, or in which the lenses are small and shrunken, or which have small, well-defined opaque striae with clear interspaces, are usually 'hard.'

The failing sight of chronic glaucoma is sometimes ascribed to cataract. This mistake is fostered by a superficial resemblance between these two conditions. By the ophthalmoscope and by palpation, the error may easily be avoided. When the lens is not opaque in glaucoma the diagnosis is simple, mere absence of opacity excluding cataract; but when, in glaucoma, the lens is also opaque, the diagnosis may present some difficulty. In chronic glaucoma the characteristic symptom is increased hardness of the eyeball. Usually a history of haloes seen round artificial light may be elicited, with perhaps attacks of neuralgia and redness of the eye. The pupils are semi-dilated and very sluggish, if not immobile, and the anterior chamber is shallow, and the sight is more impaired than the cataract accounts for. In advanced (absolute) glaucoma there is an entire abolition of the perception of light.

PROGNOSIS.—Cataract may be considered, as regards its prognosis, from two points of view: first, when it is immature, as to its probable course; and, secondly, when it is

mature, as to the prospects of recovery of sight after operation. Some cataracts may remain stationary throughout a long life: such are anterior polar, anterior pyramidal, and zonular cataracts. Others progress slowly, such as posterior polar cataracts. Cortical cataracts, as a rule, advance more rapidly than nuclear ones, and soft cataracts more rapidly than hard. It is not possible in all cases to decide whether a cataract is likely to progress slowly or quickly. Generally the progress is quicker the earlier the cataract occurs, and the closer its association with morbid states of the eye or with injury. When cataracts are sharply defined, confined to the equator of the lens, or present only a haze or blur in or about the nucleus, or when the cortical striæ are few and fine and well-defined, with clear interspaces, and the eyes are otherwise healthy, and the sight good, the progress will usually be slow; but when the opacities are dense, irregular, ill-defined, diffuse, especially where they extend far along the anterior or posterior cortical layers, with rapidly failing sight, or with sight bad out of all proportion to the amount of actual opacity, or when there is obvious irritative or inflammatory disease of the eye, or when there is grave visceral or other constitutional disease, such as gout, diabetes, nephritis, then the progress will probably be rapid. But it should be remembered that cataracts which may have been very slowly progressing may at any time take a sudden and rapid start and progress quickly. The usual course, in senile cataract, is from one to four years or more.

As regards *mature* cataract, the prognosis in reference to operation is good in proportion as the eye is otherwise sound, and the general health good. Where there is a history of attacks of pain or of redness in the eye, or where there are signs of past disease, whether iritis or choroiditis or retinitis, or where there is a high degree of myopia, or where the iris is discoloured and lustreless, and sluggish or immobile, or where it is tremulous, or where the anterior chamber is much deeper than natural or much shallower, or where there is a chronic conjunctivitis, ciliary blepharitis, or catarrh of the lacrimal sac, or where the tension of the eye is too high or too low, the prognosis is more or less unfavourable. Nystagmus, squint—especially divergent squint—are unfavourable concomitants; and the prognosis is, of course, less favourable when it is known that there was antecedent disease of the fundus, or when, failing this knowledge, the perception of light is poor, and the projection bad, and the field of vision contracted or irregular. Where there is no perception of light, the prognosis is hopeless.

**TREATMENT.**—From a medical standpoint, the treatment of cataract is rather preventive and palliative than operative.

**Prevention.**—From a consideration of the

causes and pathology of cataract, it is evident that, while some cases are beyond the reach of prophylactics, there are others which do fall within the category of preventible diseases. In the present state of knowledge and therapeutical art, little can be done, directly, to prevent many of the embryonic forms of cataract, and those other cataracts which are due to simple diminution of the *quantity* of nutrition, of which pure senile cataract is the type. Our control over the processes of development and of senile decay is, at best, slight and limited. On the other hand, it is at least ideally possible to prevent the occurrence of many of those cataracts which are due to alterations of *quality* of nutrition. By the prevention or the avoidance of those general and local conditions and morbid states which induce these qualitative alterations, or by their early and effectual treatment, *secundum artem*, by obedience to the laws of personal and public hygiene, by conforming to the requirements of healthy sight, by the scientific correction of errors of refraction and accommodation, by adequate protection of the eyes during those occupations which entail risks of physical, thermic, or chemical injuries, cataract may often be prevented, or its progress arrested, if perchance initial changes have already begun. For the prevention of cataract, as well as in its general treatment, the twofold origin of the lens-substance and its capsule should be borne in mind. The fibres and the endothelium of the lens, and the inner layer of the capsule, being epiblastic in origin, are in intimate relationship with the central nervous system, the cuticle, hair, nails, enamel of the teeth, and other tissues derived from the external layer of the blastoderm, and are, therefore, apt to participate in many of their pathological states. Similarly, by its mesoblastic origin, the external layer of the capsule is directly related to the bones, muscles, blood-vessels, and other fibro-vascular tissues, and is liable to their affections. In these affections the lens-fibres and the endothelium may become secondarily involved. Whatever promotes the nutrition, or averts breakdown, or arrests or cures disease, of one portion of these groups of tissue-elements, will, presumably, in some degree influence other portions. Hence general principles are capable of a particular application to the several parts of the lens and its capsule. It should, however, be borne in mind that though the epiblastic and the mesoblastic portions of the lens have their proper constitution and peculiar modes of activity, they are nourished by fluids chiefly, if not exclusively, furnished by the ciliary body and other parts of the uveal tract.

**Palliation.**—If opacity of the lens exists, but not to an amount sufficient to materially impair sight, it is sometimes possible, by general and local measures, to improve the special nutrition of the lens, and thereby to

retard the progress of the cataract. In some cases, where slight endothelial and cortical opacities occur during transitory inflammation of the cornea, iris, or choroid, or during exacerbations of diabetes and other diathetic diseases, the opacities may gradually disappear on the resumption of normal nutrition after the subsidence of the inflammation or the melioration of the general health. But, except within these narrow limits, genuine cataractous opacities are incurable and irremovable by any means known to positive science other than operation. Notwithstanding the advancement of medical knowledge and the increased resources of surgical art, the earliest dictum recorded in medical literature respecting the curability of cataract still holds good. Nearly nineteen hundred years ago Celsus remarked: 'Cum [suffusio] recens incidit, medicamentis quoque sæpe discutitur; sed ubi vetustior facta est, manus curationem desiderat' (*De Medicina*, vii. 7, 13). Increased knowledge of the nature of cataract lessens rather than increases the hope and expectation of dispersing the opacity by local applications or internal medicaments; indeed it tends more and more to demonstrate the futility of such pretensions and attempts.

Nevertheless the inconveniences which attend progressing cataract may be palliated by alleviating local irritation, by the use of tinted glasses, by shading the eyes from bright light, by correction of optical errors, or by the use of stenopæic glasses. When the opacity, whether congenital or acquired, is limited to the pupillary area of the lens, vision may be improved by moderate dilatation of the pupil, by means of weak solutions of atropine (gr.  $\frac{1}{20}$  to gr.  $\frac{1}{8}$  in an ounce of water), or other mydriatic. In any case, if the eyes be otherwise healthy, moderate use of the eyes may be indulged in, short of discomfort and fatigue.

*Operations.*—The operations for the cure of cataract may, with some allowance made for differences of local conditions, be compared with the three procedures employed for the removal of vesical calculus—namely, simple crushing, with escape of the fragments *per vias naturales*—lithotripsy; crushing with instant removal of the fragments by aspiration—litholapaxy; and, lastly, extraction of the calculus *en masse*—lithotomy. The corresponding operations for cataract are: First, *discission*, or *keratonyxis* (κέρας, horn [cornea]; νόσσω, I prick), in which the anterior capsule is torn by a fine needle passed through the cornea. The lens-substance, being thereby exposed to the disintegrating action of the aqueous humour, gradually liquefies and is ultimately eliminated by the absorbent vessels. Secondly, *discission* with *evacuation*. The capsule is torn as in discission, but the lens is more freely broken up. The fragments are then, or within a few days, removed by a grooved curette through a

narrow incision immediately within the corneal margin (linear extraction), or withdrawn by aspiration through a fine tube (suction). The third operation is *extraction*, in which an incision is made at or near the corneal margin, the capsule divided and the lens extruded by gentle pressure. Until quite recently a portion of the iris was in the modern operation removed (iridectomy) at the time of the extraction-operation, or a few weeks or months previously (preliminary iridectomy). But some surgeons now try to dispense with the iridectomy, or only do it when the cataract cannot otherwise be safely and efficiently removed. The first and second of the operations above referred to are applicable to 'soft' cataracts, while extraction is usually reserved for 'hard' cataracts. Soft cataracts may be operated on almost at any time and in any stage; but hard cataracts are, as a rule, not extracted until they are 'mature,' though there are many circumstances which may suggest and justify departure from this rule. An immature cataract may be operated on whenever both eyes are so affected that the patient is unable to read ordinary print or to follow his occupation or profession.

It may here be remarked that when zonular cataract is slight, and does not cause appreciable impairment of sight, operation may be indefinitely deferred. If, on the other hand, the opacity is more pronounced, and the degree of impairment of vision is such that education or occupation cannot be comfortably pursued, operative interference is called for. The precise procedure will depend upon circumstances. If the cataract be small, dense, with defined margins and surrounded by a broad clear zone, a small iridectomy downwards and inwards (artificial pupil) may suffice to give useful vision; but if it be large, and not very opaque, or if its edges be ill-defined and broken by projecting striæ, and the surrounding zone narrow and hazy, then discission, with or without evacuation, will be indicated.

When the eye has recovered from the effects of the operation, spectacle-lenses are needed to correct the error of refraction due to the absence of the crystalline (*aphakia chirurgica*). Except in very short-sighted eyes convex lenses are required. The strength of the lens will depend upon the refraction of the eye, but ordinarily for distance a lens of about 10 dioptries (4-inch) is needed, and for near work about 14 D. ( $2\frac{3}{4}$ -inch). If astigmatism (*see VISION, Defects of*) be present, then cylindrical lenses must be added to the sphericals. In any case, it is desirable that an eye which has been operated on should not be used too soon, nor, at first, for too long a time.

Sometimes it happens after the removal of a cataract that, owing to the presence of hazy or opaque lens-capsule within the pupil, it is

not possible to obtain useful vision by the most accurate optical correction. It will then be necessary to tear through the membrane with one or two fine needles, and thereby effect a clear opening opposite the pupil.

JOHN TWEEDY.

**CATARRH** (κατά, down; and ῥέω, I flow).  
SYNON.: *Coryza*; *Catarrhus* (Cullen); *Catarrhus communis* (Good); *Rheuma*; Fr. *Catarrhe*; *Coryza*; Ger. *Katarrh*; *Schnupfen*.

DEFINITION.—The term catarrh is applied generally to inflammation or congestion of the mucous membranes, attended with increased secretion. Thus authors speak of *catarrh of the stomach, intestines, bladder, &c.* In the present article the term is limited to the inflammatory affections of the upper part of the air-passages, resulting from cold, and attended by discharge from the nostrils, soreness of the throat, hoarseness, and cough. The term *coryza* is, however, more especially limited to the cases in which there is copious discharge from the nasal passages, while catarrh is applied to affections of the whole mucous membrane, including the fauces and larynx.

SYMPTOMS.—The attack generally commences, shortly after exposure to cold or more particularly to cold and damp, with a feeling of indisposition, sense of cold down the back or general chilliness, weight in the forehead, headache, especially frontal, and dryness of the nares and throat. These symptoms are succeeded by the discharge from the nostrils of a thin acrid fluid, watering of the eyes, pains in the face, soreness of the throat and hoarseness, with aching in all parts of the body, and disinclination to bodily and mental exertion.

At first the affection is often confined to one nostril, and there is pain in the corresponding temple, eyebrow, eyeball, and side of the face, and lacrymation on that side; but it soon appears in the other nostril, and involves both eyes and all parts of the face; and there is great sense of weight and pain in the forehead and eyebrows. The discharge, also, loses the thin character and becomes mucous, and is often very profuse; there is copious lacrymation, the throat becomes decidedly sore, the hoarseness is greater, and there is pain in speaking and sometimes almost entire loss of voice. There are also transient pains in the chest, with a sense of tightness and some wheezing. The appetite from the first is impaired, and there may be entire distaste of food, and sometimes sickness and vomiting; not infrequently there is some sense of weight in the right hypochondrium, and sallowness of the complexion; the bowels are usually confined, but there may be diarrhoea. The tongue is generally white, the pulse may be a little quickened, the skin may be dry, the temperature is raised, and the urine is scanty and

somewhat high-coloured, and deposits a little sediment. The pains in the head and face especially affect the forehead, the eyebrows, the root of the nose, the eyeballs, and the course of the dental and other nerves; they generally increase towards night, and may be so severe as entirely to prevent sleep. Not infrequently there is more or less deafness, and usually loss of smell and taste. Herpetic spots often appear about the mouth, and the nostrils may become ulcerated from the discharge; the throat is more or less red and swollen, and often there is stiffness and pain of the neck, and tenderness on pressure over the larynx.

After these symptoms have continued for two or three days they generally gradually subside; but the cough may continue troublesome, and the patient be able to take very little food, and may still feel weak for a week or more. In persons of delicate constitution, also, the weakness is often very persistent; and, if care be not taken, more serious inflammation of the bronchial mucous membrane or of the lungs may supervene, and may lapse into phthisis.

TREATMENT.—In the slighter forms of common cold but little treatment is required, except the use of the ordinary household remedies: the feet may be placed in hot water, some warm diluent beverage may be taken, and a light diet must be had recourse to for a day or two. In the more serious cases febrifuge medicines may be given, with an anodyne to relieve the cough, if troublesome, or to procure rest at night, if the neuralgic pains be very severe.<sup>1</sup> When the attack has continued for two or three days a more stimulating diet may be given, and during convalescence tonics and stimulants may be required.

Dr. Ferrier recommends in catarrh the local application to the nose of the following powder in the form of a snuff: Hydrochlorate of morphine 2 grains, subnitrate of bismuth 6 drachms, gum acacia in powder 2 drachms. From one-quarter to one-half of this may be taken in the course of twenty-four hours.

It not infrequently happens that in delicate persons a cold is very difficult to get rid of, and the slightest exposure is followed by an aggravation or renewal of the symptoms. When this is the case the most effectual remedy is change of air, and the patient after leaving home often rapidly improves, and soon gets well. THOMAS B. PEACOCK.

**CATARRHAL** (κατά, down; and ῥέω, I flow).—Pertaining to catarrh, both in its pathological and clinical significations—for example, catarrhal products, catarrhal pneumonia, catarrhal fever, catarrhal attack.

<sup>1</sup> From 20 to 60 minims of Spiritus Ammonia Aromaticus in a claret-glass of any effervescing water may be taken at bed-time or oftener with marked benefit.—EDITH.

**CATHARTICS** (*καθαίρω*, I cleanse).—This word is sometimes used as a synonym for purgatives; but in a more limited signification it means purgatives of moderate activity. See PURGATIVES.

**CAUSES of Disease.**—See DISEASE, Causes of.

**CAUSTICS** (*καίω*, I burn).—DEFINITION. Substances or measures which destroy organic tissues with which they may be brought in contact.

ENUMERATION.—The caustic substances in most common use are Potash, Soda, and Lime; Nitric, Hydrochloric, Sulphuric, Chromic, Lactic, and Glacial Acetic Acids; Red Oxide, Acid Nitrate, and Perchloride of Mercury; Carbolic Acid; Chloride of Zinc; Chloride of Antimony; and Arsenic. The ordinary caustic measures are the galvanocautery; Paquelin's cautery; the red-hot iron; and *moxæ*. See also POISONS.

USES.—Caustics are chiefly employed to destroy unhealthy, exuberant, or malignant growths; to establish issues for the purpose of counter-irritation (see COUNTER-IRRITATION); and to destroy poisons when introduced into the body by breach of the external surface.

T. LAUDER BRUNTON.

**CAUTERETS, in the French Pyrenees.**—Sulphur waters. See MINERAL WATERS.

**CAVERNOUS.**—A peculiar quality of sounds heard on auscultation of the lungs, indicative of the presence of a cavity. See PHYSICAL EXAMINATION.

**CAVITY, Pulmonary.**—As the result of certain morbid processes which terminate in the destruction of portions of the pulmonary tissues, abnormal spaces or excavations are frequently formed in the lungs, which are designated *cavities* or *vomica*. These are usually associated with, and are by far most important in that large class of cases which are grouped under the term Phthisis. They may, however, originate under other conditions, namely, as the result of abscess or gangrene of the lung; of the destruction of morbid growths or hydatid cysts; of dilatation of the bronchi; or of destruction of the pulmonary tissue from without, in connexion with glandular disease, empyema, and other lesions. The most recent observations on this subject are given in the article VOMICA.

Pulmonary cavities present wide variations in different cases as regards their number, size, shape, condition of their walls, amount and nature of their contents, and other particulars. Usually they begin to form in the upper part of one lung, but subsequently they are produced in other parts, frequently both lungs becoming more or less involved, and any portion may be excavated in the first instance. A cavity frequently goes

through certain stages, namely, those of formation and extension; of arrest; and of healing or contraction, which may terminate in ultimate closure and obliteration of the vomica. Enlargement of cavities is effected either by progressive implication of their walls, terminating in their disorganisation and removal; or by coalescence of adjacent spaces, the intervening lung-tissue becoming destroyed. During this process of destruction some of the tissues often escape more or less, especially the vessels, which may not uncommonly be seen traversing the spaces or running along their walls, their channel being obliterated. When an excavation is arrested in its progress, it becomes lined by a smooth membrane, and a more or less purulent fluid is secreted within it. This cessation of active mischief may not take place until a whole lobe, or even the greater part of the lung, is involved, a huge cavity being formed, which presents no tendency to contract. In other instances the progress of destruction is stayed, the formation of purulent matter is checked and finally ceases, a fibroid tissue forms, and the space undergoes a process of contraction or cicatrisation, which may end in a complete cure, but more commonly merely diminishes the size of the vomica more or less. At a post-mortem examination in cases of phthisis it is common to find numerous cavities in the various conditions and stages indicated above. Occasionally a vomica gives way into the pleura, followed by pneumothorax and its consequences.

Clinically, the existence of cavities in the lungs can only be ascertained positively by physical examination of the chest; and as a rule not only their presence, but their conditions, may by this means be determined with tolerable accuracy. The physical signs vary considerably in different cases, and are more conveniently described in other articles. See PHTHISIS; PHYSICAL EXAMINATION; and VOMICA.

FREDERICK T. ROBERTS.

**CELL** (*cella*, a cell in a honeycomb; a closed chamber).—The term 'cell' was for a long time applied in anatomy to various spaces in the body large enough to be recognisable by the naked eye; hence 'cellular' tissue, a name which is still often used to designate areolar or connective tissue. But the word came afterwards to be applied by botanists in an entirely different sense, namely, to the hollow, bladder-like particles of which many of the parts of plants were shown by the microscope to be composed; and the name was extended so as to apply to similar particles in the animal body. It has since been retained and used in histology in that sense, although it has long been recognised that these particles, whether in plants or animals, but especially in the latter, do not necessarily conform to the original definition of a cell, the principal factor in which was the presence of a definite cell-wall.

Cells may be either *free* or *fixed*. Instances of free cells are to be found in the white corpuscles or leucocytes which occur in blood, in lymph, and pus, and which constitute the bulk of lymphatic or lymphoid (adenoid) tissue. As instances of fixed cells may be mentioned the various kinds of epithelium cells; the fixed cells of the connective tissue, including cartilage and bone; the cells of nervous tissue; and those which constitute the several kinds of muscular fibres. To these may be added the cells of most tumours.

All the cells in the body can be shown to have been ultimately derived from the ovum or egg-cell, which, after impregnation by and union with a spermatozoon or sperm-cell, divides at first into two, these again into two, and so on by a binary process of subdivision until a small mass of cells is ultimately produced. As the cells continue to multiply, this mass presently becomes hollowed out by the accumulation of fluid in its interior, and is converted into a vesicle the wall of which is eventually found to show an arrangement of its component cells in three layers, which collectively form the *blastoderm*. The three layers are termed respectively, from without in, the *epiblast*, *mesoblast*, and *hypoblast*, and give origin in course of development to the different tissues and organs of the body. The epiblast produces the cuticle and nervous tissues, and the essential parts of the sense organs; the mesoblast the muscular and connective tissues, including the blood and blood-vessels; the hypoblast forms the epithelium of the alimentary canal, and of the glands which open into it, including the epithelium of the pulmonary air-passages.

Cells vary very much in size, but few are large enough to be visible to the naked eye. Every cell in the body possesses one or more nuclei, although in some cells, in consequence of chemical alterations, the nucleus may have disappeared completely. Such cells are, however, invariably dead, and undergo only passive changes; of the living cell the nucleus appears, at least in all the higher animals and plants, to be an essential part.

The main part of the cell, in which the nucleus is embedded, is known as the *cell-body* or *cell-substance*. In most cells it is chiefly formed by a soft albuminous material, named *protoplasm*. It is upon the presence of this material that the so-called 'vital' phenomena which are manifested by the cell obviously depend, such as the amoeba-like movements or changes of shape which are exhibited by free cells such as the white blood-corpuscles; the production, storage, and eventual expulsion from the cell of materials which are to take part in the formation of the secretions, as in the fixed cells of glands; and the internal changes, molecular or molar, which result in the wave-like transmission of impulses or movements,

as is seen in the manifestations of activity which occur in muscle, nerve, and in ciliated cells. Most of these changes are or may be produced or modified by the incidence of external stimuli; and the property by virtue of which the protoplasm responds to these or other (unknown) stimuli is termed its 'irritability' or 'excitability.'

When protoplasm is examined chemically in the dead state it is found to be mainly composed, besides water, of certain forms of globulin and albumin, which do not materially differ from those which occur in fluids, such as egg-albumen and blood-serum, which exhibit none of the phenomena of life. But there is reason to believe that the molecular constitution of the protoplasm is very different during life from that which is found after death, being in the former condition of an especially unstable character and undergoing changes with great readiness. It is indeed in the highest degree probable that chemical and physical changes are *continually* proceeding during life, and that the chemical changes are of two antagonistic kinds, the one kind tending to produce a building up of fresh protoplasmic substance from the proteid and other material supplied by the blood and lymph, and the other kind tending to produce a breaking down of such substance, and a formation of simpler products. To these two hypothetical antagonistic processes which proceed during life, the terms *anabolic* and *katabolic* have been applied, while to the whole of the chemical changes which go on, both in the individual cells and in the collective organism, the name *metabolism* is given.

The forms assumed by the cells are very various; thus they may be spherical, ovoidal, spindle-shaped, flattened or scale-like, columnar, stellate—in short, of any conceivable shape. Fixed cells retain the same shape for an indefinite time, or undergo only passive alterations; free cells are frequently observed to alter their shape continually by the spontaneous protrusion and retraction of processes of their protoplasm (*pseudopodia*). These spontaneous changes or amoeboid movements may, under certain circumstances, produce an actual locomotion of the cell, as is noticed to occur in the process of *diapedesis*, or passage of the white blood-corpuscles through the walls of the capillaries and venules.

Cells may lie scattered and isolated in a tissue, or they may be united by their processes into a network, as in the connective tissues, or closely packed and joined edge to edge to form either a membranous stratum or a solid mass of cells, which may constitute the bulk of the tissue to which the cells belong, as in the epithelial tissues. Or the cells of a tissue may undergo special development and structural modification, associated with the assumption of special properties of conduction and contractility, as in the nervous and muscular tissues. In ciliated epithelium

the protoplasm of the cell is prolonged at the free border into minute hair-like projections, which during life execute a spontaneous lashing movement (*vibratile cilia*).

In spite of the numerous researches which have been made of late into the structure of cells, very little is known regarding the structure of the protoplasm. Until a comparatively recent date it was described as entirely without structure; and in many cells, especially those of the free type, it is difficult to make out any definite appearance of structure, especially in the living condition. It is true that under the influence of certain hardening re-agents, such as alcohol and chromic acid, a reticular appearance is produced in protoplasm, but the same appearance can equally well be produced in solutions of albumin and mucin under similar circumstances, so that the reaction in question by no means proves that the apparent structure is preformed; and it is still the opinion of some histologists that protoplasm is essentially structureless. It is certain, however, that in many *fixed* cells a network may be observed even in the living condition, the nodes of the network causing a characteristic finely granular appearance in the protoplasm, and most cytologists accordingly describe the living substance of a cell as being composed of a spongework (*spongio-plasm reticulum*), and interstitial matter or matrix occupying the meshes of the spongework (*hyaloplasm enchylema*). Whether the vital phenomena exhibited by protoplasm depend upon the spongio-plasm or the hyaloplasm, or belong to both these parts of the living substance, is entirely unknown.

The protoplasm of a cell may contain other materials embedded in its substance, which may either have been formed by the protoplasm from materials obtained from the blood, or in the case of 'free cells' may have been taken bodily into the cell-protoplasm by aid of the amoeboid movements of which these cells are capable (inception of particles). These materials may be in the form of granules of albuminous, fatty, or carbohydrate nature, or of globules of watery fluid containing substances in solution (vacuoles). Such non-protoplasmic ingredients of the cell-substance, when occurring within the cell as ordinary products of its nutrition, are collectively termed *deuteroplasm*. This term also includes such portions of protoplasm as may have become converted within or at the surface of the cell into non-living material, as when the external layer of protoplasm becomes transformed by physical and chemical alterations into a firmer and more resisting covering to the cell, which thus becomes invested with a *cell-wall*. Pathological changes in cells are usually due to chemical transformations of the protoplasm, which in this case are usually termed *degenerations*, such as the *fatty*, *mucous*, and *colloid*.

Every cell in the body possesses at least one

nucleus, but some cells—*e.g.* the white blood-corpuses—may have two or three nuclei; and others, such as the giant-cells which are met with in normal red marrow and in certain pathological formations, may contain a large number. The nucleus of a cell is usually spherical; it is situated near the centre of the cell, and appears to possess the function of presiding over the nutritive changes of the protoplasm, as well as that of initiating the division of the cell.

Most nuclei have a well-marked reticular structure, which is visible even in the living cell unaltered by reagents. The filaments of the network have the property of becoming darkly stained by hæmatoxylin and many other staining reagents; hence the substance of which they are composed has been termed *chromatin*, to distinguish it from the part of the nucleus which remains unstained, and which is termed in contradistinction *achromatin*. The filaments are aggregated at the surface of the nucleus into a closely reticulated membrane, which serves to limit the nucleus externally. The nodes of the network, as seen in optical sections of the nucleus, give the latter a coarsely granular appearance, especially because the substance of the filaments tends to become aggregated at the nodes. Besides these aggregations there are usually present in the nucleus one or two larger masses of chromatin, staining somewhat differently from the rest, and not always situated at a node of the nucleus network. To these larger masses the term *nucleoli* is applied. In some nuclei the usual network is replaced by a sort of convoluted skein of nuclear filaments, the latter being somewhat thicker than usual, and exhibiting, when sufficiently magnified, a peculiar transversely striated appearance. This condition is, however, usually only met with in nuclei which are about to undergo division.

The division of the nucleus was formerly believed to occur by a simple process of fission, preceded by the division of the nucleolus. It is doubtful, however, if this simple process of *direct* division occurs regularly in any cells. In nearly every case of cell-division, both in normal tissues and in pathological formations, the nucleus undergoes a complete series of changes, which are collectively known under the name *karyokinesis* or *karyomitosis*, and which result in the division first of the nucleus and ultimately of the cell (so-called *indirect* division). The changes which are thus undergone are as follows:—The whole of the chromatin, including the substance of the nucleoli and the membrane of the nucleus, first becomes collected into one or more filaments, which have a convoluted skein-like arrangement, the skein being at first closely wound, and its filament or filaments fine, but becoming gradually more open, and its filaments coarser. Next, it is found that the skein has become subdivided

into a definite number of short filaments, which often assume a bent, V-shaped form, with the apices of the V's directed towards the centre of the nucleus (*aster phase*). The V's then undergo an alteration in relative position, becoming interlocked at the equator of the now elongated nucleus, and having their apices directed towards the two poles. Usually at this *equatorial phase* of karyokinesis the filaments undergo a process of longitudinal cleavage, so that there are now twice as many as before. The resulting finer V-shaped filaments then begin to pass into two groups, which gradually tend towards either pole of the nucleus, assuming as they pass thither a radiate arrangement, with the apices of the V's towards the centre of each group. This is the *dyaster phase*. Finally, in each of the two groups, the V-shaped filaments unite end to end to form a skein, which subsequently becomes converted into a network, and the formation of the daughter-nuclei may now be regarded as complete. Around each one the corresponding half of the protoplasm of the cell becomes aggregated, so that two daughter-cells have become formed by division of the parent-cell.

While these changes are going on in the chromatin of the nucleus, others are proceeding in the achromatin substance. Within this a spindle-shaped system of the finest possible fibrils makes its appearance, both the poles and the equator of the spindle corresponding with those of the nucleus. At each pole of the spindle is a spherical particle termed the *polar particle*, and from this particle other fine fibrils radiate into the adjacent protoplasm, so that a system of achromatic fibres penetrates not only through every part of the nucleus, but throughout the greater part of the protoplasm of the cell as well. The fibres of the nuclear spindle appear to serve as directing lines along which the above-described movements of the chromatic filaments towards the poles of the nucleus occur, and the equator of the spindle determines the plane of separation both of the nucleus and of the cell.<sup>1</sup>

E. A. SCHÄFER.

**CELLULITIS.** — DEFINITION. — Cellulitis is the term applied to inflammation of the cellular or loose connective tissue, chiefly the subcutaneous areolar tissue, but also that interposed between muscles and viscera, or surrounding various organs.

The areolar connective tissue is so universally distributed throughout the body, that it is necessarily concerned in most inflammations—no matter of what structure; and in

it, in fact, the chief changes generally take place.

To consider completely the pathology of inflamed connective tissue would be more or less to review the whole series of the acute diseases. We must limit our consideration to cases in which the cellular tissue is the chief or only tissue involved, or where changes in other parts are secondary to those primarily affecting the cellular tissue.

Beneath the skin, over the whole surface of the body, lies a layer of this tissue, containing within its meshes more or less adipose matter. It will be convenient to consider the changes which occur in it when inflamed, as they are identical with those in cellular tissue elsewhere.

Inflammation of the subcutaneous cellular tissue may be either *diffuse* or *circumscribed*, which are varieties rather in degree than in kind. The former is nearly always acute in type; the latter often, but not invariably so. A *chronic* form of cellulitis causing thickenings is observed in various regions, or it may be a sequel to the acute disease. The acute form of the disease is clinically similar to phlegmonous erysipelas, and is characterised by a spreading inflammation without tendency to arrest.

**1. Circumscribed Cellulitis.**—**ÆTIOLOGY.**—Any injury to a part, whether of the nature of a wound or contusion; an impacted foreign body; or a fragment of bone, may cause cellulitis. Septic absorption from any decomposing secretion in a wound, altered blood, or infiltrated urine is prone to produce marked inflammatory changes in the connective tissue in different parts of the body. The poison introduced in a dissection- or *post-mortem* wound often occasions an acute cellular inflammation. Frost-bite, burns, inflammation of viscera, arteries, veins, or periosteum may produce inflammation of the adjacent cellular tissue; thus, inflammation of the kidney may cause perinephritis; inflammation of the uterus may lead to pelvic cellulitis; or some mischief in the greater bowel or rectum may produce inflammation and abscess in the loose cellular tissue around them (*perityphlitis* or *ischio-rectal abscess*); the poison also of scarlatina causes cellulitis of the submucous areolar tissue of the throat; and *angina Ludovici* is the name given to the cellulitis of the floor of the mouth and neck which is often associated with symptoms of intense septicaemia. A *sympathetic bubo* is an irritated lymphatic gland causing inflammation of the cellular tissue around it.

**PATHOLOGY.**—Pathologically, areolar connective tissue is of the greatest importance in the organism, being the most frequent seat of inflammatory and other changes. It mainly consists of loosely interlaced bundles of fibrous tissue, with flattened connective-tissue corpuscles adherent to them, and leucocytes, or amœboid corpuscles, in

<sup>1</sup> It is difficult to make these changes in the cell and cell-nucleus perfectly clear without the aid of illustrations. The reader who desires a more complete account of the subject is referred to *Quain's Anatomy*, 10th edition, vol. i., where also a list of recent articles dealing with the question will be found.

the areolæ. The exact part played in inflammation by the cellular elements is not quite settled. Under ordinary circumstances the leucocytes doubtless proliferate, and the fixed corpuscles probably do so also. Even under the influence of a slight irritation the flattened corpuscles in a few hours become globular, and present many nuclei in their interior—changes certainly pointing towards proliferation; while the very rapid increase of cells which takes place points to their derivation from cells pre-existing in the part, although the immigration of leucocytes from the blood into the inflamed part adds considerably to their numbers. Whether the perversion of nutrition which forms the starting point of the disease first induces a local cell-proliferation, or an immigration of leucocytes, or what proportion these two processes bear to each other, is difficult to determine.

The disease consists essentially in a very active cell-proliferation and increase. Whether the cellulitis be circumscribed or diffuse, similar changes occur; the difference between the varieties being that in the former there is formed a limiting zone of vascular tissue resembling granulation-material, which is absent when the inflammation is diffuse. There are otherwise no anatomical differences. When cellular tissue inflames, the part swells from the sero-fibrinous exudation poured out from the distended capillaries; its meshes are filled with young round cells, partly by proliferation of the connective-tissue corpuscles, in part by the accumulation of migrating leucocytes; and the circulation is interfered with by the pressure of the effusion, complete stasis sometimes taking place. While the cell-increase is proceeding, the fibrillar intercellular substance gradually disappears, in part by necrosis, and in part by becoming liquefied; and the tissue is finally transformed into pus. When this has happened the deeper layers of the skin disintegrate; it becomes undermined and gradually thinner; necrosis in one or more places follows; and the pus mingled with shreds of dead cellular tissue escapes, the latter resembling nothing so much as soaked washleather. The pus, at first thin and serous, subsequently becomes laudable. There is always a great tendency to suppuration, the vitality of areolar tissue being very low; but resolution sometimes takes place without formation of pus. The consequences of cicatrization differ, according to the tissue or organ involved and the extent of the disease; but essentially they are similar everywhere. A gradual contraction sets in. In external parts we can observe atrophic changes taking place, followed sometimes by deformity or loss of function, while in the viscera the condition is known as cirrhosis. The special tissue of an organ or of a muscle cannot be reproduced; it is replaced after an injury by

connective-tissue cicatrix. In such tissues as bone, tendon, and nerve, however, the cicatrix will usually be converted into the normal tissue of the part.

**SYMPTOMS.**—The amount of fever varies with the extent of the disease and the nature of the cause. When the cellulitis is quite limited there may be little or none, but deep-seated or extensive cellulitis produces considerable constitutional disturbance. Painful swelling of the inflamed part will first be observed; the skin soon becomes tense, red, and œdematous, although at the outset it is sometimes paler than normal. The redness is gradually lost towards the periphery of the swelling, and is darker, or of a bluish-red tint in the centre, from the obstruction to the exit of blood; the swelling is doughy, inelastic to the touch, and pits on pressure. The inflamed region feels hard, the induration ceasing by no well-defined border. If resolution occur all these symptoms subside. A greater or less amount of thickening of the tissue may, however, persist—often for a lengthened period, the parts slowly returning to their normal state. In the central portion suppuration is, however, the rule. When it occurs, the pain and tension diminish; fluctuation is felt—obscure at first; the pus by degrees approaches the surface, and escapes spontaneously, or by an artificial outlet which may be provided. When the inflammation is more deeply placed, especially when beneath strong fasciæ, there will at first be no well-marked redness or swelling of the skin, or only a slight pinkish hue, with some œdema, to indicate the changes taking place beneath; and fluctuation may be difficult or impossible to make out long after pus has formed; but the pain and fever are considerable. This variety of the disease may also terminate in resolution—especially when early and appropriate treatment has been adopted; or in suppuration. It may also become chronic; or relapses may take place after temporary amendment. If the cause of irritation be a slight one, but repeatedly renewed, permanent thickenings or atrophic changes in the tissue may result; or the circumscribed may be converted into diffuse cellulitis. A very intense irritant sometimes induces gangrene. The same event may happen if a previously diseased tissue be attacked, as an anasarctous limb; or pressure, associated with the cellulitis accompanying the formation of bed-sores, may be sufficient to cause it.

**2. Diffuse Cellulitis.**—**SYNON.**: Diffuse phlegmon; Pseudo-erysipelas; Diphtheria of the cellular tissue.—This is a severe disease, attended by general symptoms of a marked character, frequently associated with septicæmia, of which it may be both a cause and an effect.

**ÆTIOLGY.**—The most frequent cause of diffuse cellulitis is some form of septic in-

fection. In the extremities the disease may originate from some trifling cause, especially in those whose constitution is impaired by age, privation, or excess; in the hand and forearm of such persons it is especially common after wounds on the finger or an insignificant whitlow. In convalescence from acute febrile diseases, especially typhus or typhoid, a local phlegmon is sometimes transformed into a diffuse cellulitis.

**SYMPTOMS.**—The local symptoms of diffuse cellulitis resemble those of the circumscribed form, but are more intense: the disease is not limited, and it is accompanied by severe constitutional disturbance.

A sudden rigor with elevation of temperature often ushers in the attack; the rigor may recur at intervals, but sweating is unusual, and vomiting infrequent. In the affected region the patient experiences a great sense of weight and distension, with severe dragging pain. When the inflammation is deep-seated the changes in the skin may not be well-marked, even after a considerable extent of the cellular tissue has perished. This peculiarity is a very dangerous one, because it leads to the nature of the affection being for a time overlooked and efficient aid postponed. When the skin participates, the redness is dusker in hue, less sharply defined, and less easily dispersed by the pressure of the finger than in the cutaneous inflammation of erysipelas, while it soon becomes oedematous. The affected area feels brawny, hard, and swollen throughout, and is extremely tender and painful; sleep is interfered with; any movement causes great suffering; the fever is often very high; the secretions are diminished; and the appetite is lost. Rigor and sweating presently announce the formation of matter. The swelling becomes less prominent and more soft; the skin is mottled, thinned, and yielding in places; and the fever and pain subside. Convalescence may take place on the evacuation of the pus; or the rigor may be renewed, the fever reappear or continue, and the patient sink with symptoms of blood-poisoning. The more deeply the inflammation extends the more tedious is recovery, and the more liable is the patient to relapse. The muscles, tendons, and adjacent joints may become involved in the suppuration; or perforation of a dangerous character of neighbouring cavities or organs may take place. Suppuration consequent upon diffuse cellular inflammation will sometimes extend up the forearm to the elbow, undermine the skin, dissect the muscles, open into the finger- and wrist-joints, cause necrosis of tendons, and terminate in the loss of the limb by amputation, or perhaps in loss of life from septic poisoning. Should recovery ensue, the limb is often permanently crippled from the matting together of muscles and tendons, the impaired mobility of the joints, and the adhesions that

take place between tissues which should freely glide over one another. Suppuration is the rule, but under favourable circumstances, and with early and suitable treatment, it may occasionally be prevented. Usually pus has already formed when the case comes under observation, and the surgeon has only to use his history to evacuate the matter and thus limit the spread of the disease.

The irregular cavities and sinuses left after the evacuation of the dead tissue often suppurate for a long time, and this may lead to amyloid degeneration of the viscera. The thromboses which form of necessity in the smaller veins implicated in the inflamed area may break down and lead to septic embolism and pyæmia. The risk of this complication is a serious and ever-present one in these cases.

**DIAGNOSIS.**—Cellulitis has chiefly to be diagnosed from erysipelas. Erysipelas may involve the subcutaneous tissues, and cause inflammation and suppuration of the connective tissue, but it always begins in the skin, which is more extensively affected. Inflammation of the cellular tissue begins beneath the skin, where the swelling and effusion first take place; the skin becomes involved later, and usually to a less extent, while it may remain, at least for some time, almost entirely free; the redness, too, is less bright, and more diffused, not presenting the distinct margins of erysipelas, but fading into the surrounding parts. In the later stages the two conditions are scarcely distinguishable.

At first it may be difficult to decide whether the case is one of inflammation of the subcutaneous cellular tissue, of the intermuscular areolar tissue, or of that connected with the periosteum, or around a vein. The greater the general swelling of the limb, the more considerable the fever and the pain, and the less the redness of the skin, the more probable is it that the inflammation affects the more deeply seated structures.

**PROGNOSIS.**—The prognosis of cellulitis depends on the extent and severity of the disease and the constitution of the patient.

**TREATMENT.**—The local cause should be removed, as far as practicable. If the wound be in a foul condition it should be rendered aseptic. Absolute rest to the inflamed parts is of great importance. So long as suppuration has not occurred, resolution is possible. Graduated pressure can rarely be tolerated. Blistering is not employed in the acute form, but may be useful in removing more chronic changes. Cold applications and ice may abate pain and inflammation, but in many cases warm fomentations and poultices give relief. In the more advanced stages, especially when they tend to become chronic, cold is useless or even dangerous, from liability to cause gangrene in debilitated subjects. Local blood-letting does not prevent suppuration, and is usually contra-indicated by the weak state of the patient. When pus forms, or

its presence is suspected, a sufficiently free outlet should be provided for it as soon as possible. Nothing so effectually checks the further spread of the disease. The incision should be made at the most prominent point. It is often better to make a number of small incisions, from half an inch to an inch in length, than one long one, which is apt to be followed by serious bleeding, and does not so efficiently relieve the strangulated tissues. When suppuration is only suspected, incisions should nevertheless be practised without delay, without waiting for fluctuation. Pus and shreds of dead cellular tissue should be frequently washed out of the wound with an irrigator. No force should be used to remove portions of dead tissue; any dragging tends to rupture the small blood-vessels, and to destroy the remaining connexions of the skin with the deeper structures. Antiseptic precautions must be zealously carried out.

When a joint is involved, or when the patient is exhausted by the discharge, and the tissues are spoiled, amputation is often necessary. Excision may be practised if the condition of the soft parts admits of it. But these are points for the surgeon's consideration.

The general treatment consists in giving nourishing food and stimulants, combined with opiates to relieve pain, and iron, quinine, and other tonics.

WILLIAM MAC CORMAC.

**CEPHALALGIA** (κεφαλή, the head; and ἄλγος, pain).—Pain in the head. See HEADACHE.

**CEPHALHÆMATOMA** (κεφαλή, the head; αἷμα, blood; and oma, a formative suffix indicating a tumour).

**DEFINITION.**—An effusion of blood occurring in newly born infants, forming a tumour upon the head; situated beneath the pericranium, upon the surface of the skull; or more rarely beneath the skull, between it and the dura mater.

**DESCRIPTION.**—This disease is of very rare occurrence, and must not be confounded with the *caput succedaneum*, which is an effusion of serum external to the pericranium, and of common occurrence. The blood is generally extravasated immediately beneath the pericranium, over one of the parietal bones, most frequently the right, but it may occur over the frontal or occipital. Combined with this, or arising independently, but of extreme rarity, may be an effusion beneath the cranium. The origin of cephalhæmatoma has been attributed to a variety of causes, but is most probably due to the constriction of the margin of the os uteri during labour. It is generally observed some hours or a day after birth, as a circumscribed swelling, slightly tense and fluctuating; and its peculiarity consists in a bony circle surrounding and limiting it.

**DIAGNOSIS.**—These tumours have been mistaken for hernia cerebri; but their situation over the bone away from the fontanelles, the absence of pulsation, and the existence of fluctuation in cephalhæmatoma should prevent this mistake.

**PROGNOSIS.**—Generally the blood becomes absorbed, but occasionally suppuration occurs, or the bone may become necrosed; if beneath the skull, serious consequences, including idiocy, may ensue.

**TREATMENT.**—As a rule, cephalhæmatoma is not to be interfered with. If suppuration take place the pus must be evacuated.

CLEMENT GODSON.

**CEREBELLUM, Lesions of.**—The cerebellum is liable to the same diseases as the brain and nerve-centres generally, such as hæmorrhage, abscess, various forms of degeneration, tumours, &c. The nature of the pathological condition is to be determined by the symptoms peculiar to each, so far as this is possible. Its locality in the cerebellum is to be diagnosticated, first, by certain symptoms which are due to the cerebellar lesion as such, which may be termed the *direct* symptoms; and, secondly, by those symptoms which depend more on the influence exerted by the lesion on neighbouring or subjacent centres and structures. These latter may be termed the *indirect* symptoms.

It is by no means easy to separate these symptoms from each other, and to say how much is due to interference with the functions of the cerebellum, and how much to interference with the functions of other parts. There are few diseases which have a purely local organic or functional limitation. Hence, in order to arrive at the symptoms peculiar to cerebellar lesions, it is necessary to exclude all pathological affections which in their very nature affect the whole of the intracranial centres, such as tumours or meningitis. The most satisfactory conclusions, from a pathological point of view, are to be drawn from cases of atrophy or degeneration of the cerebellum, and, from a physiological point of view, from the results of the experimental lesions of this organ in the lower animals.

The evidence from these two sources is mutually supporting.

**DIRECT SYMPTOMS.**—The characteristic symptoms of cerebellar disease are disorders of equilibrium, shown, on attempts at locomotion, in a reeling or staggering gait (titubation), and a continual tendency to stumble or fall over the most trifling obstacle, or on hurried movements.

These symptoms may be confounded with locomotor ataxy, but careful observation will show that in cerebellar disease there is no true ataxy. The movements are quite coordinated with each other, and are such as would instinctively be made to prevent fall-

ing, or to preserve the equilibrium; and have none of the precipitate, irregular, and sprawling character seen in ataxy. They are not specially intensified on closure of the eyes, which is such a marked feature in ataxy. Nor are they accompanied by any of the sensory affections of ataxy, whether in the form of pains or anæsthesia.

There is no true motor paralysis in cerebellar disease as such, and the various volitional movements of the limbs can be carried out perfectly well in the recumbent posture. Sensation, general and special, is not directly affected in cerebellar disease. Nystagmus and strabismus have been observed, more particularly in connexion with lesion of the cerebellar peduncles. Defects of articulation have been noticed, but it is very doubtful whether they should be regarded as direct symptoms.

Lesions of the cerebellum may be entirely latent, or not cause any very obvious symptoms during life. This has been observed in cases of congenital atrophy, or when the disease has been of a slowly progressive character, or limited to one lobe. Affections of the middle lobe are specially calculated to induce the characteristic symptoms.

**INDIRECT SYMPTOMS.**—*Pain in the head*, more particularly at the back, though not constantly situated there, is frequently associated with organic disease of the cerebellum.

*Vomiting* is also very frequently observed, perhaps more constantly than in connexion with diseases of other parts of the brain. There is, however, no reason to regard this as due to cerebellar disease as such. It is probably due to indirect effects on the medulla. As a general rule, diseases tending to encroach on the space of the posterior fossa, or to increase the pressure in this region, have a similar effect.

*Hemiplegia* is not uncommon in connexion with cerebellar disease, and more particularly in cases of tumour or hæmorrhage in the lateral lobe of the cerebellum. The hemiplegia is on the side opposite the lesion. This does not prove that the hemiplegia is due to the destruction of the cerebellar lobe, or that the cerebellar lobes have cross relations with the limbs. Experimental physiology and anatomical investigations tend to show that the cerebellar lobes are functionally related to the motor tracts on the same side. This is also borne out by the fact that atrophy of the lateral lobe of the cerebellum follows disease and degeneration of the opposite cerebral hemisphere. The hemiplegia from cerebellar disease is, therefore, in all probability, due to compression or some affection of the subjacent motor tracts, which decussate at the pyramids. The fact that it occurs chiefly when the disease is limited to the lateral lobe is what might be expected on anatomical grounds.

*Affections of sensation*, common and

special, have been observed in cases of cerebellar disease. Diminution of tactile sensation on the opposite side of the body, when the disease is situated in the lateral lobe, is to be accounted for in the same way as the motor paralysis. As regards the special senses, affections of sight have been most frequently noted. Sight is certainly not abolished by destruction of the cerebellum in the lower animals, and when blindness occurs in man in connexion with diseases of this organ, it is due either to implication of the corpora quadrigemina, functionally or organically, or to secondary degeneration in the optic tracts, as the result of neuritis.

A special feature of *tumours* of the cerebellum, more especially of the middle lobe, is a tonic rigidity of the muscles of the back of the neck, with retraction of the head, associated frequently with flexion of the forearms, and extension of the lower extremities and pointing of the toes. In these cases also, psychical affections, more particularly mental hebetude or stupor, occur as the result of secondary dropsy of the ventricles, caused by pressure on the veins of Galen. The symptoms then become those of hydrocephalus.

Hæmorrhage into the middle lobe of the cerebellum, in addition to the ordinary symptoms of apoplexy, has been frequently found to cause vascular excitement of the genital organs—in the male marked *priapism*. This symptom, of which several cases were first reported by Serres, led this observer to modify the view of Gall that the cerebellum, as a whole, was related to the sexual instinct, and to regard the middle lobe only as having any such function. The facts, however, are susceptible of a totally different interpretation, and one more in harmony with other data of physiology and pathology. It has been found experimentally by Segalas and by Eckhard that irritation of the posterior surface of the medulla and pons gives rise to vascular excitement of the generative organs, and hence the symptoms in cases of hæmorrhage into the middle lobe are to be ascribed to irritation of this region. This also explains the absence of the symptoms when the hæmorrhage occurs into the lateral lobes. There is absolutely no evidence of the slightest value in support of Gall's hypothesis. The facts of clinical medicine go a considerable way in diametrical opposition to it, if they are not themselves sufficient entirely to overthrow it.

**Cerebellar Peduncles.**—Respecting the effects of disease of the restiform tracts or inferior cerebellar peduncles we have no definite knowledge.

Cases, however, are on record in which lesions have been found involving principally either the superior or middle cerebellar peduncles. The symptoms, in the main, agree with those observed by Magendie on section of the middle peduncle in the lower

animals. The chief effect of this lesion was to cause an irresistible tendency to roll over towards the side of lesion. Together with this rotatory disturbance of the equilibrium, a peculiar distortion of the optic axes was observed, the eye on the side of lesion being directed downwards and inwards, the other looking upwards and outwards.

In a case described by Curschmann (*Deutsches Archiv f. klin. Med.* xii. 356), along with appearances of basilar meningitis, which somewhat complicated the case, there was found a focus of softening, surrounded by capillary hæmorrhage, in the right superior cerebellar peduncle. The symptoms observed in this case were a rotatory distortion to the right side, to which position the patient invariably returned when resistance to this movement was withdrawn. There was no motor paralysis, nor was there any distortion of the optic axes.

A case has been put on record by Nonat (*Comptes Rendus*, 181) of apoplectic extravasation into the right middle peduncle of the cerebellum and right cerebellar hemisphere. In this case the head and trunk were twisted towards the right side, and the ocular symptoms were also present, the eyes being immovable in a position of skew deviation. Other cases are on record in which the cerebellar peduncles have been involved in more extensive lesions; but the symptoms, though not opposed to those above related, are incapable of differential analysis. The special diagnostic symptoms, therefore, of lesion of the cerebellar peduncles are what are frequently termed 'forced movements' (*Zwangsbewegungen*), or distortions of the normal axis of the trunk. The exact direction of the distortion, in consequence of lesion specially limited to one or other peduncle on the right or left side respectively, is somewhat doubtful, though as a rule it has been found towards the side of lesion. Much, however, will depend on whether the lesion is of an irritative or inflammatory, or of a destructive character. A lesion of an irritative nature, though occupying the same position as a destructive lesion, would exactly reverse the direction of the distortion of the head and trunk. D. FERRIER.

**CEREBRAL ABSCESS.**—See BRAIN, Abscess of.

**CEREBRAL APOPLEXY.**—See APOPLEXY, Cerebral.

**CEREBRAL ARTERIES, Diseases of.**—See BRAIN, Vessels of, Diseases of.

**CEREBRAL HÆMORRHAGE.**—See BRAIN, Hæmorrhage into.

**CEREBRITIS.**—Inflammation of the brain-substance. See BRAIN, Inflammation of.

**CEREBRO-SPINAL FEVER.**—**SYNON.**: Epidemic Cerebro-spinal Meningitis; the Black Sickness (popular, Dublin); Fr. *Méningite cérébro-spinale épidémique*; Ger. *Cerebral-Typhus*; *Epidemische Meningitis*.

**DEFINITION.**—An acute epidemic febrile disease, characterised by sudden invasion, with extreme nervous shock, vomiting, excessive pain referred to the back of the neck and spine, spasmodic contraction of muscles, excessive sensibility of the skin, and frequently delirium; accompanied by purpuric eruptions, either circumscribed, raised, hard, and shotty to the feel, or extensive purpuric spots or patches, frequently accompanied by vesicular eruptions, usually of herpetic but sometimes of a pemphigoid character; and often purulent inflammation of the eyes. *Post mortem* there are found: inflammation of the membranes of the brain and spinal cord, especially of the arachnoid, with deposit of white, yellow, or greenish-yellow lymph upon the surface of the arachnoid, especially at the base of the brain and anterior portion of the medulla oblongata and spinal cord, and effusion of serum into the ventricles and sub-arachnoid spaces.

**ÆTIOLOGY.**—*Age.*—Cerebro-spinal fever is more commonly met with among children under ten years of age; after childhood it usually attacks those approaching the age of puberty or in early adult life; it is seldom met with after the age of thirty-five years, and is very rare after forty.

*Sex.*—The disease is much more frequent in adult males than females—robust males between the ages of fifteen and thirty are its chief victims; but among children it seems to attack both sexes almost equally.

*Occupation.*—It seems specially to affect young recruits in the army, as was the case in the French epidemics. In Dublin it was specially severe among the recruits of the Royal Irish Constabulary stationed in the police barracks in the Phoenix Park. There does not appear to be any other occupation which predisposes to cerebro-spinal fever. Excessive fatigue seems to increase the liability to the disease: it has arisen after a hard day's hunting, foot-racing, long walks, dancing, or in children exhausted from outdoor play.

In epidemics it occurs in isolated localities far apart from one another: it does not seem to extend from a district to adjacent districts; thus resembling affections connected with marsh miasms, and evidently requiring some very special local conditions to secure its spread. Micro-organisms similar to those described in pneumonia have been found in connexion with this disease, but no microbe has been discovered which can as yet be specifically associated with it.

*Season and Climate.*—Cerebro-spinal fever is widely distributed in the temperate zone. It prevails more in cold than in hot weather;

in Ireland it has usually prevailed in winter and early spring.

*General Sanitary Conditions.*—It seems to be less influenced than most other epidemic affections by general sanitary conditions.

*Communicability.*—It is generally believed not to be contagious. A few doubtful cases of contagion have been recorded; except, however, where it has appeared as an epidemic among recruits, there are comparatively few instances of more than one case arising in any particular house or circumscribed locality.

*Epidemic Influence.*—The disease is undoubtedly epidemic in its character.

*Unwholesome Food.*—It has been suggested that cerebro-spinal fever owes its origin to the use of breadstuffs made from diseased grain, but there is no proof of such being the case.

**ANATOMICAL CHARACTERS.**—Cadaveric rigidity is well-marked. Large purpuric patches form after death, even in cases where there were no purpuric symptoms during life; in some cases the whole body has turned black. On incision a large quantity of dark, tarry-looking blood exudes; the muscles are darker than usual, and in prolonged cases much weakened and wasted. There is increased vascularity of the scalp; the cerebral sinuses are much distended with dark blood; and serum is found in the sub-arachnoid spaces and ventricles. All the membranes of the brain may be more or less congested, the arachnoid being always extremely vascular and opaque from deposits of lymph—this opacity varies from slight milkiness to thick and dense deposits. The most marked intracranial lesion is the white-yellowish or yellowish-green ‘fibrino-purulent’ deposit found at the base of the brain. This deposit varies somewhat with the duration of the disease: in cases which die early the deposit is usually slight, whitish, and soft; in those which live for a week or so it is yellowish or greenish; in prolonged cases the deposit is more white and pure, the effused serum greater in quantity, and the vascular fulness less. The origins of the nerves seem to be buried in and compressed by the deposit. The brain-substance itself is more vascular than normal, but not otherwise altered. In the spinal cord the lesions are similar to those found in the brain and its membranes. In some cases purulent infiltration of the eyeball, and effusion into the joints have been met with. The lungs, liver, and spleen have been found much congested and softened in many cases.

**SYMPTOMS.**—The patient is usually attacked suddenly, when apparently in vigorous health, by faintness, vomiting of greenish matter, and intense pain referred especially to the back of the head and neck; the extremities become cold; the patient becomes insensible and sometimes convulsed; the limbs become rigid. On recovering from the collapse, the patient

complains of great pains in the head, back of neck, and along the spine; the head is drawn back, so as to be almost at a right angle with the spine; the whole back is sometimes arched, as in tetanus; the muscles become rigid, and the skin excessively sensitive; neuralgic pains are also complained of in all parts of the body. In most of the severe cases eruptions appear within the first twenty-four hours. The eruption usually appears first on the legs, and is frequently confined to the lower extremities. The spots are usually black, raised, about a line in breadth, and feel like a grain of shot under the skin. Sometimes these raised spots are surrounded by a dark purplish areola; in most cases large purpuric patches of many inches in extent form on various parts; sometimes they coalesce and cover the entire body. Vesicular eruptions are also common; these usually have the character of herpes, and are most frequent on the face, neck, and shoulders. The herpetic eruptions are met with as frequently in mild as in severe cases. Pemphigus sometimes appears in the advanced stages of the disease. When reaction sets in, the temperature is found to have risen to from 100° to 103° or 104° F., and in some cases has reached 107° F. In many cases the temperature never rises, and the patient dies in the stage of collapse; the respiration becomes of a sighing character; the pulse rises to about 120, and has a peculiar jerking character, giving a sharp upstroke to a sphygmographic tracing.

The foregoing symptoms are very variable; any one, or even a considerable number of them, may be absent throughout the whole course of the case.

**COMPLICATIONS AND SEQUELE.**—Complications connected with the nervous system are the most often seen. Paralysis of one or more limbs is common, of a hemiplegic character, and most frequently attacking the arm. Deafness is not very common, but has been met with in several cases, sometimes becoming permanent. The eye-complications are among the most frequent and most serious. The eye may be attacked with a low form of inflammation, terminating in purulent infiltration of the whole or part of the organ; the cornea is more frequently involved than any other portion; and this sometimes giving way, the whole contents of the globe escape, causing hopeless loss of the eye. The sight is also often permanently impaired by iritis, or opacity of the cornea. In many cases, however, the inflammation completely subsides. It is remarkable that it is the right eye which is usually attacked, that both eyes are seldom affected, and rarely the left eye alone. Acute inflammation of the larger joints is a frequent complication in some epidemics; this often terminates in purulent intra-articular effusion. Hæmorrhages are frequent in the more malignant forms, and are almost

always present in cases where the purpuric blotches are of large extent; these hæmorrhages have occurred from the nose, uterus, bowels, kidneys, and ears in about the foregoing order of frequency. Gangrene is occasionally met with, and the cases in which it occurs are usually fatal; although toes have been lost, and yet the patient has recovered. The purpuric patches have also sloughed without serious danger to the patient's life.

**DIAGNOSIS.**—Cerebro-spinal fever is liable to be confounded with typhus fever, on account of the petechial rash, but is distinguished from it by the eruption appearing suddenly, without any previous mottling of the skin. The nervous symptoms also distinguish it from typhus, although in a case of typhus complicated with cerebro-spinal meningitis the diagnosis is extremely difficult, and may be impossible. It is distinguished from *purpura hæmorrhagica* by the intensity of the fever, and the localised nervous symptoms. The malignant cases are more likely to be mistaken for malignant scarlatina than for any other disease, and must be distinguished therefrom by the rash, sore-throat, and nervous affections; yet in some cases the two diseases have been indistinguishable from each other, especially where death occurred within twenty-four hours and both were epidemic at the time. In other cases malignant small-pox has been mistaken for this disease.

**PROGNOSIS, DURATION, TERMINATIONS, AND MORTALITY.**—The prognosis, duration, &c., of cerebro-spinal fever depend much upon the form the affection assumes, and for convenience we may divide it into the following forms:—

1st. Cases of a very mild form, terminating in recovery; the duration being usually from one to three weeks.

2nd. Cases of a very severe form, setting in suddenly; the symptoms being very violent and well-marked, accompanied by purpuric spots and blotches, with a tendency to hæmorrhages, with deep collapse and coma; usually terminating fatally in from a few hours to three days.

3rd. Cases of medium severity, where all the nervous symptoms set in with less suddenness than in the second class of cases, purpuric blotches not being usually present, and no hæmorrhages. These usually yield to treatment, and terminate in recovery in from two to six weeks.

4th. Cases which set in either in a mild or in a severe form, but in which, on the subsidence of the fever, the strength does not return, convalescence is retarded or arrested altogether, and the patient falls into a general atrophic condition, and usually dies, in from three to six months, of marasmus.

By deciding to which of the above classes the case belongs, the prognosis will be to a great extent determined. The chief indication of danger is the early appearance of pur-

puric and hæmorrhagic conditions. Patients who have for from ten days to a fortnight escaped serious complications usually recover.

The mortality in cerebro-spinal fever is very high—probably on an average about 60 per cent. In some of the American epidemics it is placed as high as 75 per cent. Among the Irish constabulary it reached 80 per cent. Like other epidemic diseases, the mortality is highest at the commencement of the epidemic. It is most fatal about the age of twenty, and less so under fifteen years.

**TREATMENT.**—The treatment in the early stage must be directed to recovering the patient from the collapse. This is best done by the application of heat, the administration of small quantities of stimulants or stimulating enemata, and the application of sinapisms over the chest and back. In the next stage of the disease attention must be almost altogether directed to allaying the spinal irritation, and promoting the absorption of the effused matters. The extreme irritation will be best diminished by the use of belladonna and bromide of potassium. Ergot has also been found useful for this purpose. The pain, which is extreme, will yield best to frequent and considerable doses of opium; indeed, many physicians have relied altogether on opium as the curative agent. Chloral hydrate has proved beneficial, in conjunction with bromides, in allaying restlessness and procuring sleep. With a view of promoting absorption of the effused matters, mercury and iodide of potassium have been chiefly relied upon. In the more sthenic cases calomel may be employed with benefit in small and repeated doses. The disease being usually of an asthenic type, mercury will seldom be well borne, and iodide of potassium should be preferred. Leeches applied to the back of the neck, behind the ears, or to the temples, produce great relief of the excessive pain in the head and upper part of the spine. The application of ice to the head and spine temporarily allays pain, but there is little evidence of permanent benefit being derived therefrom. In prolonged cases blisters applied along the spine have been favourably spoken of. The local complications must be treated as they arise, and according to general principles applicable in each case. Stimulants are required in considerable quantity in a very large number of the cases which present adynamic symptoms.

T. W. GRIMSHAW.

**CEREBRUM, Diseases of.**—See BRAIN, Diseases of; also CONVOLUTIONS OF THE BRAIN, Diseases of; CORPUS STRIATUM, Diseases of; &c.

**CERES, in Cape Colony.**—See AFRICA, SOUTH.

**CHALAZION** (χάλαζα, hail).—A small encysted tumour of the eyelids, colourless and transparent, somewhat resembling a hailstone.

**CHALK-STONES.**—This name is applied to the deposits which are formed in connexion with the gouty diathesis, especially in the joints. They are thus denominated from their appearance and physical characters, in which they more or less resemble chalk; but in their chemical composition they are entirely different, consisting mainly of urate of sodium. See GOUT.

**CHALYBEATE WATERS** (*chalybs*, steel).—Mineral waters which contain iron. See MINERAL WATERS.

**CHANCRE** (Fr. *chancre*).—Hard chancre is the initial manifestation of syphilis. See SYPHILIS. For Soft chancre, see VENEREAL DISEASE.

**CHANGE OF LIFE.**—SYNON.: Climacteric epoch; Sexual involution; Fr. *Méno-pause*; Ger. *Menstruationsende*.

**DEFINITION.**—The time of life in a woman when the functions of the uterus and ovaries cease, menstruation terminating—a period when disease of these organs is especially prone to occur, and when various constitutional disturbances are almost certain to arise.

**ANATOMICAL CHARACTERS.**—Great changes occur in the sexual organs—after the ovaries lose their smooth outline, and after a while become shrivelled up, occasionally only a trace of them remaining; the Fallopian tubes diminish in size, and sometimes become obliterated; the walls of the uterus atrophy, its cavity becomes much smaller, and the cervix disappears altogether.

**SYMPTOMS.**—The term 'change of life' is used among women very widely to signify everything which affects them at this critical time. It is so rare for the transition from activity to inactivity to take place without some disturbance locally, or constitutionally, that women are apt to neglect seeking advice for symptoms which should demand careful treatment, believing as they do that it is natural to suffer in such ways at 'the change.' There is no fixed period for the climacteric epoch, though roughly it may be said to occur between the ages of forty-five and fifty. Certain causes are apt to determine the time—amongst these are parturition and lactation, febrile attacks, such as typhus or acute rheumatism, profuse hæmorrhages, fright, &c.

The symptoms vary much. In some women the change is abrupt, menstruation ceasing all at once after perfect regularity; in others, and more frequently, the change is prolonged, the catamenia being irregular for many months, and varying as to periodicity and quantity. Frequently, after a long interval, a profuse flow with clots occurs, and this is very often attributed to a miscarriage. This loss is frequently beneficial, and if it do not take place, and if relief be not derived from vicarious discharges, such

as bleeding from piles, the excess of blood gives rise to headaches, flushes, vertigo, and a host of other unpleasant symptoms. The balance between the nervous and circulatory system is upset; irritability of temper, hyper-sensitiveness and all sorts of fancies arise, or depression sometimes amounting to melancholia ensues. If germs of disease exist, the uterus is especially prone at such times to develop them, so that carcinoma, fibroid disease, and polypus frequently present their first symptoms at this epoch. The importance, therefore, of an early examination cannot be too forcibly dwelt on, or the mischief of delay from considering the abnormal condition as typical of 'the change of life,' and as a natural consequence, which will right itself. At these times pruritus of the vulva, vascular growths at the orifice of the urethra, and cutaneous eruptions are especially likely to occur. There is a tendency to grow fat, and to become coarse; hairs frequently appear on the face. The breasts often become very large and pendulous, and this, with the increase in the size of the abdomen from flatus, and the deposition of fat in its walls, together with the cessation of menstruation, not infrequently gives rise to the supposition of pregnancy. To this imaginary state the term *pseudocyesis* has been applied, and it is often almost impossible to set aside the opinion of the woman regarding her supposed condition. The headaches, neuralgia, loss of memory and nervous symptoms appear to be due to disturbance in the ganglionic system of nerves, with which the uterus and ovaries are largely supplied. If insanity arise, the most common form it assumes is hypochondriasis or melancholia.

**TREATMENT.**—This must be directed to regulating the secretions. Generally constipation, previously troublesome, becomes aggravated; and portal congestion frequently occurs. Saline purgatives are especially beneficial, and these may be administered in the form of mineral waters, such as Rubinat, Hunyadi János, or Friedrichshall. Blue pill with aloes is often very useful. The headaches and reflex nervous symptoms may be best combated by the administration of bromide of potassium, and this drug appears to act as a direct sedative to the sexual organs, besides diminishing the amount of blood determined to them. Occasionally, bleeding from the arm or cupping gives great relief. Attention must above all be paid to the diet. It should be plain and unstimulating: beer and spirits should be prohibited, and only light wines, if any, allowed. Tepid baths are useful. Late hours, heated rooms, and excitement of all kinds should be avoided. If local troubles arise, they must be treated according to their indications; as a rule, abstracting blood from the uterus does harm, but leeches to the anus are some-

times beneficial. It is clearly impossible to map out any empirical line of treatment for a condition in which the symptoms are so variable.

CLEMENT GODSON.

**CHAPPED NIPPLES.**—See BREAST, Diseases of; and NIPPLE, Diseases of.

**CHAPS.**—SYNON.: *Rhagades*.—Cracks or fissures of the skin occur where the integument has become hardened by infiltration, as in the erythema of the hands and wrists in cold weather, in chronic eczema, psoriasis and lepra vulgaris. The treatment for chaps consists in protection from the atmosphere; careful drying after the hands have been washed or wetted; and the use of zinc ointment and glycerine soap. Diluted glycerine, vaseline, and cold cream are also popular remedies.

ERASMUS WILSON.

**CHELOID** (χηλόη, a claw).—SYNON.: Cheloma; Fr. *Chéloïde* (Alibert); Ger. *Keloid*.

**DEFINITION.**—A tumour of the skin resulting from overgrowth of connective tissue within the corium.

**ÆTIOLOGY.**—Cheloma may be idiopathic or accidental; the former is, however, of very rare occurrence; and in both cases it is referable to a diathesis. When of accidental origin it is commonly associated with a cicatrix, and is then developed in the midst of the cicatrix-tissue. This form of the tumour has been denominated *cheloïdes spuria* or *traumatica*, and, as such, has been seen scattered numerously over the chest and shoulders as a sequel of acne.

The traumatic cheloid may follow a light as well as a severe injury of the skin, such as a boil, a blister, a leech-bite, or even the irritation of a stimulating liniment; it is sometimes met with in the scars of strumous abscesses or ulcers, but is most common in the cicatrices of burns or scalds.

**ANATOMICAL CHARACTERS.**—At its first development cheloma occupies the fibrous portion of the corium. As it increases in bulk, it pushes the vascular layer outwards and stretches the corpus papillare, obliterating the capillary network more or less completely. In its aggregate form, when it presents itself as a flat plate, raised for a quarter of an inch above the level of the adjoining skin and sinking to a similar extent into the corium, it has the appearance of being tied down by strong cords or roots at either end, and frequently overlaps the healthy skin along its borders. In this state it is seen to be composed of strong fibrous bands enveloped by a smooth transparent pinkish layer, in which may be detected a scanty vascular plexus converging to venules which sink between the meshes of the fibrous structure. Around the circumference of some of these larger flattened tumours, such as are commonly met with on the sternum, and measuring several inches in diameter, there

will generally be observed a few scattered knots. These are developed in the fibrous sheath of the arteries at a short distance from the mass, and, being thus linked to the central growth, are subsequently drawn into the focus of the tumour. And the development of the so-called 'roots' is explained by the propagation of the proliferating process by the coats and sheaths of the blood-vessels communicating with the central tumour. Fully-formed cheloid consists of dense white glistening fibrous tissue, the fibres of which are closely packed and arranged in more or less parallel bands. Occasionally bands of fibres are seen running obliquely, but they follow a definite course, and present quite a different appearance from the irregular mesh-work of fibres seen in the scar tissue. The cheloid grows up in the cicatricial tissue and pushes the latter aside, so that it can be seen surrounded by scar tissue. Cheloid also invades the healthy corium in the neighbourhood, and in this respect differs altogether from hypertrophied scar.

**DESCRIPTION.**—Cheloid has received its name from its habit of throwing out spurs from its circumference, these spurs having been compared to crab's claws. It originates in a round, oval, or oblong tubercle or knot in the skin, and this may be followed by a second in its immediate neighbourhood, or sometimes by a cluster of three or four. When two of these knots are situated at a short distance apart they are apt to become connected by a cord of the same structure as themselves, and to give rise to what has been called a *cylindrical, club-shaped, or dumb-bell* cheloid. When three or four knots are grouped near together they are disposed to become blended by growth, and produce an oval or square-shaped cheloid; and when the growth extends from these latter into the surrounding integument the appearance denominated *radiciformis* is established. Cheloma being due to a tendency to overgrowth or hyperplasia of connective tissue within the skin, it may appear on several parts of the integument at the same time, one of the most common seats of its development being the sternum, which it generally crosses transversely. It is often solitary; is more commonly composed of five or six tumours, discrete or confluent; but has been met with occasionally to the number of fifty or sixty tubercles or more.

**COURSE AND PROGNOSIS.**—Cheloid rarely gives rise to much inconvenience or attains any considerable magnitude. When left to itself, it progresses very slowly or remains stationary for a number of years or for life; and it has been known to disappear spontaneously. Its subjective symptoms are of no great severity, being limited to itching, tingling, and smarting, and more or less uneasiness in moving the limbs, or from pressure when sitting or lying in bed. It has no tendency to desquamation or ulceration.

TREATMENT.—Cheloid which forms in scar tissue should never be removed by the knife or caustics, as it almost always returns quickly after removal. It has a tendency to undergo slow atrophic changes if left to itself, and these changes are favoured by the pressure of a plaster. The mercurial plaster is a very suitable one.

ERASMUS WILSON. ROBERT LIVEING.

**CHELtenham**, in Gloucestershire.—Common salt waters. See MINERAL WATERS.

**CHEMOSIS** (χήμη, a hole).—A swollen condition of the conjunctiva, caused by effusion into its tissue around the cornea, which thus appears as if placed in a hole or hollow. See EYE AND ITS APPENDAGES, Diseases of.

**CHEST, Diseases of the.**—Following the plan adopted in the general article on the ABDOMEN, it is proposed in the first place to give an outline of the diseases of the chest; and then to indicate the principal points bearing on their clinical investigation.

**GENERAL SUMMARY.**—The diseases of the chest may be conveniently divided into certain groups, namely:—

I. Diseases of the walls of the chest, or extending inwards from the walls.

II. Diseases of the respiratory apparatus contained within the chest-walls, namely:—  
1. Pleuræ. 2. Trachea. 3. Main Bronchi. 4. Lungs.

III. Diseases connected with the circulatory system, including:—1. Pericardium. 2. Heart. 3. Great vessels within the chest, both arteries and veins.

IV. Diseases originating in the mediastinal cellular tissue.

V. Diseases of the absorbent vessels or glands within the chest; of the thoracic duct; and of the thymus gland or its remains.

VI. Diseases of the œsophagus.

VII. Diseases of either of the important nerves traversing the chest.

VIII. Diseases of the diaphragm.

IX. Diseases encroaching upon the thoracic cavity from the abdomen or from the neck.

The particular diseases comprehended within most of the groups just mentioned are very numerous, and they will be found described in their respective articles. Affections connected with the chest constitute a large proportion of the cases which come under observation in practice. This will be readily understood when we remember that the thorax encloses organs essential to life, which are never at rest, and which are constantly more or less exposed to influences liable to injure them. They may be mere *functional disorders*, and to these the heart is especially prone; but serious *organic diseases* are also exceedingly common, and rank very high as causes of death. Moreover, they may either come under the category of *local* affections,

though even then they frequently depend upon some cause acting through the general system, such as exposure to cold or wet; or they arise in the course of some *general* malady. For instance, pulmonary complications are of common occurrence in connexion with fevers and various other diseases; the heart is implicated in a large proportion of cases of rheumatic fever; and malignant disease is not infrequently manifested by a local development of cancer in connexion with certain of the thoracic contents. The structures within the chest have an important mutual influence upon each other; and they may also be affected, either directly or indirectly, in connexion with local diseases involving other parts of the body, such as the abdomen or the central nervous system.

**CLINICAL INVESTIGATION.**—It may be confidently affirmed, that the means which we now possess for investigating diseases connected with the chest are so adequate and precise, that anyone possessing the requisite knowledge and skill, and who carries out the clinical examination properly, can, in the very large majority of cases, arrive at a diagnosis with certainty and practical accuracy. At the same time it must be remembered that every complaint referred to this region, however trivial it may appear to be, does require systematic and thorough investigation, otherwise very serious mistakes are constantly liable to be made. Moreover, chest-cases do come under observation occasionally which are obscure, and which may call for repeated examination before a correct diagnosis can be formed; and exceptional instances occur in which no satisfactory conclusion can be arrived at. The previous general condition of the patient, the hereditary history, and the account given of the origin and progress of the illness, often afford signal aid in the diagnosis of chest-affections, and ought never to be overlooked. The symptoms to which these affections give rise are necessarily various. Pain or other morbid sensations are very commonly complained of, but only in a comparatively few instances are these at all significant, and they can never be positively relied upon in making a diagnosis, while they are often absent in diseases of the most serious character. The important organs, namely, the lungs and heart, usually present more or less disturbance of their functions when they are affected, but grave diseases may exist without any such evidence. The different structures within the chest have a mutual influence upon each other, and thus other symptoms besides those connected with the part actually diseased are often apparent. For instance, the lungs and heart are thus very intimately associated; while aneurysms or growths often disturb these organs seriously, or interfere with the air-tubes, nerves, veins, and other structures. In consequence of more or less interference with the circulation,

various symptoms in parts remote from the chest are frequently originated; and distant organs may become the seat of organic lesions as the result of long-continued mechanical congestion, a new train of symptoms being thus set up. The general system may be in this manner affected; whilst pyrexia, wasting, and other general symptoms are often associated with chest-diseases. Lastly, morbid conditions within the thorax may directly affect the abdominal organs; or may actually make their way through the diaphragm into the abdominal cavity.

*Physical examination* constitutes an essential and most important part of the clinical investigation of the chest and its contents. Indeed, without such examination no certain and exact diagnosis can ever be made. This subject is fully discussed in other appropriate articles, and here it need only be mentioned that the methods of examination which are usually required, and which should in every case be practised, are:—1. *Inspection*. 2. *Palpation*. 3. *Percussion*. 4. *Auscultation*. Other modes which may be called for include:—5. *Measurement* (not uncommonly). 6. *Succussion*. 7. The use of *special instruments*, directed to the investigation of particular organs, such as the spirometer, the stethometer, cardiograph, sphygmograph, aspirateur, œsophageal bougie, &c. See PHYSICAL EXAMINATION; and the articles relating to the several organs.

FREDERICK T. ROBERTS.

**CHEST, Deformities of.**—Under this head are included all deviations in shape from the normal chest.

Deviations from the shape of the typical thorax are appreciable by careful physical examination. Of the various methods employed for this purpose, by far the most valuable are inspection and palpation. Although in some few cases it may be important to determine the exact amount of deformity by mensuration, there are very few deviations in shape or size of the thorax, the degree of which cannot be sufficiently estimated for clinical purposes by the eye and hand, without the aid of any special instruments for measuring.

Deformities of the chest may be due either to abnormality of the parietes, or to disease of internal structures.

**DESCRIPTION.**—Deviations from the form or size of the typical thorax may be either *general* or *local*—that is, the abnormality may involve the whole thorax, or a part only.

**I. General Deformities.**—1. *General Diminution.*—The chest may be too small—that is, diminished in all its diameters without being in other respects deformed. Diminution of the thorax simultaneously and uniformly in its antero-posterior and lateral diameters is effected mechanically by an increase in the obliquity of the ribs. The

smaller the chest (having regard to the height of the person) the more obliquely are the ribs arranged, and the more acute the angle formed between each of the true ribs (excepting the first) and its cartilage. The intercostal spaces of the true ribs are widened about the junction of the ribs with their cartilages, and at the same time the ribs posteriorly are approximated more closely to each other, the closeness of the approximation being in proportion to the diminution in the size of the thorax. The vertical diameter of the thorax is lessened by an increase in the height of the arch of the diaphragm. The very oblique position of the false ribs, and the height to which the diaphragm rises into the chest, cause several of the false ribs to lie in contact with the diaphragm, and thus no portion of lung is under these ribs. They are, practically speaking, no longer part of the chest-walls. The costal angles are diminished in proportion to the diminution of the size of the thorax—that is, to the obliquity of the ribs. The obliquity of the ribs also causes the shoulders and the sternal ends of the clavicles to droop, and at the same time to incline forwards; the upper part of the scapula is carried by the shoulder forward, the inferior tilted backward.

General and symmetrical diminution in the size of the thorax has one and only one cause, namely, small size of the lungs. Small lungs may be congenital, due to original conformation; or the consequence of atrophic degenerative changes incident to age. In both these cases the lungs are, in relation to the length of the ribs, disproportionately small, and, as a necessary consequence, the relatively too long ribs are arranged more obliquely than they are in a well-formed chest, and the diaphragm is pushed by the abdominal organs higher into the thorax. When the small size of the lungs is due to atrophy, the supra-clavicular fossæ are deepened and the vertical diameter of the chest proportionately diminished. In advanced life the congenitally small lungs are frequently reduced still further in size by the supervention of atrophous emphysema. The congenitally small lungs with the consequently small chest is one of the characteristics of tuberculosis—that is, of that congenital organisation in which tubercle is likely in subsequent periods of life to occur. Atrophous emphysema is especially common in those who have either manifested symptoms of tubercle in their youth, or belong to tubercular families. It is the congenitally small lungs of childhood which are prone to become the seat of tubercle in youth, and the subjects of atrophous emphysema in old age.

2. *General Enlargement.*—The thorax may be too large, increased in all its diameters, without being otherwise deformed. It is simply bigger than it should be, having regard to the height of the subject.

When the thorax is abnormally large, the ribs, instead of being more obliquely situated than natural, as they are in the small thorax, are placed more horizontally than they are in the normal thorax. The angle formed between each rib and its cartilage is greater than in health; while the intercostal spaces, especially the lower, are widened, and the ribs less closely approximated, the arch of the diaphragm is lessened in depth, and a considerable mass of lung lies under the lower false ribs, between them and the diaphragm. The chest is increased in all its diameters. The shoulders are raised. The costal angles are greater than natural.

Increase in the size of the whole thorax has but one cause, namely, increase in the size of the lungs. Increase in the size of the lungs generally, and pretty uniformly, is the consequence of disease, and of one disease only, namely, large-lunged or hypertrophous emphysema. When the increase in size of the thorax attending large-lunged or hypertrophous emphysema is moderate in degree, this is effected by the altered position of the ribs; but when the lung-disease is extreme, then a certain amount of the enlargement is caused by pressure on the inside of the chest during the violent expiratory efforts of severe cough.

**3. Irregular General Deformities.**—In the deformities above described the antero-posterior and the lateral diameters retain more or less perfectly their normal proportion—both are increased or both are diminished; in the former case the chest is on the whole more barrel-shaped than natural, but the deviation from the normal form is not considerable. If, however, the chest-walls are from any cause unduly soft or unduly rigid, then the actually or relatively soft portions will recede during each inspiratory act, and local deformity of the chest follows. The diameter of the chest at the part where the absolutely or relatively soft portion of the parietes is placed will be diminished. The special deformities of the chest which result are due, therefore, primarily to the state of the parietes, and are not, as those previously described, secondary to conditions of the lungs themselves.

*a. Diminution in the antero-posterior diameter of the thorax.*—The antero-posterior diameter of the thorax is frequently less than that of the normal thorax, the lateral diameter being proportionately increased. The chest has an oval form—it is flattened from before backwards.

The thorax flattened from before backwards is usually associated with small lungs, but the mechanical cause of the flattened form is the want of full resisting power in the ribs and considerable strength in the cartilages. These conditions of thorax are common in the subjects of tuberculosis.

The flattening of the thorax is increased by

all impediments to the free passage of air through the air-tubes. In some children suffering from even slight bronchial catarrh, the flattening of the chest is seen to be increased at each inspiration; and if the impediment to the entrance of the air to the pulmonary tissue be constant or extreme, not only is the flattening increased at each inspiration, but the sternum is also depressed, especially at its lower half, below the level of the costal cartilages, and thus the antero-posterior diameter of the thorax is still further diminished in the median line.

*b. Increase in the antero-posterior diameter of the thorax.*—In rickets the cartilages of the ribs are very firm, whilst the ribs themselves are softer than natural, and especially so near to their enlarged growing ends—the softest part of the ribs; that is to say, just outside the nodule formed at the spot where cartilage is in the process of growing into bone. The consequence of the extreme softness of the ribs at this part is that at each inspiration the weight of the atmosphere presses inwards the softest part of the ribs, while the sternum is borne forward by the firm cartilages. The result is great increase in the antero-posterior diameter of the thorax, and diminution of the lateral diameter at the parts corresponding to the softest part of the ribs. The depression of the softest part of each rib is increased by the want of resiliency of the softened structures.

A groove is thus formed in the thoracic walls just posterior to the rickety nodules; and this groove being deepened at each inspiration, the part of the lung adjacent is compressed in place of being expanded during the inspiratory act. At the same time, in consequence of the cartilages and sternum being thrust forward at each inspiration, air enters with undue force into the lung-tissue subjacent to these parts. The consequence of the excessive expansion of the anterior part of the lung is vesicular emphysema, and the recession during inspiration of the softened and imperfectly resilient and therefore deeply grooved part of the chest-wall leads to collapse of the subjacent pulmonary tissue; and, as the effect of these two conditions, the lungs, when the chest is opened, present a vertical groove corresponding to the groove in the chest-walls. The antero-posterior diameter of the thorax in rickets is still further increased by the curvature of the spine. The muscles are weak, the child is unable to sit upright, that is to say it is unable, in consequence of the weakness of its muscles, to support the weight of the upper part of its body; the bones of the spine are, in common with the other bones of the body, softened; and the result of the weakness of the muscles and the softness of the vertebræ is the dorsal bow.

When deformity of the chest is the result of undue softness of the chest-walls, the position of the solid organs subjacent to the

parietes is frequently perceptible to the eye. The liver supports the lower ribs on the right side, the heart supports the ribs and cartilages over it on the left side, and thus these organs cause local prominence of the chest-walls, without being themselves in any way abnormal.

In the so-called *pigeon-breast*, the antero-posterior diameter of the thorax is increased in the middle line, the lungs are small, the ribs and cartilages are firm, the ribs are placed obliquely and the chest-walls are flattened laterally, and the sternum as a consequence is thrust forwards; thus the chest in the pigeon-breasted has a triangular form, the apex of the triangle being the sternum. Impediment to the free entrance of air into the lower lobes of the lungs will favour the production of and increase the deformity. The chests of children who suffer from repeated attacks of bronchitis, but are otherwise healthy, are commonly the subjects of this deformity, while there is increased expansion and subsequent enlargement of the upper part of the chest, the lungs being more or less collapsed below and emphysematous above.<sup>1</sup>

*c. Transverse anterior constriction of the lower part of the thorax* is the consequence of small size of the lungs, or of imperfect inspiratory expansion, permanent or frequently recurring in youth. In these cases the lower ribs are little used in respiration, while below they are borne outwards or supported by the liver, stomach, and spleen, and thus an imperfectly formed transverse depression is produced in the front of the chest on a level with the base of the ensiform cartilage.

The deviations from the type of the normal thorax hitherto described are bilateral, and more or less symmetrical.

**II. Local, Unsymmetrical, and Unilateral Deformities.**—1. *Fullness of the supraclavicular region.*—The supraclavicular region, corresponding to the portion of the thoracic cavity above the clavicle, may be fuller than natural. The causes of this local bulging are—*a.* Development of adipose and cellular tissue. *b.* Distension of the deep-seated veins. *c.* Large-lunged emphysema, in which disease there is occasionally distension of that part of the cavity of the thorax which lies above the level of the clavicle. The distension is due to pressure on the inside of this part of the thoracic cavity; air being forced violently into the corresponding part of the lung during the powerful expiratory effort of cough.

2. *Depression of one supraclavicular fossa* is caused by any pathological condition of the apex of the lung which produces diminution of its bulk, for example, atrophous emphysema, or chronic consolidation of the apex.

3. *Elevation of one shoulder.*—Occupation is a common cause of elevation of one shoulder;

<sup>1</sup> Nurses should be taught not to compress the chest laterally when carrying a child.—EDITOR.

thus in clerks, who sit much at the desk, the left shoulder is permanently a little higher than the right, and the upper portion of the spine is slightly curved, the convexity being to the left; so in those who carry heavy weights on one arm, the opposite shoulder is elevated and the spine curved. Whatever necessitates an increase in the capacity of one side of the thorax causes elevation of the shoulder on the same side; thus, considerable dilatation of the heart, fluid in the pericardium, fluid in the pleura, aneurysm of the arch of the aorta or of the innominate, all lead to elevation of the shoulder. The shoulder is depressed and carried forward when, from any cause, the whole or upper part of one side of the chest is diminished in size, as when the apex of the lung is the seat of chronic pneumonia or chronic phthisis.

4. *Uniform dilatation of one side of the thorax* is due, with one exception, to fluid or air in the pleura. The exception is those rare cases of encephaloid cancer of the lung, in which the formation of cancer is uniformly diffused through the lung-tissue, and in amount so great that the lung 'infiltrated' with cancer very decidedly exceeds in bulk the healthy lung inflated with air by inspiration.

In uniform dilatation of one side of the thorax, the shoulder is raised, the ribs are placed more horizontally than on the healthy side, the intercostal spaces are widened, and the spine is slightly curved. When the enlargement is moderate in amount, the increase in capacity is effected by the altered position of the ribs; but when the increase in size is very considerable, then it is due in part to the pressure exercised by the air, fluid, or cancer-loaded lung on the inner side of the chest-wall.

5. *Uniform contraction of one side of the thorax* is the consequence of any pathological condition which leads to general and uniform reduction in the size of the lung, such as cirrhosis of the lung, infiltrated cancer of the lung, chronic tubercular disease of the lung, chronic pneumonia, or the change in the texture of the lung which follows long-continued compression by fluid in the pleura. When the whole of one side of the thorax is reduced in size, the shoulder on that side is depressed, the ribs are placed more obliquely and are more closely approximated than on the opposite side, the intercostal spaces are narrowed, and the spine is curved, often considerably, the concavity of the curve being towards the contracted side.

6. *Lateral curvature of the spine*, instead of being the consequence, may be the cause of deformity of the thorax: the ribs are then approximated on the side and at the part where the concavity of the curvature is placed, while they are separated and the shoulder is raised on the side of the convexity.

7. In *angular curvature of the spine* the deformity of the thorax varies with the seat

and the extent of the vertebral disease; but, speaking generally, it may be said that in angular curvature of the spine the antero-posterior diameter of the thorax is increased in proportion to the amount of destruction of the bodies of the vertebræ, and that the ribs are in a corresponding degree approximated.

8. *Extreme depression of the lower part of the sternum* is the consequence of softness of the cartilages of the ribs, and impediment to the free passage of the air to the pulmonary tissue. This deformity is never congenital, although the subjects of it often affirm it to be so; it may, however, commence to be formed directly after birth if there be a congenital impediment to the entrance of air into the lungs—for example, atelectasis.

This deformity may be the result of direct pressure. In certain occupations pressure has to be exerted on the lower part of the sternum—thus, some shoemakers use a wooden instrument which has to be kept in its place by pressure against the lower part of the sternum. For direct pressure to produce this deformity it must have been applied in early youth, while the parts are still flexible, and have been exerted frequently over a long period of time.

9. *Congenital deformities* of the thorax are few in number and are due to arrest of development—for example, cleft sternum, and defective formation of one or more ribs or cartilages.

10. *Unsymmetrical diminution in size of a part of the thorax* is produced by any pathological change which reduces the size of the subjacent part of the lung. All chronic inflammatory or congestive conditions of the apex of the lung, whether primary or the consequence of the concomitant of the formation of tubercle, are attended by diminution of the bulk of the part of the lung which is the seat of the lesion. Considerable loss of pulmonary tissue is usually accompanied by falling inwards of the chest-wall over the cavity.<sup>1</sup> The formation of a cavity is almost invariably attended by chronic inflammatory condensation, and this increases the local depression of the chest-wall. In chronic thickening of the pleura, the chest-wall at the part is, by the contraction of the fibrin, drawn inwards, and the lung subjacent to the thickened pleura being condensed, the chest-wall is also forced in during inspiration by atmospheric pressure. Hence, after pleurisy limited in extent it is common to find permanent flattening of the thoracic parietes at the base of the chest on the side affected.

In cancerous infiltration of the lung, limited in extent, the lung-tissue is sometimes so much condensed that the bulk of the cancer and lung are less than that of the healthy lung, and the chest-walls as a consequence are flattened over the seat of disease.

<sup>1</sup> It is said that a very large air-containing cavity may give rise to local bulging.

11. *Unsymmetrical localised bulging*.—If the ribs are, in relation to the size of the lungs, disproportionately long, and their cartilages soft, then one or more of the cartilages may be knuckled forwards; the cartilage, being compressed between the end of the rib and the sternum, bends in an angle outwards. Although the prominence is trifling, it often causes anxiety to parents and its subject. Local deformity of this kind is occasionally the result of repeated lateral compression of the chest-wall in the athletic sports of young boys, such as cricket.

All the diseases of the chest which are accompanied by general enlargement of both or one side of the chest, when localised, are attended by local bulging; thus, a common cause of abnormal fulness of the lower part of the left side of the thorax, posteriorly, is emphysema of the corresponding part of the lung; and a moderate amount of fluid in the pleura is attended by fulness of the lower part of the chest on the same side. In both these cases the ribs are raised into an abnormally horizontal position; the chest-walls are not pushed outwards, but the ribs are raised, and the intercostal spaces are to that extent widened. The ribs are put into the position which gives the greatest capacity to the thoracic cavities containing the fluid or the enlarged lung. Local bulging may be produced by aneurysm of the arch of the aorta or of the innominate artery; by growths, malignant or other, within the chest; by chronic pleurisy with effusion circumscribed by dense false membrane; by hydatids; or by abscess; and in all these cases the prominence is due to direct pressure on the inner side of the chest-wall, and to changes in the chest-wall itself.

Hypertrophy and dilatation of the heart and fluid in the pericardium are attended by fulness of the præcordial region. The bulging from these diseases is much greater in the child than in the adult. In these cases a little of the fulness is produced by a more horizontal arrangement of the ribs; but when the prominence of the præcordial region is at all considerable, it is the result of the pressure exercised by the fluid or by the large and powerfully acting heart on the inner surface of the corresponding part of the chest-wall.

At the part corresponding to the junction of the first and second bones of the sternum, opposite the cartilage of the second rib, the sternum projects forward. This prominence is called the angle of Ludovicus. Any impediment to the free entrance of air into the lungs may cause depression of the lower part of the sternum; if the ossification of the sternum is not complete at the junction of the first and second bones, undue prominence of this part is the result. Subsequently a formation of bone takes place at this spot, and increases the prominence.

**CHEST, Examination of.**—See PHYSICAL EXAMINATION.

**CHEST-WALLS, Deformities of.**—See CHEST, DEFORMITIES OF.

**CHEST-WALLS, Morbid Conditions of.**—The walls of the chest may be the seat of various morbid conditions, and the affections of this portion of the framework of the body demand more attention than they are accustomed to receive. All that can be done within the limits of this article is to indicate their nature; to notice briefly such of them as are not described in other parts of these volumes; and to point out the principles of treatment. They may be practically considered according to the following arrangement:—

1. **Superficial Affections.**—Under this group are included morbid conditions of the skin and subcutaneous structures. (a) Cutaneous eruptions are of common occurrence over the chest. Among these may be specially mentioned the eruptions of some of the exanthemata, herpes zoster, and chloasma. (b) The superficial vessels are liable to become enlarged under certain circumstances. This enlargement is usually seen in the veins over the front of the thorax, which may be distended on both sides, or only on one side, or in some particular region. The larger divisions may alone be involved; or a more or less extensive network of smaller veins is visible, and occasionally even the capillaries seem to be implicated. This condition generally arises from some obstruction interfering with the circulation through one or other of the principal veins which, either directly or indirectly, receive the blood from the veins of the thoracic wall. Thus the superior vena cava, or either innominate, subclavian, or axillary may be implicated, being, for example, pressed upon by new-growths or enlarged glands, the distribution and extent of the venous distension varying accordingly. Occasionally one of the smaller veins is thus interfered with. The writer has met with cases in which considerable enlargement of the veins was visible over portions of the thorax, where the cause was by no means evident, the patients asserting that this condition had existed ever since they could remember, and being regarded by them as perfectly normal. Probably it has resulted from some local obstruction occurring during early life. It must be remembered that women who are suckling frequently present considerable enlargement of the superficial veins over the front of the chest, which usually subsides when the period of lactation is at an end, but may become permanent after several children have been nursed. Again, more or less venous and capillary engorgement in this region may occasionally be observed in cases of cardiac or pulmonary disease. Sometimes a ring of enlarged veins and capillaries is seen round

the lower part of the chest. When the venous distension is due to obstruction of the vena cava superior, the skin presents a more or less marked cyanotic tint; and in cases of general cyanosis, the chest, in common with other parts, may have a cyanotic appearance. Occasionally one or other of the small arteries which supply the thoracic walls is enlarged, and it may attain a considerable size. (c) Subcutaneous œdema is sometimes observed over the chest. In most instances this is a local condition, being the result of venous obstruction; but it may be a part of general dropsy, particularly in connexion with renal disease. This morbid state is evident on inspection and palpation, and the affected part pits on pressure. (d) The thorax is one of the most common seats of subcutaneous emphysema, in which gas, consisting usually of air which has escaped from the respiratory apparatus, collects in the cellular tissue, and travels, to a greater or less extent, over the body. The causes of this condition are described elsewhere (see EMPHYSEMA, SUBCUTANEOUS). Subcutaneous emphysema is attended with evident swelling of the part, which usually develops rapidly, and may be very great, all the normal anatomical outlines being obliterated. It has an elastic feel, and pits readily on pressure with the finger, but speedily rises again; the peculiar minutely crepitant sensation accompanying this condition is elicited on palpation and percussion; percussion yields a curious, superficial, muffled, tympanitic sound; and on auscultation a superficial small, dry, crepitant sound is heard. (e) As belonging to the superficial affections of the chest may be just mentioned diseases of the mammary gland or nipple, which of course constitute a most important class of diseases in females. See BREAST, Diseases of.

2. **Muscular and Tendinous Affections.**—(a) The muscles of the chest or their tendinous attachments may be the seat of certain painful affections. These are of the nature of so-called muscular rheumatism or myalgia, of inflammation, or of more or less injury or strain; being induced by cold, constitutional conditions such as gout, overwork, straining, violent coughing, fatigue from prolonged sitting, and other causes. The painful condition is usually localised, but different muscles are involved in different cases, sometimes those which are superficial being affected, in others the deeper muscles, including the intercostals; or the complaint may be confined to a single muscle. Pleurodynia, dorso-dynia, and scapulodynia are the terms applied to muscular rheumatism affecting the side of the chest, the upper part of the back, and the scapular regions respectively. As the result of severe coughing, muscular pains are very common around the lower part of the chest. The pain is usually more or less aching in character, and not severe, but it may be very intense, especially in acute cases. Whatever

brings the affected muscles into play aggravates the suffering, such as moving the arms or shoulders when the superficial muscles are affected, coughing, sneezing, and similar actions. In some instances the pain is not felt when the affected structures are kept quite at rest. There may be local tenderness on pressure, or diffused pressure may give relief; while posture often influences the sensations experienced, such as whether the patient assumes the recumbent or sitting posture, or lies on one or other side. Fatigue generally increases the pain. Muscular affections connected with the chest are not necessarily accompanied with any other local symptoms; and physical examination reveals nothing, except that perhaps the act of breathing is voluntarily restrained, on account of the pain thus induced. (b) The muscles of the chest may be affected, on the one hand, with spasm or cramp; on the other, with paralysis. The former is attended with more or less pain, usually localised, which may be very severe; the latter is indicated by loss of power in the muscles involved. These disorders generally depend on some lesion of the central nervous system, but may result from local nerve-disease. Muscular cramp may arise from a sudden twist of the body, and it is usually supposed to be the cause of the 'stitch' in the side brought on by running, but this is doubtful. In cases of hemiplegia from cerebral mischief, the muscles of the thorax on the affected side are often temporarily weakened, but they usually subsequently soon regain their power. When the upper part of the spinal cord is injured or diseased at a certain point, all the muscles of the chest become paralysed, which is a very serious matter, as respiration and the acts connected therewith cannot be carried on, except by the diaphragm, and consequently the blood is imperfectly aerated, secretions accumulate in the lungs, respiratory paralysis follows, and the patient dies of apnoea. (c) Atrophy or degeneration may involve more or less of the thoracic muscles. In cases of pulmonary phthisis either the whole of these muscles or certain of them are not uncommonly wasted out of proportion to the general emaciation. They may also be implicated in progressive muscular atrophy; and occasionally a single muscle undergoes marked wasting. The writer has seen a striking example of this local atrophy in connexion with the pectoralis major, but the serratus magnus or other muscles may be implicated. The wasting is probably in most cases due to disease of the nerves supplying the affected muscles. It is quite evident on examination, and the movements which are usually performed by the involved structures cannot be executed properly. (d) On the other hand, the chest-muscles may become hypertrophied. This may be a natural result of athletic exercises and training; or it may occur in consequence of their being

called upon, either habitually or at intervals, to act excessively, as in cases of emphysema or asthma. In exceptional instances the condition known as pseudo-hypertrophic muscular paralysis has extended up to the chest. (e) The thoracic muscles not uncommonly present marked irritability under percussion or friction. This has been regarded as an important sign of phthisis; but the writer has often found it equally if not more evident in cases where the lungs were perfectly healthy. (f) As the result of injury and other causes, some portion of the muscular structures of the chest may be lacerated, ruptured, or perforated, either alone or along with other structures. This condition will be further alluded to presently.

**3. Nervous Affections.**—(a) Neuralgia is very common in different parts of the chest, especially in the side, and particularly the left side—*intercostal neuralgia*. The pain is localised, being usually referred to a point where a branch of nerve becomes superficial. It is more or less constant, in some cases being at times very severe. It often precedes or follows an eruption of herpes. It may be increased by deep breathing or coughing, but is not, as a rule, so much affected by these and similar actions as are other painful chest-affections. Shooting and darting sensations often radiate from the principal point, and certain spots of tenderness—*points douloureux*—may be recognised (see INTERCOSTAL NEURALGIA). (b) Intercostal neuritis is occasionally met with, and this affection is attended with great pain, localised and radiating, with much tenderness, the suffering being aggravated by whatever causes any local disturbance. It is probable that the severe pains experienced in some instances where the complaint is supposed to be muscular, are due to branches of nerve being inflamed. Severe pain often precedes or follows herpes zoster, and this probably depends upon actual nerve-changes. It may also be mentioned here that pains around the sides may be associated with disease of the spine, or of the spinal cord. (c) Some individuals, especially females, exhibit a remarkable superficial tenderness or hyperæsthesia over the thorax or in parts of this region, especially the anterior and upper portions. The slightest touch is resented, and the most delicate percussion cannot be borne. This condition may exist without any actual disease, or it is sometimes observed in phthisical cases. (d) Sensation may be more or less impaired over the chest, or in limited portions, either in connexion with central or local nervous disease, or in hysterical and neurotic persons. Various paræsthesiæ are also frequently referred to this region by the class of individuals just mentioned.

**4. Diseases of the Bones or Cartilages.**—The morbid conditions which may be referred to the bony and cartilaginous

framework of the thorax are as follows: (a) The chest-walls are often unduly rigid and firm, owing to an excessive deposit of calcareous matter in the sternum and ribs, with ossification or petrification of the cartilages. This is a normal condition in old people, being one of the degenerative changes to which they are liable, but it also occurs not uncommonly in younger individuals, as the result of hard work, or excessive athletic exercises, or in connexion with certain pulmonary diseases. This state of rigidity interferes more or less with the respiratory movements, and not infrequently causes serious embarrassment. (b) On the other hand, the ribs and cartilages may be deficient in firmness, and consequently too yielding and elastic. This is observed in children and young persons who are ill-nourished, but especially in connexion with rickets, and it becomes a condition of great moment when any disease sets in which causes obstruction to the entrance of air into the lungs, such as bronchitis. The chest-walls are then liable to fall in more or less during the act of inspiration, and may become permanently deformed, 'pigeon-breast,'<sup>1</sup> 'rickety,' and other abnormal forms being thus originated (see CHEST, Deformities of). (c) Acute or chronic periostitis or perichondritis is sometimes observed in connexion, respectively, with the sternum or ribs, or with the cartilages. The acute affection gives rise to limited pain and tenderness, which may be accompanied with superficial redness and swelling, and may simulate some more serious disease. The chronic complaint usually assumes the form of a node, being the result of syphilis. The writer has occasionally observed a small swelling at the junction of one of the ribs with its cartilage, painless, unaccompanied with redness, but with fluctuation. (d) The bony and cartilaginous structures themselves may be the seat of disease in some part of the chest, and here must be included the portion of the spinal column which limits this region posteriorly. Thus there may be acute inflammation, caries, necrosis, or so-called scrofulous disease. Among the more important causes which may originate these conditions are injury, syphilis, scrofula, empyema opening externally, and thoracic tumours or aneurysms growing outwards. They may lead to serious consequences, both local and general, and frequently cause more or less deformity of the chest. (e) Permanent thickening and distortion of portions of the ribs occur after fractures which have united improperly.

**5. Inflammation and Abscess.**—It is expedient to make a separate group of those cases in which inflammation, resulting in the formation of one or more abscesses, occurs in

<sup>1</sup> A condition often produced by pressure of the nurse's hand in holding the child.—EDITOR.

some portion of the soft structures entering into the construction of the chest-walls. This may be of local origin, arising from injury, bone-disease, or other causes; or it may be due to the opening of an empyema into the tissues; to suppuration extending and burrowing from the axilla or other parts; or to pyæmia. If deep-seated, an abscess may be difficult to detect with certainty, but usually the signs of this condition become sufficiently obvious. Sinuses or fistulæ may be left as a consequence of suppuration in the chest-wall, especially when pus makes its way outwards from within.

**6. Tumours and New-Growths.**—These morbid conditions also demand brief notice. They may be connected with any of the structures of the chest-walls, and are of various kinds; among those which have come under the writer's notice may be mentioned molluscum, fatty growths, cystic tumours, enlarged sebaceous glands, and infiltrated carcinoma. Tumours may make their way inwards from the chest-walls, encroaching upon the cavity of the thorax; or, on the other hand, the walls may be involved by growths extending from within. It need scarcely be mentioned that mammary tumours constitute a distinct, and by far the most important group associated with the structures covering the chest. See BREAST, Diseases of.

**7. Perforations and Ruptures.**—The muscular structures of the thoracic wall may be more or less destroyed in some part, either by sudden rupture or gradually, allowing a hernial protrusion of the lung to take place between the ribs. As already noticed, empyema may make its way externally through the chest-wall. Aneurysms and tumours extending outwards from within the thorax frequently cause serious destruction of the tissues, including the bony and cartilaginous, as well as the soft structures. This destructive process is often attended with severe pain and suffering, and leads to grave mischief. Congenital clefts or more extensive deficiencies are sometimes met with in the sternum or other parts of the thoracic walls.

**8. Variations in Form and Size.**—The chest often presents deviations from the normal shape and size, and these are so important that they demand separate consideration. See CHEST, Deformities of.

**TREATMENT.**—In many cases where the chest-walls are in a morbid state, they either do not need any special treatment, or no treatment can be of any avail. The chief circumstances under which the practitioner may be called upon to interfere, and the measures to be adopted, may be briefly indicated as follows:—

(a) Painful affections of the chest-walls, depending upon conditions of the muscles or nerves, frequently demand local applications

for their relief. Thus in different cases it may be requisite to employ hot fomentations, dry heat, or cold applications, in the form of wet rags frequently changed, ice, or evaporating lotions; or to use anodyne applications of various kinds, such as belladonna plaster or liniment, opium plaster or liniment, oleate of morphine, tincture or liniment of aconite, or ointment of aconitine or of veratrine. Anodynes may also sometimes be added to fomentations with advantage. Friction is often of much service, and at the same time stimulating liniments may be employed, such as one containing camphor, chloroform, or turpentine. For ill-defined muscular pains about the chest, which are frequently complained of, free douching with cold water every morning, followed by friction with a rough towel, is often highly efficacious. When pain is localised and obstinate, much benefit may in many cases be derived from the application of a sinapism, mustard-leaf, or even a small blister. In other instances the use of the ether-spray is serviceable, repeated more or less frequently; or sometimes much relief may be obtained from applying over a painful spot a mixture of equal parts of chloroform and belladonna liniment. Galvanism is another agent which may be of the greatest service in relieving painful sensations about the chest, whether connected with the muscles or nerves. Subcutaneous injections of hot water, morphine, or atropine may be demanded in some cases, and constitute a most valuable mode of treatment if pain cannot otherwise be assuaged.

In the treatment of many painful affections of the chest-walls much assistance may be derived from attention to posture, especially in connexion with certain occupations; from the avoidance of undue fatigue, or of any violent actions which are known to influence this part, such as cough; and from the adoption of measures tending to support the structures, or to keep them in a state of rest. The writer has found great benefit in a large number of instances from strapping the side more or less extensively, in the manner advocated by him for the treatment of pleurisy; and if there is any localised pain, some limited anodyne application, such as a piece of belladonna plaster, may be placed over this spot under the strapping.

(b) In many affections of the chest-walls, treatment directed to the general system, or to some special constitutional condition, is often of the greatest service. Thus, in the painful complaints already noticed, there are frequently marked general debility and anæmia; and essential benefit is derived from the administration of quinine, preparations of iron, strychnine, cod-liver oil, preparations of phosphorus, and other tonic medicines, or such as improve nutrition. Some of these are also most useful when there is disease of bone and its consequences.

Again, certain affections of the thoracic wall may be associated with rheumatism, gout, or syphilis, and then the particular treatment indicated for these several diseases is called for.

(c) When disorders of the muscles of the chest-walls occur, such as paralysis or spasm, associated with some disease of the central nervous system, the treatment must usually be directed to this disease, and but little can be done for the local disturbance. In some instances, however, electrical or other modes of treatment may be of some service, by influencing the action of the muscles, but no definite rules can be laid down.

(d) Local inflammation and its results in connexion with the thoracic walls must be treated as in other parts of the body, but it is unnecessary to discuss this subject in the present article.

(e) Surgical treatment may be called for under certain circumstances. Of course this will be the case if the chest-walls are injured in any way. Among other conditions likely to demand surgical interference may be specially mentioned subcutaneous emphysema, abscesses, disease of the bones, and tumours.

FREDERICK T. ROBERTS.

**CHEYNE - STOKES RESPIRATION.**—A peculiar disturbance of breathing, first described by the late Drs. Cheyne and Stokes, of Dublin. See RESPIRATION, Disorders of.

**CHICKEN-POX.**—SYNON.: Varicella; Fr. *La Varielle*; Ger. *Wasserpocken*.

**DEFINITION.**—An acute specific infectious disease, characterised by the appearance, in successive crops, of red spots, which in the course of about a week pass through the stages of pimple, vesicle, and scab.

**ÆTIOLOGY.**—The origin of this disorder is unknown. It is certain that it arises from contagion, and that childhood is its predisposing cause. It occurs in children at the breast, and is seen with increased frequency up to the fourth year, at which period it attains its maximum. It is less often found between four and twelve, and after twelve it may be said to disappear, although it is occasionally seen in adults.

**SYMPTOMS.**—The illness commences with out any, or with but slightly-marked, premonitory symptoms. There is usually some feeling of lassitude, and the patient goes to bed earlier than usual. Within a few hours an eruption appears, generally on some part of the back or chest, but there are many exceptions to this rule. It may commence on the face, neck, chest, abdomen, or extremities, or upon several of these parts at the same time. The eruption consists of small, faintly papular rose-spots, varying in number from twenty to one or two hundred. These, in the course of eight, twelve, or, at the most, twenty-four hours from their appearance, change into

vesicles, which, at first small in size and clear as to their contents, become quickly large; globular, or semi-ovoid, in form; translucent, glistening, and opalescent in appearance; and surrounded with a faint areola. Towards the end of the second day of illness, the vesicles attain complete development, and about this time a few may be seen on the sides of the tongue, on the lips, cheek, or palate, and sometimes upon the mucous membrane of the genitals. About the third day a few of the vesicles may have a pustular appearance, and sometimes a few pustules are seen; but, regarding the eruption as a whole, pustulation forms an incident rather than an essential feature in its progress. On the fourth day the vesicles begin to dry up, and by the sixth complete scabs are formed. These fall off in a few days, leaving in their place faintly red spots, and sometimes a few pits. A single crop of eruption may be said to complete itself in five or six days; and, as two or three crops appear on as many successive days, the illness will last rather more than a week. In the event, however, of there being four or five crops, it may be prolonged for another week, but this is unusual. With the appearance of the eruption, the temperature rises two, three, or even more degrees, and this rise recurs with each successive crop of spots. The pulse is sometimes slightly increased in frequency; the tongue is moist, and sometimes covered with a light fur. As a rule, however, there is but little constitutional disturbance, although it is occasionally severe.

**PATHOLOGY.**—Chicken-pox is due to the reception of a specific poison, which after an incubation of about thirteen days, shows itself by an eruption upon the skin. What this poison is, how it enters the body, and what, if any, changes it produces upon the internal organs, the present state of our knowledge does not enable us to say. It affects the same individual once only, and it is perfectly distinct from modified small-pox, as the following considerations will show:—1. Chicken-pox is characterised by the *rapidity* with which it runs through its stages; modified small-pox, on the contrary, is characterised by an interruption in the course of the disease at one or other of three points—the papular, the vesicular, or the pustular. 2. The chicken-pox eruption attains complete development by the end of the third day; in modified small-pox, should the eruption attain complete development, this will not occur before the ninth day, however much the disease may be modified. 3. In modified small-pox the premonitory symptoms are usually well-marked, often quite as severe as in the natural disease, and these last forty-eight hours, after which there is an eruption of small hard papules on the forehead, face, and wrists, followed by a *fall* of

temperature. In chicken-pox the premonitories are most often wanting, and when present are slightly marked, and the eruption is followed by a *rise* in the temperature. It appears, moreover, upon any part of the body indiscriminately, and less frequently on the face than on other parts; and within a few hours—at the most within twenty-four—it has become vesicular; whereas in modified small-pox the vesicular stage is only reached forty-eight hours after the appearance of eruption. 4. The vesicles of chicken-pox are globular or ovoid in form, without any central depression; glistening or translucent in appearance; and unicellular in structure. They collapse on pricking; and attain their maximum development in from twelve to eighteen hours. Modified and natural small-pox vesicles are flat and circular in form, always depressed in the centre, and sometimes umbilicated, of an opaque dirty white colour, and multicellular in structure. They do not collapse on pricking, and attain their maximum development at the end of the third day from their origin. 5. Small-pox is an inoculable affection; chicken-pox, according to reliable authority, is not. 6. When cases arise which all recognise to be modified small-pox, they are always accompanied by others which are more severe; and in epidemics these latter gradually become more numerous up to a point of maximum intensity, when they decline and the modified forms reappear. In chicken-pox there is no such gradual increase in the intensity of illness, and neither serious nor fatal cases form part of its epidemics, which prevail independently of small-pox. 7. Small-pox and vaccinia are often early followed, in the same individual, say within two or three years, by chicken-pox, and *vice versa*. 8. Chicken-pox, vaccinia, and small-pox have been known to follow in immediate succession in the same individual.

**COURSE AND TERMINATION.**—Varicella always runs a favourable course. It has no sequelæ.

**DIAGNOSIS.**—It should be borne in mind that a sure diagnosis cannot be made in less than forty-eight hours. The appearance, however, of a crop of vesicles, followed on the next day by a second crop, points almost certainly to chicken-pox. Attention to this, and to the points noted under the head of Pathology, ought to make the diagnosis easy.

**PROGNOSIS.**—This is always favourable.

**TREATMENT.**—The patient should be confined to his room, perhaps in the more marked cases to his bed. His food should be that which is easy of digestion; and although no physician has recorded a fatal case, a child whose temperature rose to three, four, or six degrees above normal should be examined with care.

**CHIGOE.**—DESCRIPTION.—The Chigoe is a minute parasitic insect, common in the West Indies and northern parts of South America. It is also popularly known as the *Jigger* or *Sandflea*. Though formerly regarded as an acarus or mite, it is now generally recognised as a true flea, belonging to the genus *Pulex* (*P. penetrans*); but several entomologists have advanced solid reasons for separating it from the ordinary fleas. Thus Westwood terms it the flesh-flea, or *Sarcopsylla penetrans*, whilst Guérin formed the genus *Dermatophilus* for its reception. Practically, these distinctions are of little moment.

The Chigoe ordinarily lives in dry and sandy situations, where it multiplies to a prodigious extent. It attacks, however, the feet, chiefly underneath the nails and between the toes; the impregnated females burying themselves beneath the skin. Here the abdomen of the parasite swells to the size of a pea; and, unless removed by operation, gives rise to acute local inflammation, terminating in suppuration and sometimes in extensive ulceration, with even fatal results to the patient. Dogs also suffer excruciating torment from the bites and immigration of the *Bicho do Cachorro*, which, however, Pöhl and Kollar regard as distinct from the human jigger (*Bicho de pé*). Be that view correct or not, it would appear from the observations of Rodschild and Westwood that the larvæ of the human chigoe are hatched in the open wounds or ulcers, which sometimes extend inwards so as to involve the bones themselves. In bad cases amputation of the toes and adjacent parts becomes necessary. Left to themselves, the larvæ escape from their host, and probably, after the manner of bots and other parasitic insects, penetrate the soil for the purpose of acquiring the pupal stage of growth. In European practice cases of jigger are rarely seen; nevertheless the writer has recorded an instance in which strong mental delusions followed the torture produced by these creatures. The patient, a middle-aged married lady, had suffered severely during her residence in the West Indies. Although she had got rid of the parasites, she constantly harpooned her own feet in the hope of destroying the young jiggers which she felt sure were still burrowing beneath the skin.

**TREATMENT.**—As regards treatment, the indications are simple. The parasite should be removed with the utmost care. Where this has not been done, and where, as a consequence, open sores exist, frequent washings with tepid water, followed by the application of carbolic acid lotions (twenty or thirty grains to the ounce) or of ointments (one drachm to one ounce of benzoated lard) will be found most suitable. Or, again, the carbolic acid putty, as sold in shops, or the application of one part of the acid previously mixed with ten or twelve parts of simple olive oil, will,

in all likelihood, be sufficient to cause the destruction of any larvæ that might remain. Lastly, it seems almost needless to say that residents and travellers in Guiana, Brazil, and in the West Indies generally, should have their feet properly protected.

T. S. COBOLD.

**CHILBLAIN.**—SYNON.: Kibe; *Pernio*; Fr. *Engelure*; Ger. *Frostbeule*.

**DEFINITION.**—A state of inflammation of a part of the skin induced by cold.

**ÆTIOLOGY.**—Chilblains are common in children and young persons, and are more frequent in girls than boys. They occur chiefly in those of a lymphatic constitution, and may be considered as an indication of debility and deficient vital power. In adult age they are rare, and are only met with when the powers of the constitution are reduced. Their occurrence is influenced more by the strength of the individual than by the degree of cold, and they continue in some persons throughout the entire year. Their tendency is to cease with the full development of the system, and they reappear occasionally in advanced life.

**DESCRIPTION.**—The regions of the body usually affected with chilblains are the feet and hands, to which are sometimes added the ears and nose. A chilblain presents three stages or degrees of severity, namely, *erythematous*, *bullous*, and *gangrenous*; and it may be arrested at the first or second stage by the withdrawal of the cause. The *erythematous* stage is restricted to hyperæmia, swelling, and severe burning and itching, the itching being increased by heat, as by that of the fire or that induced by exercise. The congested spot is circular in figure, somewhat tumid, brightly red at first, but later on roseate crimson, purple, or livid in colour. The second or *bullous* stage exhibits the blain or blister resulting from effusion of serum beneath the cuticle; the permanent colour of the swelling is now purple or livid; and the contents of the blister a limpid serum, generally reddened with blood—sometimes, indeed, the fluid of the blister may be semi-purulent. In the *gangrenous* stage the blister is broken, the surface of the derma is in a state of gangrene, and the gangrenous layer is subsequently removed as a slough by ulceration.

**TREATMENT.**—The treatment of chilblain requires to be modified to suit its different degrees. In the first, the indication is to restore normal circulation by gentle friction, and, when the part is severely chilled, it is usual to rub it with snow: then some soothing liniment may be employed; and, finally, a stimulating liniment, covering the part afterwards with zinc ointment and cotton-wool, or shielding it with lead or opium plaster spread on washleather. The applications most in favour for this purpose are the

soap liniment with chloroform and laudanum, the compound camphor liniment, the turpentine liniment, and the tincture of iodine. In the bullous stage a similar treatment may be used to the erythematous portions, whilst the blister should be snipped and the broken surface pencilled with the compound tincture of benzoin, and afterwards dressed with unguentum resinæ or an ointment of Peruvian balsam. In the third stage the erythematous phenomena still require attention, and the ulcer should be dressed with unguentum resinæ, either alone, or in combination with antiseptics.

To obviate constitutional debility, the diet should be nutritious and generous, and recourse may be had to tonic remedies, such as iron and quinine and good air.

ERASMUS WILSON.

**CHILL.**—A subjective sensation of coldness, accompanied with shivering, and most frequently experienced in connexion with febrile or inflammatory diseases, in nervous individuals, and after exposure to cold and wet. In popular language 'taking a chill' is used as synonymous with 'catching a cold,' as from some undue exposure. See RIGOR.

**CHIN-COUGH.**—A synonym for whooping cough. See WHOOPING COUGH.

**CHIRAGRA** (χείρ, the hand; and ἄγρα, a seizure).—Gout in the hand. See GOVT.

**CHLOASMA** (χλόα, a green herb).—SYNON.: Liverspot; Fr. *Ephelide*; Ger. *Leberfleck*.

A pigmentary discoloration of the skin, of a yellowish-brown or liver-colour tint, occurring in blotches, and due to constitutional causes. Its synonym, *epheles gravidarum*, indicates its occasional association with pregnancy. See PIGMENTARY SKIN DISEASES.

**CHLORAL HYDRATE, Poisoning** by.—SYNON.: Fr. *Empoisonnement par l'Hydrate de Chloral*; Ger. *Chloralhydratvergiftung*.

Poisoning by chloral hydrate is a very common occurrence, this medicament being frequently taken in fatal quantity by misadventure. There is reason to think that it is also largely used for suicidal purposes. The so-called 'chloral habit' is a growing evil. A syrup containing about twenty-two grains of this substance per fluid drachm is largely and injudiciously sold in this country under a patent-medicine stamp.

**ANATOMICAL CHARACTERS.**—There may be an entire absence of any characteristic appearances after death by hydrate of chloral; and at most these consist in more or less modified signs of asphyxia—especially a dark colour of the blood, and pulmonary and cerebral hyperæmia.

**SYMPTOMS.**—The most striking symptoms of poisoning by chloral hydrate is the rapid

supervention of quiet sleep, at first simulating natural sleep. In this stage the patient can be easily roused, but he speedily drops off again. The pupils are contracted; the respirations are full, deep, and regular; the pulse is not much affected. This condition rapidly deepens into full coma. The respirations slacken; and the pulse is either weak and slow, or, more commonly, rapid and irregular. The temperature of the body is reduced; the muscular system is totally relaxed. The pupils now dilate; and with feeble, thready pulse, the anæsthesia and paralysis gradually end in death, preceded by lividity and collapse. Exceptionally, in fatal cases, burning pain in the mouth, fauces, and throat, and symptoms of gastritis have been observed. In one case of recovery the patient became idiotic. The urine contains an abundance of glycuronic acid.

**DIAGNOSIS.**—The history of the case, or the finding of a vessel containing the medicine, coupled with the symptoms, will usually set all doubts at rest. Otherwise the case may be mistaken for poisoning by opium or other narcotic, for carbolic acid poisoning, or for cerebral congestion. The pupil is not so contracted as in opium poisoning; and, as the coma deepens, the pupils dilate instead of undergoing further contraction. There is an absence of the olive-green or black urine so commonly noticed in carbolic acid poisoning, of the peculiar odour of the breath, and of stains about the mouth and lips.

**PROGNOSIS.**—This will depend upon the state in which the patient is found, and upon the length of time which has elapsed since the ingestion of the poison.

**TREATMENT.**—Evacuation of the stomach by the aid of the stomach-pump is the first step in the treatment of a case of poisoning by chloral hydrate. Emetics, unless given early, usually fail to excite vomiting. The patient must be roused, if possible, as in opium poisoning (see OPIUM, Poisoning by). The temperature of the body must be kept up by warm applications. Stimulants may be freely given, and hot coffee injected into the rectum. Strychnine ( $\frac{1}{32}$  gr.) has been recommended for use as a counter-poison, by subcutaneous injection; also picrotoxin. The former is a dangerous remedy; the latter also would not perhaps be a safe antidote, if given in full doses. Inhalations of amyl nitrite, and artificial respiration are advisable.

THOMAS STEVENSON.

**CHLOROFORM; Use of.**—See ANÆSTHETICS.

**CHLOROSIS** (χλωρός, green or sallow). SYNON.: Green-sickness; Fr. *La chlorose*; Ger. *Chlorose*; *Bleichsucht*.

**DEFINITION.**—A variety of anæmia occurring in a peculiar diathesis or habit of body, which is characterised by defective

growth of the blood-corpuscles and vascular system.

The subjects of this diathesis are said to be *chlorotic*. They ordinarily enjoy good health, unless exposed to any of the causes of anæmia, when they readily suffer from aglobulism; and this aglobulism may proceed to complete anæmia. The term *chlorosis* is properly applied to the first and simpler form of anæmia in these subjects; the second and more complex condition is designated *chlor-anæmia*; or, more commonly, *anæmia*. See ANÆMIA.

**ETIOLOGY.**—Chlorosis occurs most commonly in young women from the age of puberty to twenty-one, but it may appear or reappear up to the thirtieth year; it is found occasionally in children and older women; it is very rare in men. It is believed to be more common in the higher ranks of life. Beyond these predisposing causes, the origin of the disease lies in peculiar characters of the blood and blood-vessels, to be presently described, which are believed to be congenital, and perhaps hereditary. In such subjects, and under the preceding circumstances, any of the numerous causes of anæmia may be sufficient to excite the appearance of chlorotic symptoms; but those which do so most commonly are bodily growth and development, the establishment of menstruation, disturbances of alimentation—particularly constipation, and an insufficient enjoyment of air, light, and wholesome muscular exercise.

**ANATOMICAL AND CHEMICAL CHARACTERS.**—The blood in chlorosis presents one essential imperfection, as far as our present knowledge extends. The individual red corpuscle contains less than the normal amount of hæmoglobin, the total amount of hæmoglobin in the blood sometimes falling to less than one-fourth. With this defect there may be associated various other abnormalities. The total quantity of blood is below the normal, though there may be an excess in relation to the calibre of the vessels (*plethora ad vasa*). Both red and white corpuscles are sometimes deficient in numbers, and that proportionately. In pure chlorosis the quality of the liquor sanguinis is believed to be unchanged; but it is possible that certain complex organic substances exist in the blood which cannot be discovered by our present methods of examination.

With this condition of blood there are associated certain remarkable abnormalities of the aorta and arterial system generally. The most striking of these is a hypoplasia or dwarfed condition of the aorta, represented by small calibre, increased elasticity, anomalous origin of the branches, and unequal thickness and fatty metamorphosis of the intima. The heart is, as a rule, small in early life; but full-sized or even hypertrophied at a later period, with traces of endocarditis. The blood-glands and lymphatic structures

are not diseased. The condition of the ovaries and uterus has been carefully examined in chlorosis and found to vary extremely. In some cases the generative organs are described as 'infantile,' while in others they are either immoderately developed, or perfectly normal in every respect. Corresponding with the aglobulism, the subcutaneous fat is abundant; and the viscera present various degrees of fatty metamorphosis. When the cardio-vascular changes are marked and advanced, there may be extensive secondary disease throughout the body.

**SYMPTOMS.**—The symptoms of simple chlorosis are those of mild anæmia, with certain important differences which become fewer and less marked, and finally disappear, as chlorosis advances to the more serious disease. The present article will be chiefly devoted to a description and discussion of these.

The appearance of the chlorotic girl is peculiar, inasmuch as the pallor of her complexion is accompanied by natural or even increased fulness, from the excess of subcutaneous fat. At the same time the colour of the skin is so remarkable as to have given its name to the disease, the general hue being a greenish-yellow. In blondes the transparency of the skin is increased; in brunettes it is diminished, and a dull yellowish-grey colour of skin is the result, which, in contrast with the greyish-blue of the eyelids, may appear of a sickly green.

The patient's usual complaint is of this alteration of colour, menstrual disorder, debility, great breathlessness, cardiac symptoms, and various pains. She probably believes that she has heart disease; her mother fears that she is consumptive. Various menstrual disorders are present—namely, premature menstruation, amenorrhœa (with respect both to the period and the amount), occasionally menorrhagia, and leucorrhœa. Breathlessness on exertion is one of the most striking symptoms. The cardiac symptoms and the cardiac and vascular signs closely resemble those of anæmia. But there is this important difference in the phenomena connected with the heart, that in many cases of chlorosis they indicate enlargement, and especially hypertrophy of the left ventricle. The characters of the blood already described are readily discovered by the use of the hæmoglobinometer and hæmacytometer. Venous thrombosis may be found in the legs. The alimentary system is often seriously deranged, constipation being an urgent symptom. The urine is abundant, watery, and pale. There is no dropsy in simple, uncomplicated chlorosis. Rheumatic symptoms are occasionally associated. Pyrexia (99°–103° F.) has been observed in some instances. Optic neuritis may occur.

The *chlorotic constitution or diathesis*

may be recognised by the following characters, which are variously associated in different cases:—Diminutive stature; imperfect sexual development; a history of peculiar anæmia in childhood, of anæmia with menstrual irregularity at puberty, and of previous attacks of symptoms of chlorosis; evidence of cardiac enlargement or mitral disease in the absence of all the ordinary causes of these; the occurrence of endocarditis during pregnancy or *post partum*; and the presence of any of the diseases which will be referred to under the head of Complications.

**COURSE, DURATION, AND TERMINATIONS.**—The commencement of chlorosis is generally gradual, but may be sudden. Its ordinary course is towards confirmed anæmia, in which it may terminate, the liquor sanguinis becoming affected, and wasting and œdema being added to the previous symptoms, which are also aggravated. It is for this reason that pure chlorosis is a rare disease, while anæmia associated with the chlorotic diathesis is comparatively common. The duration of the disease is variable; it rarely declines until the determining circumstances have been removed, and the patient subjected to careful treatment. Chlorosis may reappear in the subject of the diathesis, and that more than once; but the probability of its return is small after the age of thirty, especially in the married woman. Death from chlorosis directly is excessively rare.

**COMPLICATIONS AND SEQUELÆ.**—According to Virchow, serious valvular disease and cardiac enlargement may be traced in some of the worst cases of chlorosis to the associated vascular condition; and the mitral valve is peculiarly liable to be attacked by endocarditis in rheumatic, puerperal, or septic fever. Hæmorrhages, gastric ulcer, and exophthalmic goitre are believed to occur with comparative frequency in persons of the chlorotic diathesis.

**PATHOLOGY.**—The deficiency of the individual red corpuscle in hæmoglobin, and the deficiency of the blood in red and white corpuscles, indicate an imperfect growth of the red corpuscles and an imperfect production of the cellular elements of the blood. With this blood-state there is undoubtedly associated a hypoplastic or dwarfish condition of the blood-vessels. In the embryo the blood and blood-vessels are developed from the same elements, the former making its appearance within the cells which produce the latter. It is highly probable, therefore, that the anomaly of blood and the anomaly of vessels are to be considered as together an expression of some congenital defect of the blood-vascular system, leading to imperfect growth both of blood and of vessels. Any individual possessing a blood-vascular system thus anomalous labours under a peculiar diathesis, or debility of the corpuscles and circulatory system, and is said to be a *chlorotic*

*subject*, or to possess the *chlorotic constitution* or *diathesis*.

If the other systems of the body are full-sized (which is not always the case), the dwarfish condition of the arteries of the chlorotic subject and the scanty supply of hæmoglobin may have some difficulty in satisfying the ordinary demands for blood, and especially for oxygen. This difficulty of the blood-vascular system will become a breakdown when the processes of alimentation are deranged from which the blood derives the materials for its maintenance, or when the blood is wasted by the excessive demands of growth, development, or hæmorrhage. Such a result is more likely to happen at periods of extraordinary demand within the economy, of which the establishment of menstruation is the chief. It is in this way that exposure to the causes referred to is sufficient to produce the symptoms of chlorosis, when they might not affect the blood of an ordinary (non-chlorotic) individual.

The symptoms of uncomplicated chlorosis are due to aglobulism or deficiency of oxygen in the system (*see* Blood, Morbid Conditions of). When chlorosis advances to complete anæmia, by the implication of the plasma, a new series of phenomena present themselves, prominent among which are loss of flesh and œdema of the extremities.

The cardiac enlargement and valvular disease are directly referable to the vascular hypoplasia, that is, to the obstruction caused by the narrow calibre of the aorta. The connexion between the blood-vascular condition and that of the generative organs is more complex. On the one hand, the chlorotic diathesis or actual chlorosis interferes with the development and activity of the ovaries and uterus; on the other hand, disorders of the sexual functions are occasionally exciting causes of aglobulism.

A variety of suggestions have been offered as to the manner in which derangement of the alimentary processes in the bowels impoverishes the blood in chlorotic subjects. Sir Andrew Clark maintains that when the bowels are inadequately relieved, ptomaines and leucomaines are produced within the intestine and absorbed into the blood, 'where they originate in girls of a nervous type of organisation those alterations of the constitution of the blood which constitute the true pathogeny of this anæmia.' Another suggestion is, that sulphuretted hydrogen generated in the bowels destroys the organic compounds of iron which go to form hæmoglobin. According to a third suggestion, substances containing animal gum, which are developed in excess within the alimentary canal of the girl or woman, being required for the after-nourishment of the embryo, act injuriously on the hæmoglobin molecule. Still another theory is, that, from a deficiency of

hydrochloric acid in the system, iron enters the blood in forms which cannot be assimilated by the corpuscles.

**DIAGNOSIS.**—Chlorosis has chiefly to be distinguished from symptomatic and pernicious anæmia. The points by which the diagnosis may be accomplished have been sufficiently indicated. Leukæmia may be readily recognised by a careful examination of the blood and spleen. See also ANÆMIA, PERNICIOUS.

**PROGNOSIS.**—The prognosis is highly favourable as regards life; and a speedy cure may be assured in uncomplicated cases subjected to careful treatment.

**TREATMENT.**—The success of a particular method of treatment of simple chlorosis is one of the strongest arguments in favour of the correctness of the preceding view of the pathology of the disease. The condition being one of aglobulism, the treatment employed will be so far simpler than that of anæmia, that the red corpuscles alone have to be restored. While the various measures recommended in the more serious blood-disorder are therefore to be employed, if necessary, it will generally be found that iron alone will be sufficient to effect a cure in chlorosis, and that rapidly. The particular form in which the drug is to be presented must be carefully selected according to circumstances. These are fully set forth in the article ANÆMIA, and need not be repeated here. A free supply of sunlight is essential, and must be insisted upon; and physiological rest of the blood and of the organs of circulation is equally necessary.

J. MITCHELL BRUCE.

**CHOLÆMIA** (χολή, bile; and αἷμα, blood). **DEFINITION.**—This term literally denotes that condition in which the blood contains some or all of the bile constituents which have been secreted by the hepatic cells, and subsequently reabsorbed, either directly by the capillaries or *viâ* the lymphatics. It is usual to include the symptoms which this state induces under the expression ‘jaundice,’ which is more properly only one of the manifestations of the circulation of a bile-containing blood; and hence the word cholæmia has come to have a more restricted application, corresponding to the *icterus gravis* of older writers, or even to have fallen into disuse.

**SYMPTOMS.**—The symptoms of cholæmia are very variable in their occurrence and severity. Jaundice is certainly the most constant, and is elsewhere fully treated of. The derangements of digestion, which depend on exclusion of bile from the alimentary canal, are scarcely to be regarded as effects of vitiated blood. But the slow pulse, bitter taste in the mouth, pruritus, cutaneous eruptions, hæmorrhages, wakefulness, yellow vision, and certain nervous phenomena, some or all of which so constantly accompany jaundice, are obviously attributable to this cause.

The explanation of these symptoms is not altogether clear, owing in great part to our ignorance of the intimate nature of bile-secretion, and of the exact share taken by the liver in urea-formation and other metabolic processes. In obstructive jaundice the bile-pigments are soon absorbed from the occluded biliary passages, and are obviously the cause of the icteric discoloration of the skin and tissues. Later the bile-salts are diffused into the blood-current, and doubtless they are responsible for the slow pulse so commonly found in jaundice unassociated with pyrexia, and also for the destruction of blood-corpuscles and consequent petechiæ and other hæmorrhages, since these conditions are well known to follow the artificial injection of bile-salts into the blood. The bitter taste, as also the pruritus, are probably due to the same cause. Whether the various cutaneous eruptions are the expression of the local irritation of a bile-containing blood, or, as is more probable, of some trophic disturbance, determined by the toxic materials acting through the nervous system, cannot be affirmed. The most interesting and important symptoms of cholæmia, to which the term has sometimes been limited, are the nervous symptoms. It is well known that many cases of bile-retention, even of considerable severity, may run a prolonged course, and, except for the jaundice, be almost unaccompanied by other symptoms directly referable to the vitiated blood; but in some, even when the jaundice is but slight or even quite absent, nervous symptoms of a grave character supervene, often with considerable suddenness. The general character of these manifestations is an increasing drowsiness, which deepens into coma, usually fatal. Preceding this, a stage of excitement, with noisy violent delirium, may occur, or sometimes convulsions of varying severity. There is a noticeable absence of headache, in the writer's experience, offering therein a marked contrast to uræmia, with which by some the state now described has been considered identical. The general condition of the patient is that of the ‘typhoid state,’ with varying degrees of coma or convulsion. With our present knowledge it is extremely difficult to suggest the cause for these symptoms: the prevailing view is that they are toxic, the poison being developed in the body as a consequence of the perverted hepatic functions. But whilst it may be said with certainty that they are not due to accumulation of cholesterolin in the blood (cholesteræmia), as was formerly stated, it remains uncertain whether the poisonous material is actually excess of bile-salts, or, what is far more probable, of nitrogenous derivatives of proteid digestion, which, in the normal action of the liver, would be converted into bile-salts or urea. The close relationship existing between the bile-forming and urea-forming functions of

the liver should not be forgotten; nor also the fact that the bile is normally one of the most important channels whereby toxic substances are eliminated from the organism, whether these be poisons introduced from without or developed in the liver or other tissue as results of morbid metabolism. The suppression of the biliary secretion or its obstructed excretion readily explains how a blood-poisoning may be engendered, though the exact source and nature of the poison may not as yet be accurately known. Another explanation of these symptoms has been put forward by Cohnheim, who, comparing them to the delirium often seen at and after the crisis of acute diseases, attributed them to cerebral inanition, coupled with an excessive albuminous disintegration in the tissues.

**TREATMENT.**—This usually resolves itself into removing the cause of the bile-retention; when this is impossible, but little can be done for the resulting symptoms. The itching of the skin may sometimes be considerably allayed by alkaline baths, or by causing profuse sweating by pilocarpine or hot-air baths. It often subsides independently of treatment, to recur again with increased severity, especially at night. The internal administration of bicarbonate of potassium, a method commonly adopted in jaundice from most causes, also relieves the pruritus. Active diaphoresis, with a brisk purge and blisters to the nape of the neck, are indicated for the cerebral symptoms. Very little benefit appears to follow such remedies as the bromides if given for the convulsive seizures, but leeches to the temples are of occasional service if indicated. W. H. ALLCHIN.

**CHOLAGOGUES** (χολή, bile; and ἄγω, I move).—**DEFINITION.**—Substances which lessen the amount of bile in the blood.

**ENUMERATION.**—The principal cholagogues are Mercury and its preparations—especially Calomel and Blue Pill; Podophyllum and Podophyllin; Euonymin, Iridin; Ipecacuanha; Aloes; Rhubarb; Sodium Salicylate, Sodium Phosphate, and Sodium Sulphate.

**ACTION AND USES.**—The liver has a twofold action—it *forms* bile, which is poured into the duodenum; and it also *excretes* the bile which has been re-absorbed from the duodenum and carried back to the liver by the portal circulation. Much bile thus circulates continually between the liver and duodenum, while part is carried down the intestine with the fæces, and its place supplied by newly formed bile. When the quantity circulating in this way is too great to be completely excreted by the liver, it enters the general circulation and produces symptoms of *biliousness*. These are removed by the so-called cholagogues, which probably act by stimulating the duodenum, and thus carrying the bile so far down the intestine as to interfere with re-absorption. Amongst

the best cholagogues are the preparations of mercury, which, with the exception of the perchloride, do not increase the secreting power of the liver, nor augment the quantity of bile formed by it. Their utility is greatly increased by combination with a saline purgative, which still further clears out the intestine, and completely prevents any re-absorption of bile. Other cholagogues, such as podophyllin, rhubarb, and aloes, actually increase the secretion of bile by the liver. At the same time, they probably prevent its re-absorption, in a similar way to mercurials and salines. T. LAUDER BRUNTON.

**CHOLELITHIASIS** (χολή, bile; and λίθος, a stone).—The condition of system associated with gall-stones. See GALL-STONES.

**CHOLERA, ASIATIC.** — **SYNON.:** Epidemic, Spasmodic, or Malignant Cholera; Fr. *Choléra Asiatique*; Ger. *Asiatische Cholera*; Ital. *Colera Asiatico*.

**DEFINITION.**—Asiatic cholera is a specific disease, characterised by violent vomiting and purging, with rice-water evacuations, cramps, prostration, collapse, and tending to run a rapidly fatal course. It is capable of being communicated to persons otherwise in sound health, through the dejecta of patients suffering from the disease. These excreta are most commonly disseminated among a community, and taken into the system, by means of drinking-water, or in fact by anything swallowed which contains the specific organisms passed from cholera patients. In badly ventilated rooms, the atmosphere may become so fully charged with the exhalations from patients suffering from cholera as to poison persons employed in nursing the sick. In the same way, people engaged in carrying the bodies of those who have died from cholera for burial, or in washing their soiled linen, may contract the malady. In a dried condition the organisms contained in cholera excreta may retain their dangerous properties for a considerable period.

Asiatic cholera is endemic in certain parts of British India, where, from time to time, it assumes an epidemic character, and is apt then to spread, through the means above indicated, along the chief lines of human intercourse, and so to extend over the world.

**HISTORY.**—Since the days of Hippocrates, medical practitioners residing in various parts of Europe have described a disease which they called cholera. The nosology of this affection was hardly a matter of doubt with them, and it is only in modern times that the question has arisen, as to whether the cholera commonly met with among us is identical in its nature with Asiatic cholera. Doubtless, if we compare isolated cases we may find that the symptoms which these affections induce are very similar; but those who have lived beyond the endemic area of

Asiatic cholera, and watched the disease spread from India over Europe and America, can scarcely mistake this malignant malady for simple cholera. Asiatic cholera was unknown in Europe before the year 1829-30, although it has existed in India for many centuries. It is true we have no accounts of cholera extending throughout the whole of Hindustan prior to the year 1817, but this arises from the circumstance that it was only at the commencement of the present century that the British Government began to bind the heterogeneous principalities of India into union, and thus render it possible for us to gather together authentic details regarding the disease, as it spread from one province to another.

We cannot here fully consider the relations which unquestionably exist between the rapidity of the diffusion of cholera from the East over Europe, and the increased facilities of communication that have recently been established between India and Persia and Arabia, also from Hindustan to Russia and the shores of the Mediterranean. For instance, sixty years ago the passage from Bombay, up the Arabian and Persian Gulfs, could only be undertaken at certain seasons of the year when the winds were favourable, and even then the voyage was difficult to accomplish; now large steamers run every week from Bombay to Bassorah and the intermediate ports along the Persian Gulf, and others pass with equal rapidity to the various towns bordering the Red Sea. But although we cannot enter further into this subject, we must, in order to appreciate the nature of cholera, glance at the chronological order of some of the principal outbursts of the disease, which have been disseminated from British India over the world.

In 1817 cholera spread rapidly throughout Bengal, extending during the following year over the greater part of Hindustan, and from thence to Ceylon, Burmah, and China. The disease was communicated from Bombay *via* the Persian Gulf in 1820-21, and travelled northward, but did not extend into Europe.

During the year 1826 cholera again broke out over Bengal, and, passing through the Punjaub, it entered Cabul in 1828, and from thence extended to Persia, and so to Russia during the years 1829-30, and over the whole of Europe and the greater part of America.

In 1840-41 cholera accompanied a British force despatched from Calcutta to China; it broke out among our troops on their voyage to that country, and, having spread throughout the Chinese and Burmese empires, it passed in 1843-44 through Kashgar to Bokhara, and so to Cabul. From Afghanistan the disease extended south into Scinde, and westward in 1845-46 through Persia to Russia and Europe, reaching America in 1848.

In 1849 cholera was very fatal over Bengal; and during the season of 1851-52 it was com-

municated through the Punjaub and Bombay respectively to Persia and Arabia; and in 1853-54 it spread *via* Russia and Egypt with frightful virulence throughout Europe and America.

During the years 1860-61-62 cholera prevailed to an alarming extent throughout Bengal and the Central Provinces, and in 1864-65 in Bombay and along the shores of the Red Sea; thence it passed with pilgrims from Mecca to Egypt, and so to Europe, and for the fourth time to America. It is well to fix our attention on the lessons to be learnt from a careful study of the history of these early epidemics of Asiatic cholera; because, subsequently to 1865, the rapid and complicated means of transit between India and Europe has rendered it difficult, if not impossible, to trace the spread of the disease westward through the Red Sea route.

These outbursts of epidemic cholera were remarkably sudden in their advent, a considerable number of people in the affected locality being attacked by the disease within a few days after it appeared among them. The malady almost invariably died out from among the inhabitants of a country under its influence during the cold seasons of the year, to re-appear on the approach of summer. As a general rule, the disease was most deadly during the first year of the epidemic; it decreased in violence the second season, and then gradually disappeared, seldom prevailing in any one locality for more than three consecutive years.

**ÆTIOLOGY.**—The more we study the early history of Asiatic cholera, the better shall we understand, that every outburst of the disease which occurred beyond the confines of India might invariably be traced back through a series of cases to that country. The disease has never broken out spontaneously in any part of the world—no amount of filth, of bad food, or climatic influences has up to the present time induced a widespread epidemic of cholera: it has invariably spread from its endemic area over the world. The inhabitants of countries far removed from Hindustan, and having limited communication with that empire, such as Australia, have not experienced the disease; whereas states in proportion as they are brought into intimate relation with India become subject to outbreaks of cholera.

Many of the earliest Anglo-Indian authors declared their conviction that the disease was contagious; others disputed this idea; but all agreed that cholera, when extending over a country, often settled on the inhabitants of low-lying, ill-drained, and overcrowded localities, and that it frequently left unharmed people residing beyond the affected area, although they might have been employed in attending patients suffering from the disease. It remained for Dr. Snow, in 1854, to explain this apparent mystery, and

to demonstrate, as he did by means of the Broad Street case,<sup>1</sup> that the poison which causes cholera is contained in the excrements of those suffering from the disease, and 'that if by leakage, soakage from cess-pools or drains, or through reckless casting out of slops and wash-water, any taint, however small, of the infective material gets access to wells, and other sources of drinking-water, it imparts to enormous volumes of water the power of propagating the disease' (Simon). Cholera patients cannot, in fact, communicate the affection to others, unless by means of the discharges which they pass. Persons attending them run no risk of contracting the disease, provided they are protected from swallowing the organic matter passed by the sick; but in badly ventilated rooms, this material, having been disseminated through the atmosphere, may be taken into the system by attendants and so poison them.

Sir William Aitken observes that the evidence in favour of the communicability of cholera by means of water or food, contaminated with cholera dejecta, has since 1854 become almost overwhelming. A remarkable instance of the kind, reported by the late Mr. Netten Radcliffe, took place in East London during the year 1866; and previous to this time, the circumstances of a case came under the writer's notice, in which a small quantity of a fresh rice-water stool, passed by a patient suffering from cholera, was accidentally mixed with some four or five gallons of water, and the mixture exposed to the rays of the tropical sun for twelve hours. Early the following morning nineteen people each swallowed about an ounce of this contaminated water—they only partook of it once—but within thirty-six hours five of these nineteen persons were seized with cholera. In this instance the choleraic evacuation did not touch the soil: as it was passed, so was it swallowed; but (and this is most important to remember) it had been largely diluted with impure water, and the mixture had been exposed to the light and heat of a tropical sun for twelve hours.

Professor Pettenkofer holds, that if the excreta from patients suffering from cholera pass into the earth, they may there, under peculiar conditions of the soil, moisture, and heat, undergo changes, and, having risen as a miasma into the air, be absorbed, and act as a poison on persons predisposed to the disease. It appears certain that cholera germs cannot multiply unless exposed to a definite range of temperature; they also require moisture, containing certain chemical substances, for their development and growth. The evidence brought together by Professor

R. Koch and G. Gaffky in their admirable report on cholera in Egypt, and in India, in 1883, published in Berlin 1887, is sufficient to convince the writer that the germs of Asiatic cholera consist of a specific bacillus. This micro-organism is found in the contents of the intestinal canal, and in the matter passed by persons suffering from cholera. We know that if such excreta gain access through water or food to the intestinal canal of persons predisposed to cholera, they may become affected with the disease, and so we conclude that the symptoms characteristic of Asiatic cholera are in some way dependent on the action of the cholera bacillus. From the researches of Dr. Cartwright Wood, Professor Hueppe, and other authorities, we learn that infectious organisms are capable of splitting up the particles of living albumen at the temperature of the body, and that these dissociated elements may combine to form specific basic poisons: organisms, constituting a definite species, produce the poison of cholera, and exist at all times in the soil throughout the endemic area of the disease; but in this, its saprophytic form, the microbe has but feeble action on living albumen; it may, however, under certain conditions of the soil, temperature, and so on, become exceedingly vigorous, in fact, parasitic, and then it can break up living albuminoid elements into combinations possessing toxic properties. We may thus form some idea as to the relation that exists between endemic and epidemic Asiatic cholera, and account for the manner in which the disease develops in certain localities, and from thence spreads at times over the world along the lines of human intercourse. As Dr. C. Wood remarks, it is possible that, as in the case of Pasteur's vaccines, the cholera microbe, when in its saprophytic form, may protect human beings subject to its influence from the more deadly action of the same organism when it has burst forth into parasitic growth, and if such be the case, may we not hope to discover means whereby human beings can be preserved from the effects of Asiatic cholera? Drs. Pye-Smith and Lauder Brunton, from experiments made with choleraic discharges, are inclined to think that the outpouring of fluid from the glands of the intestinal canal in this disease, is due to paralysis of the mesenteric nerves in connexion with ganglia derived from the solar plexus. It may be that the poison produced by the cholera bacillus has some such effect; but it is a long step from the fact that microbes are capable of producing certain toxic compounds to the proof that Asiatic cholera depends on the influence of this poison on the mesenteric nerves.

*Predisposing Causes.*—As far back as 1869, in a work published by the writer on Asiatic cholera, the following observations occur (p. 420):—'With the exception of the specific cholera-infecting matter, I entirely ignore all

<sup>1</sup> By this discovery, the result of rigid scientific investigation, Dr. Snow rendered an immense service to mankind which has not been recognised as fully as it should be.—*Editor.*

other causes, or combination of causes, as capable of producing this disease. The circumstances under which people live may predispose them to the action of the organism; but neither air, water, nor any other agency can induce an attack of cholera, though many of them may serve as media by which the infecting matter is conveyed into the intestines.' P. 383 and p. 402:—'From researches I have instituted into this matter, there is reason to believe that the acids of a healthy stomach destroy the organic molecular matter in decomposing choleraic dejecta, so that when swallowed in a concentrated form they may be so acted on by the gastric juice as to have their infecting power destroyed; but when swallowed diluted with water, a portion of the fluid probably passes at once through the stomach, and reaching the alkaline contents of the small intestines, begins its work of destruction upon the epithelium.' 'Persons in bad health, whether suffering from actual disease, or in the less-defined ill-health which results from imperfect hygienic conditions, are those most apt to be attacked by cholera. This is the case with the poor of our large towns, which are generally situated on alluvial soil. May not this be due to the want of a healthy acid secretion from the walls of the stomach, which their more vigorous neighbours are blessed with?' Recent investigations have added little beyond this to our knowledge of the predisposing causes of Asiatic cholera.

**ANATOMICAL CHARACTERS.**—The external appearances of the bodies of those who have died of cholera include the mottled skin, shrunken and livid appearance of the limbs, and other features hereafter described as characteristic of the disease during the stage of collapse. The temperature of the body rises after death, and it remains warm for some time. Rigor mortis sets in speedily, and is sometimes accompanied with muscular contractions, which displace the limbs of the corpse.

After death in the stage of collapse from Asiatic cholera, the only alteration to be discovered in the tissues and the blood is to be accounted for by the rapid drain of water into the intestinal canal which has occurred during life. The mucous surface of the stomach and small intestines is injected and swollen, and its epithelium is shed during life, and drops off from the mucous membrane in large patches within an hour and a half after death. Dr. Koch states that the cholera bacillus in many cases is found between the epithelium and the basement membrane, and also within the tubular glands; 'it also settles in large numbers on the surface of the villi of the intestines, and often had penetrated into their tissue, and in some instances had passed as far as the muscular layers of the intestine.' The epithelial

cells lining the kidney-tubules, bladder, and other organs are found detached from the basement membrane, but they, like the blood, do not contain the cholera bacillus.

Abnormalities of a specific nature, especially with reference to the amount of blood contained in the right side of the heart and lungs, have been described by pathologists as being characteristic of Asiatic cholera; and in many instances after death from this disease, if the *post-mortem* examination is delayed for a few hours, the right side of the heart will be found full of blood, together with the pulmonary artery and its divisions, while the lungs are collapsed and bloodless. But there are numerous exceptions to this state of the heart and lungs, and the condition above described is not infrequently due to *post-mortem* changes; for if the bodies of those who have died of cholera be examined immediately after death, the left side of the heart will often be found as full of viscid blood as the right side. A peculiar shrunken condition of the lungs exists, depending on the dry and empty state of the bronchi, which allows the elasticity of the organs to drive the air out of them more completely than usual after the chest is opened.<sup>1</sup> On the other hand, when death has occurred during reaction, the smaller tubes are often found full of pus, and parts of the lungs may be œdematous, or even in a state of broncho-pneumonia (Fagge, *The Principles and Practice of Medicine*, p. 287).

**SYMPTOMS.**—Asiatic cholera is most deadly at the commencement of an epidemic, and then usually begins without premonitory symptoms. The patient feels well up to within a few hours of the attack, or, it may be, goes to bed and sleeps soundly through the night, and immediately on rising in the morning is seized with violent purging and vomiting. After the first outburst of the disease, as a rule, cholera commences with diarrhœa, the stools being copious and watery, followed by great prostration of strength, with a peculiar feeling of exhaustion at the pit of the stomach; the sick person suffers from nausea, but seldom from actual vomiting or pain at the outset of the attack. If judiciously treated, many patients recover from this, the *first stage* of cholera; but if neglected the tendency of the disease is to grow rapidly worse. The stools become very frequent, and resemble in appearance and consistency the water in which rice has been boiled. These liquid evacuations flow away from the sick person with a sense of relief rather than otherwise. But the patient now commences to vomit, first throwing up the contents of his stomach, and subsequently all the water he drinks, mixed with mucus and disintegrated epithelium.

<sup>1</sup> On this subject see the chapter on Epidemic Cholera in Dr. George Johnson's *Medical Lectures and Essays*.—EDITOR.

The fluid is rejected from his mouth with considerable force, and this adds to the increasing prostration which is one of the most urgent and marked features of the disease. The patient complains of intense thirst, and a burning heat at the pit of his stomach; he suffers also excruciating pain from cramps in the muscles of the extremities; he is terribly restless; and his urgent cry is for water to quench his thirst, and that some one might rub his limbs, and thus relieve the muscular spasm. Although the temperature of the sick person's body falls below the normal standard, he complains of feeling hot, and throws off the bed-clothes in order that he may keep himself cool. The pulse is rapid and very weak; the respirations are hurried; and the patient's voice becomes husky. His countenance is pinched, and the integument of his body feels inelastic and doughy, while the skin of his hands and feet becomes wrinkled and purplish in colour. The duration of this, the *second stage* of cholera, is very uncertain. It may last for two or three hours only, or may continue for twelve or fifteen hours; but so long as the pulse can be felt at the wrist, there are still good hopes of the sick person's recovery. The weaker the pulse becomes, the nearer the patient is to the *third, or collapse-stage* of cholera, from which probably not more than thirty-five per cent. recover. The result, however, depends much on the condition of the patient's heart. It is quite possible, although the cases must be rare, that a sudden outpouring of fluid into the intestinal canal has been sufficient to cause syncope and death in persons suffering from weak heart, before the liquid contents of the bowels have had time to be ejected through the mouth or anus.

In the third stage of the disease the vomiting and purging continue, although in a mitigated form; and the skin is covered with clammy perspiration, especially if the cramps are still severe. We now cease to be able to feel the pulse at the wrist, the lividity of the extremities and surface of the body increases, the patient cannot speak above a low whisper, his breathing is very rapid, his eyeballs are deeply sunk in their sockets, and his features are marvellously changed within a few hours. The urine is suppressed. The temperature of his body may fall as low as 94° F. The patient remains terribly restless, longing only for sleep, and that he may be supplied with water. His intellect is clear, but he seldom expresses any anxiety regarding worldly affairs, although fully conscious of the dangerous condition he is in; sleep, and a plentiful supply of drinking-water, are the sole desires of a person passing through the collapse-stage of cholera. This condition seldom lasts for more than twenty-four hours, and reaction either commences within that period, or the

patient dies in collapse, or he passes on into the *tepid stage*, which in ninety-nine cases out of a hundred ends speedily in death. In the tepid stage of the disease the sick person's body feels cold to the touch, but the temperature, as shown by the thermometer, begins to rise rapidly, sometimes marking 99° or 100° F. The purging and vomiting cease; and the patient lies in a semi-comatose state, his eyes half-open, the ocular conjunctiva being deeply congested, the cornea hazy, and the pupils fixed. The pulse can be felt at the wrist, but the respirations are very hurried, suppression of urine continues, the patient's body is bathed in a cold clammy perspiration, the skin becomes of a dusky red hue, and death too frequently closes the scene within a few hours.

On the other hand, the sick person having been in the collapse-stage of cholera some twenty-four hours (it may be a longer or shorter period), the temperature of his body may begin to rise, gradually creeping up to the normal standard; the respirations diminish in frequency; the pulse returns; the patient can sleep, and after some thirty-six hours may pass a little urine: in fact, the functions of animal life are slowly restored, and the sick person recovers his health. This desirable result, however, is not infrequently thwarted by various complications which arise during the stage of reaction. Of these complications the following are the most important: suppression of urine; gastritis and enteritis; pulmonary congestion; meningitis; sloughing of the cornea; abscesses over the body; the formation of coagula in the right side of the heart or pulmonary arteries; hæmorrhage from the bowels; and roseola choleraica.

**DIAGNOSIS.**—The question of the diagnosis of Asiatic cholera is discussed in the article on CHOLERAIC DIARRHŒA.

**PROGNOSIS.**—The means of forming a prognosis in cholera may best be gathered from the preceding account of the disease. Speaking generally, the prognosis depends largely upon the stage of the disease, and upon the period in the epidemic at which the case occurs—that is, according as the patient has been seized at the outbreak, at the height, or towards the end of an epidemic.

**TREATMENT.**—In the first stage of Asiatic cholera we should endeavour to stop the purging, and without doubt opium is the drug upon which we may with the greatest confidence rely for effecting this purpose. When practising in the endemic area of cholera, the writer was in the habit of carrying about pills containing one grain of opium and four of acetate of lead, so that, if called to see a patient suffering from the disease in its early stage, no time was lost in administering one of these pills suspended in water. The next thing done was to make a large mustard poultice, and apply it over the whole surface

of the patient's abdomen. The sick person was ordered to remain in bed, and to be allowed nothing in the shape of food or water; but he might suck as much ice as he felt inclined for.

If the patient was again purged after the first pill, a second was given, and a third (but not more) after each loose motion. It often happened that the first or second pill, together with the mustard poultice, ice, and rest, sufficed to check the progress of the disease, and the patient recovered. Supposing this treatment not to succeed, or that on first seeing the patient it be found that he has passed into the second stage of the disease, we should still prescribe the pill, as above directed, suspending it in water, because in the solid form it might be rejected entire, and under any circumstances it would take time to be dissolved by the fluid contained in the stomach; the mustard poultice also should be applied, and the sick person kept warm and in bed. Ice is invaluable in this stage of the disease, and unless a person has passed through an attack of cholera, it is impossible to realise the immense relief it affords. It should be given in small lumps, the sick person eating and swallowing as much as he chooses. He will frequently devour a pound or two of ice in the course of an hour, and he cannot take too much of it. In the treatment of cholera there can be no question as to the value of ice. The patient should be prohibited from drinking water or any other fluid beyond that which he gets from the ice. The practitioner must be firm on this point, turning a deaf ear to the entreaties of the sick man or his friends that he may be permitted to swallow even a small quantity of water; for if they once break through this rule, it will be impossible to limit the amount of liquid the patient may consume. If this treatment does not check the progress of the malady, we may prescribe three grains of acetate of lead and fifteen drops of diluted acetic acid in water every second hour, and fifteen drops of diluted sulphuric acid in water every alternate hour, so that the patient should take a draught, first of one mixture, then of the other, every hour. Five drops of spirit of camphor may be added to each dose of the medicine, but this drug requires care in its administration, and should seldom be continued beyond five or six doses of from five to ten drops each. Should the vomiting be very severe, in spite of the free administration of ice, a second mustard poultice should be applied over the abdomen; all medicine must then be omitted for an hour and a half, after which time a scruple of calomel may be sprinkled on the patient's tongue, and he should be made to wash it down with a little iced water. The cramps are best relieved by hand-friction, and if very severe, ease may from time to time be given by allowing the

patient to inhale some ether. Hot-water bottles should be applied to the soles of the patient's feet, and also to his legs and abdomen.

Should the disease have reached the collapse-stage there is but little we can do for the patient. Ice must still be given, and, if the purging is frequent, the sulphuric acid draught (but no opium) may be administered every hour; heat and friction may with advantage be applied to the surface of the body; and the patient may now be permitted to drink iced water in moderation, provided it does not increase the vomiting. Wine and stimulants, if given by the mouth, do harm in this stage of cholera; but, if the purging has abated, enemata of warm beef-tea and brandy may be administered by the rectum every third hour. When reaction comes on, we must guard against doing too much—it is very rare indeed to see a patient in this condition sink from exhaustion, but probably many lives are lost by endeavours erroneously made, under the idea of keeping up the patient's strength. Iced milk or arrowroot is all that should be allowed to be given by the mouth for some time after reaction has set in; but enemata such as those above-mentioned, administered per rectum every five or six hours, or nutrient suppositories, are often beneficial, especially if the stomach remains irritable. Under these circumstances we not infrequently find that a small quantity of solid food is digested, when soup and liquids are rejected. In each case, however, the dictates of common sense and experience must guide the medical practitioner in his treatment of the sick person through the convalescent stage of the disease.

With reference to the treatment of suppression of urine after cholera, we should get the patient to drink about half a pint of water every second hour, so as to add fluid to his blood. Dry-cupping over the loins should be employed; and ten drops of the tincture of cantharides in water administered every hour, until a drachm of the drug has been given. It need hardly be remarked that suppression of urine after cholera is a most dangerous complication, and there is very little that can be done to restore the suspended functions of the kidneys.

PREVENTIVE TREATMENT.—Among persons predisposed to its influence, the infecting organisms of Asiatic cholera will manifest their effects on the system within five days of having been swallowed, but the germs of the disease do not always engender symptoms of virulent cholera. Nevertheless, in milder cases of the disease the evacuations passed by the patient may contain the microbes of cholera, and may therefore, under favourable conditions for its development, produce a deadly type of the malady. Consequently, the following remarks are applicable to instances of so-called *cholérine*, as well as

to the severer forms of cholera. If the disease has appeared within a neighbourhood, a searching examination must be made into the condition and source of the local water-supply, not overlooking that of the milk, which is too often diluted with water before being sold. All surface and doubtful wells or reservoirs (especially those in the proximity of drains and cesspools) should be immediately closed; and it is desirable that the drinking-water, before being consumed, should be carefully boiled and filtered. All accumulations of house refuse and filth must be removed; and dirty places, both within and without uncleanly premises must be freely disinfected and cleansed. There is no necessity when the disease is prevalent for making any alteration in the usual diet; but in times of cholera we cannot too strongly insist on at once checking any tendency to diarrhœa, especially if it be of a watery nature. Many cases of incipient cholera have been prevented from running on into dangerous disease, by the early administration of pills containing four grains of acetate of lead and one of opium, one pill to be taken after each loose motion, to the extent of three pills.

If called to treat a case of Asiatic cholera, care should be taken that the rice-water stools, and the matters vomited, are disinfected by means of a solution of mercury perchloride or some other germicide, which should be poured over the bottom of the vessel into which the evacuations are received from the patient; and directly the dejecta are passed from the sick person, a solution of one part of carbolic acid to twenty of water should be sprinkled over them, and they must be immediately taken from the patient's room, and disposed of as follows:—If the sewage of the locality is conveyed away by means of a constant water-supply, the disinfected cholera evacuation should be thrown at once into the sewer. Drains used for a purpose of this kind must, however, be constantly flushed with a mixture containing about an ounce of ferrous sulphate to a pint of water. But if the drainage of the place passes into a cesspool, the disinfected cholera stools should be buried in a deep hole in the ground, removed from wells, and, if possible, from human habitations. It is a most dangerous practice, however carefully cholera stools have been disinfected, to allow them access to a cesspool. The room in which the patient has been treated must be freely disinfected, and his bedding subsequently burnt. If the sick person should die, the corpse is at once to be placed in a coffin containing a mixture of lime, charcoal, and carbolic acid: in fact, the body should immediately be buried in a mixture of this kind, and the coffin with its contents committed to the grave, or, better, consumed by fire within twenty-four hours of the patient's death.

By far the most important preventive measures to be adopted against cholera are to provide a pure supply of drinking-water, good drainage, ventilation, and cleanliness; for these means, if rightly enforced, must prevent the cholera germ from spreading among human beings.

The Vienna Cholera Conference has decided that quarantine is inapplicable to the circumstances of cholera; but this subject, together with the duties incumbent on sanitary and port authorities with reference to the preventive treatment of the disease, hardly falls within the scope of this article. See QUARANTINE. C. N. MACNAMARA.

**CHOLERAIC DIARRHŒA.**—SYNON.: Choleric; Sporadic Cholera; Fr. *Choléra sporadique*; Ger. *Sporadische Cholera*.

**DEFINITION.**—An acute catarrhal affection of the mucous membrane of the stomach and small intestines, attended with vomiting and diarrhœa. The stools consist of a serous fluid, containing a little albumen. The whole system is implicated to a greater or less extent, through the rapid loss of water from the body, and its imperfect absorption through the intestinal canal during the active stages of the disease.

**ÆTIOLOGY.**—As remarked in the preceding article, it is only of late years that the question has arisen as to whether Asiatic cholera and simple cholera are identical diseases; but it seems probable that any obscurity which may exist on the subject occurs from the impression that similar symptoms are necessarily produced by precisely the same causes. It appears reasonable, however, to believe that, if the infecting matter of Asiatic cholera, when introduced into the intestinal canal, induces changes such as we have described, decomposing animal or vegetable substances, under certain conditions, may excite analogous changes in the mucous membrane of the alimentary canal, the consequence being that in both diseases a drain of serous fluid takes place from the bowels, followed by symptoms of cholera. In the case of Asiatic cholera, however, we believe that the discharges have the power, under favouring conditions, of propagating the disease, whereas the evacuations in simple cholera are in this respect less pernicious. Doubtless climatic and meteorological influences materially affect the susceptibility of the human subject to disease, and consequently we find that simple cholera, like the malignant form of the malady, is apt to prevail as an epidemic in moist or wet seasons of the year, and especially among people whose bodies are predisposed to pass into a diseased condition, from their having habitually breathed impure air and consumed unwholesome food and water, or become debilitated from other causes.

In a hot and moist climate like that of

Lower Bengal, choleraic diarrhœa is an affection which we meet with at all seasons of the year, and it is especially prevalent among infants who are being reared on cow's milk, or on other kinds of food prone to undergo putrefaction. Among the fish-eating Hindoos we frequently see several members of the same family who have been seized with symptoms of choleraic diarrhœa, attributable to the patients having partaken of fish which was slightly tainted. In fact, there are few more certain sources of this form of diarrhœa than stale fish; and it is evident that, whatever the deleterious influence may be which food of this description contains, the mere fact of keeping it in boiling water for some time does not destroy its poisonous properties.

It occasionally happens that cases of choleraic diarrhœa occur among people residing in malarious districts, the diarrhœa taking the place of the cold stage of a fit of ague; patients in these circumstances may be seized with all the symptoms of severe cholera, but they almost invariably recover from the attack.

**SYMPTOMS.**—Choleraic diarrhœa begins suddenly; that is, the patient, whether an infant or an adult, has probably up to the commencement of the attack been in good health; there are, in fact, seldom any premonitory symptoms. A *child* may perhaps look somewhat paler than usual, and has a dark ring under his eyes, but beyond this appears to be perfectly well. Shortly after taking food, the infant vomits up a quantity of uncoagulated milk, the evacuation not being curdled like that from an overloaded stomach, the gastric secretion no longer having the power of coagulating the casein of milk. Soon after vomiting, or it may be before, the child commences to pass from the bowels an acid greenish-yellow fluid, containing flakes and often lumps of undigested food. The patient becomes very thirsty, is restless, and evidently in considerable pain, crying and drawing his legs up towards the abdomen. If these symptoms continue, the evacuations become colourless, resembling in appearance the rice-water stools of Asiatic cholera. The temperature of the body falls, the face becomes of a dusky hue, the features are pinched, and the eyeballs deeply sunk in their sockets. The fontanelles are depressed; the child is evidently terribly prostrated, his pulse can no longer be felt at the wrist, and his crying passes into a weak whimpering; he eagerly drinks water when offered him; and, as the exhaustion increases, convulsions supervene, and the child dies within a few days or even hours. On the other hand, the symptoms may abate at any stage of the disease, and the little patient gradually recover his health.

In the *adult* the symptoms of choleraic diarrhœa are much the same as those above

detailed. There are seldom any premonitory symptoms, and the attack begins with nausea and vomiting, together with a sensation of exhaustion referable to the pit of the stomach; the vomiting is speedily followed, or it may be preceded, by purging; copious watery discharges are thus passed out of the body, and the larger and more rapid the evacuations the more they come to resemble the serum of the blood, which, in fact, drains into the intestinal canal and passes away from the stomach and bowels. The patient naturally complains under the circumstances of intense thirst. He is very restless; and at the commencement of the attack suffers from colicky pains in the abdomen, and subsequently from spasms and cramps, which often seize the muscles of the extremities. The pulse becomes small and weak, the respirations are hurried, the voice feeble, and the countenance pale and shrunken. The urine is scanty or suppressed; and the temperature of the body falls one or two degrees below the normal standard. These symptoms, as a rule, gradually subside, the purging and vomiting cease, and the patient falls off to sleep, waking more or less exhausted in proportion to the severity of the attack, but he usually recovers his health rapidly.

**DIAGNOSIS.**—The question naturally arises, Are there any symptoms by which we can distinguish a case of choleraic diarrhœa from one of Asiatic cholera? In reply it may be affirmed that there is no characteristic symptom by which these affections can be diagnosed from one another. But, taking all the circumstances of any particular case into consideration, it is difficult, except on paper, to confound the two diseases; for unless a patient has recently imbibed the poison which produces Asiatic cholera, he cannot be suffering from that malady. Should the sick person reside in a neighbourhood affected by Asiatic cholera, we must, in forming an opinion as to the nature of the affection, be guided by the previous history of the case, the nature of the food consumed, and so on, and above all by the severity of the symptoms. Choleraic diarrhœa, even in the tropics, rarely passes on into collapse within a few hours from the commencement of the attack, such as is commonly seen in cases of Asiatic cholera; and, in the early stages of the former disease, there is seldom that complete loss of voice and pulse so characteristic of the malignant form of this affection. An experienced medical practitioner, placed at the bedside of a person suffering from Asiatic cholera, even in its earliest stages, feels no doubt whatever as to the nature of the affection, and is at once impressed with the grave responsibility of the charge which rests upon him: his anxiety is infinitely less when he meets with an instance of choleraic diarrhœa, although he is unable to lay down any hard and fast rules, by means of which he could define the

difference that exists between the symptoms present and those occurring in a case of Asiatic cholera.

**PROGNOSIS.**—Although choleraic diarrhœa in its more severe forms resembles mild cases of Asiatic cholera, it is a comparatively harmless disease. Unless among young infants, or old and sickly people with weak hearts, no matter how threatening the symptoms may be, however great the collapse and depression of the patient may seem, a previously healthy adult seldom dies of choleraic diarrhœa.

**TREATMENT.**—In cases of simple cholera occurring in the child, the important point we must enforce in our treatment is that the affected part shall have rest. In practice, however, it is often difficult to persuade parents and nurses that an infant can exist uninjured for ten or fourteen hours on iced water; nevertheless, we must insist on a plan of this kind being carried out. The little patient will eagerly swallow cold water, either from a bottle or spoon, and the child may be allowed to take as much cold water as he requires, and to suck ice, which may be wrapped up in the corner of a handkerchief and put into his mouth. A poultice made of equal parts of mustard and flour, applied over the abdomen, is often very useful in this form of disease. With reference to drugs, should the treatment above indicated not relieve the symptoms, or should the vomiting be very constant, from two to four grains of calomel may be given, and repeated if necessary in an hour's time; however, if the diarrhœa is the more prominent symptom, calomel is not required, but a teaspoonful of castor oil should be administered. After the bowels have been cleared out, if the serous discharge continues, we should order astringents, in the form of  $\frac{1}{2}$  of a grain of acetate of lead every hour, or  $\frac{1}{16}$  of a grain of nitrate of silver, until the purging subsides. Tannic acid, in combination with diluted sulphuric acid and sugar, is frequently a useful combination to administer to children in cases of this description. With reference to opium, much as we dislike prescribing it for infants, it may be necessary in cases of simple cholera; but it should hardly be given in a mixture to be administered from time to time by a nurse. Opium under these circumstances can only be admissible when given by the medical attendant himself, in doses of one, two, or three minims of laudanum in a little weak brandy and water, carefully watching its effects. If the drug causes the child to sleep for a few hours, it may act almost like a charm: the infant awakes comparatively well. But if the opium has no such effect, we may be tempted to repeat the dose, but can scarcely give it a third time, at any rate until some hours have elapsed since the administration of the second dose. The symptoms having subsided, the child's diet must be strictly attended to,

a good healthy wet nurse as a rule being an urgent necessity in the case of infants. Lime-water may with advantage be mixed with the child's food. The diet must be most carefully ordered. See DIARRHŒA.

With reference to the treatment of adults suffering from choleraic diarrhœa, we must bear in mind that, unless among old and debilitated persons, the patient will, as a rule, recover without medicine. If therefore called to prescribe for a case of this complaint, we may order fifteen minims of laudanum, or a drachm of the compound tincture of camphor in water, to be taken (supposing the patient is very sick) immediately after vomiting; half the above dose may be given at the expiration of one hour; and again after another hour, unless the symptoms have in the meantime subsided. A large mustard poultice should be applied over the abdomen, and the patient must be confined to bed, and kept on ice and iced water; he should not, however, be permitted to swallow too much liquid. If the vomiting is severe, a scruple of calomel may be given to an adult, or in the first instance an effervescing mixture with hydrocyanic acid may be employed to allay the sickness. On the other hand, should the diarrhœa be excessive, we may with advantage prescribe four grains of acetate of lead and ten drops of diluted acetic acid every second hour; or pills containing a drop of creasote, a quarter of a grain of nitrate of silver, a grain of camphor, and two grains of Dover's powder, to be repeated after each loose motion.

Among old or weakly persons, and also in the case of infants, it is often necessary to administer brandy and water from time to time, according to the state of the patient's pulse. C. N. MACNAMARA.

**CHOLERINE.**—A term applied to a class of cases which occur during the prevalence of cholera, in which the milder symptoms of the disease are present. It has also been used to designate the poison on which cholera is supposed to depend. See CHOLERA, ASIATIC; and CHOLERAIC DIARRHŒA.

**CHOLESTEATOMA** (χολή, bile; and στέατομα [Galen], a fatty tumour).—An encysted tumour, consisting chiefly of cholesterolin. See CYSTS.

**CHOLESTERIN** (χολή, bile; and στερόρος, solid. The term *cholestearin*, occasionally used, is derived from χολή, bile; and στέαρ, suet).—SYNON.: Fr. *Cholestérine*; Ger. *Cholesterin*; *Gallenfett*.

**CHEMICAL AND PHYSICAL PROPERTIES.**—Cholesterolin is a monatomic alcohol, represented by the empirical formula  $C_{26}H_{44}O$ , first discovered, in 1755, by Conradi in ox-bile, its exact composition being subsequently ascertained by Chevreul. It is probable that

there are several isomeric substances included within the term, though one only is known to occur in the human body. When pure it occurs as white, tasteless, inodorous, glittering scales, which form needles when crystallised out from solution in chloroform, and as rhombic plates, often deficient at one corner, when derived from alcoholic and ethereal solutions (*see* MICROSCOPE IN MEDICINE). It is insoluble in water, alkalis, and dilute acids, but readily dissolves in ether, chloroform, boiling alcohol, benzol, turpentine, and solutions of the bile-acids, and its solutions are lævo-rotatory. It melts at 145° F. The crystals heated with strong sulphuric acid give a carmine-red colour, or with strong nitric acid a yellow colour; with the former acid and tincture of iodine a violet which becomes blue-green.

**SOURCES.**—The exact physiological significance of cholesterol is not known, but it is generally regarded as a product of the metabolism of the nervous tissues, which should be eliminated by the liver in the bile. It is a normal constituent of certain of the tissues, forming nearly 50 per cent. of the solids of the white nervous matter, and 18½ per cent. of the solids of the grey matter, being probably combined therein with true fatty substances; it is estimated to form .25 per cent. of the red corpuscles of the blood; .4 per cent. of human bile; and it occurs in minute quantities in the spleen, urine, milk, and serum, and as a constant though quantitatively variable ingredient of the feces, where it has been erroneously supposed to be represented by sterocorin, which substance is in reality an impure form of cholesterol (*see* FÆCES, Examination of). It is also found in the yolk of egg. A cholesterol is of widespread occurrence among plants (such as peas and maize, also in almond and olive oils), and may be regarded as a decomposition-product of some of the albuminous constituents of the cells.

**PATHOLOGICAL RELATIONS.**—The pathological occurrence of cholesterol is varied and of unknown import. It is liable to be much increased in quantity in the normal places of its occurrence; thus in association with jaundice, though apparently not always, the blood has been known to contain 4 or 5 per cent.; in the bile it may be so abundant as to form crystals or be deposited as gall-stones, of which it forms the greater portion; in the feces, when they are free from bile, a considerable increase of cholesterol and fats may be met with. In exceptional cases of renal disease crystals have been deposited in the urine. The fluid of cysts, especially hydatid and ovarian, seems to be more liable to contain cholesterol than effusions into serous cavities; and this fact has been considered of some value in the diagnosis of ascites from ovarian dropsy. But it sometimes occurs in considerable quantities in

hydrocele fluids, and it has been met with in old pleural and peritoneal effusions, where it appears as glistening particles suspended in the fluid. In the caseous degeneration of pus and other inflammatory products crystals of this substance are sometimes formed, and in this way may be found in sputum and other excreta.

The train of nervous and other symptoms which often accompanies long-continued jaundice has been attributed to the retention of cholesterol in the blood, and hence the term *cholesteræmia* was employed to denote this condition. It is certain, however, that cholesterol as such is quite inert and harmless to the tissues, and it is undesirable to perpetuate the expression. *See* CHOLÆMIA.

W. H. ALLCHIN.

**CHORDEE** (χορδή, a harpstring).—

**DEFINITION.**—Painful imperfect erection of the penis during gonorrhœa.

**ÆTIOLOGY AND PATHOLOGY.**—Chordee is most common in the second and third weeks of gonorrhœa, and rarely attacks the patient after the third week. In exceptional cases, on the contrary, chordee, absent in the acute stage, is violently developed after the inflammation has become chronic and very slight. The bulbous part of the urethra is generally intensely inflamed when chordee happens; and, further, chordee is very uncommon when the urethritis is limited to the anterior or posterior portions of the canal.

The *mechanism* of chordee is imperfectly understood. Two explanations have been put forward:—(a) That the corpus spongiosum surrounding the urethra being affected by inflammation through the effusion of lymph into its substance, proper distension of its spongy tissue and elongation during erection cannot take place. Hence it is drawn tight like a bowstring by the arching of the distended corpora cavernosa. (b) That the inflammatory condition of the mucous membrane and submucous tissue at the bulbous part excites reflex spasm of the muscles surrounding that part of the corpus spongiosum. This prevents distension of the parts compressed; while the corpora cavernosa, being untrammelled, continue to expand in the ordinary manner. The first explanation is insufficient to account for some cases where the inflammatory action is very slight, and there is no evidence that lymph has been effused into the erectile tissue; for example, after a plastic operation on the penis. Again, natural erection has been known to take place very shortly after the subsidence of gonorrhœal inflammation, and, it is fair to suppose, before effused lymph can have been absorbed. The second explanation is unsatisfactory, because spasm of other muscles of the perinæum is often absent. In our present imperfect knowledge of the purposes served by the *nervi erigentes*, these

theories must be accepted with reserve. Probably both methods may be active in producing chordee.

The causes of chordee are *indirect* or *direct*. The most common indirect cause is urethritis or urethral congestion. Direct causes are the reflex irritants which usually produce erection during sleep, such as stimulating food and drink, strongly acid urine, great superficial warmth of the body, or a distended bladder.

**SYMPTOMS.**—The organ grows suddenly turgid and assumes a bowed or crooked form, causing acute pain, which is felt at the part and towards the perinæum. In severe cases the strain causes rupture of the mucous membrane and spongy tissue, with hæmorrhage. The loss of blood is usually limited to a few drops, and gives relief to the pain. Rarely the hæmorrhage is rapid and prolonged.

**TREATMENT.**—Abstinence from stimulants of all kinds, and late suppers; light clothing, and a hard mattress at night, are the best means of preventing chordee. Micturition at short intervals during the night must be enjoined. Of medicines the best is a suppository, at bed-time, of one grain of crude opium in ten grains of cacao-butter. The subcutaneous injection into the perinæum of one-sixth grain of acetate of morphine is also an effectual remedy. Both these applications should be followed by an aperient saline draught the next morning. A drachm of spirit of camphor thrown just before it is swallowed into an ounce of water, and taken on lying down at night, is also useful, and it may be repeated once if chordee awakes the patient; but it is a very uncertain remedy. More trustworthy are twenty or twenty-five grains of chloral hydrate in syrup and water at bed-time, and repeated in four or six hours if needed. Bathing the genitals and perinæum with very hot water for ten minutes before going to bed sometimes proves successful. The application of a spiral coil of narrow india-rubber tubing round the penis and scrotum, through which a continuous current of ice-cold water flows, is also an excellent preventive.

To subdue an attack of chordee the best measures are voiding urine; the application of cold to the perinæum, by sitting on a cold seat, or applying an evaporating lotion or ice; and the upright posture.

BERKELEY HILL.

**CHOREA** (*χορεία*, a dance).—**SYNON.**: Chorea Minor; St. Vitus's Dance; Fr. *Danse de St.-Guy*; *Chorée*; Ger. *Veitstanz*.

**DEFINITION.**—A disease of the nervous system, characterised by a succession of irregular, clonic, involuntary movements of limited range, occurring in almost all parts of the body.

The distinctive features of the movements

are the entire absence of either rhythm or method in their recurrence; that not individual muscles but co-ordinate groups are affected; and not one or more groups only, but almost all the muscles in turn. There is not actual loss of command over the muscles, but voluntary movements are interfered with by superaddition of involuntary movements. As a rule the movements cease during sleep.

**ÆTIOLOGY.**—Chorea is a disease of childhood: it is most common between the ages of eight and twelve, very rare before six, and rare after sixteen. It is more than twice as frequent in girls as in boys, especially after the age of nine. It occurs more frequently in families in which nervous diseases are hereditary than in others. It is more common in large towns than in the country; and far more frequent among the poor than among those in comfortable circumstances. Want of proper food, neglect, ill-usage, with the weakness and anæmia induced by these means, are very common antecedents, or chorea may follow measles or other febrile disease of childhood. Children well-nourished and with a good colour, exposed to none of these causes, may however suffer. An intimate association between chorea and rheumatism has long been recognised. A large proportion of the children suffering from chorea are found to have had acute or subacute rheumatism, and some of the most terrible cases met with, especially after the age of puberty, are those in which the chorea comes on during or just after acute rheumatism. Whether traceable to rheumatism or not, there is very frequently found in chorea a cardiac murmur, usually mitral systolic, sometimes aortic. This may or may not disappear after recovery. In almost all the fatal cases of chorea which have been examined after death, endocarditis with fibrous vegetations on the valves has been present. In adults, pregnancy divides with rheumatism the causation of this affection; recovery generally speedily follows delivery, and can rarely be brought about till this has taken place. Bad habits, and disorders of menstruation, are also undoubtedly capable of inducing chorea. Intestinal worms again have appeared to set up the disease, and instances are on record in which the expulsion of worms has been followed at once by cessation of the movements, but this must be extremely rare. Fright or some powerful emotion is very frequently assigned as the cause; and it is seldom that parents are not prepared with the instance required. But, making allowance for this, and notwithstanding the fact that endocarditis may be present in cases said to have originated in fright, it does not seem possible to exclude fright as a cause of chorea. The influence of imitation is less certain. The disease is said to be far less common in negroes.

**ANATOMICAL CHARACTERS AND PATHOLOGY.** The study of chorea, as of epilepsy and many other affections of the nervous system, has been hampered by its being regarded as a morbid unity. The view here maintained is that it is a symptom rather than a disease, and that the characteristic movements are in relation not with the *nature* of the morbid change, but with its *seat*. The seat of the disturbance is the corpus striatum, its character probably different in different cases; but the anatomical condition cannot amount to actual breach of structure, since that is known to give rise to hemiplegia, while it must obviously be of a kind to impair the functional vigour of the ganglia. Choreia has been called 'insanity of the muscles'; a better phrase (as the writer has said elsewhere) would be 'delirium of the sensori-motor ganglia.' In delirium there is loss of control over the mental processes, with rapid succession of incoherent and imperfect ideas; in choreia loss of control over the motor apparatus, with movements excessive in point of number and extent, but wanting in vigour and precision.

In some cases of choreia nothing abnormal has been detected after death, but usually the minute methods of investigation now pursued yield positive results. The largest series of examinations published is contained in a communication to the Royal Medical and Chirurgical Society, in the session of 1875-6, by Dr. Dickinson. He describes dilatations of the minute arteries as existing throughout the brain and cord, more especially, however, in the corpus striatum and optic thalamus, with small hæmorrhages; and considers the disease to be due to a widely spread hyperæmia of the nerve-centres. He did not find capillary embolisms, but does not appear to have drawn out the arterioles to look for them. The appearances he describes are very much those producible by impaction of microscopic particles of fibrin in the minute vessels. Capillary embolisms have been found by Dr. Tuckwell and other observers, predominantly in the central ganglia, but also in the central convolutions and spinal cord, accompanied by patches of softening and minute hæmorrhages. In almost all fatal cases of choreia there is endocarditis with deposits of beads of lymph on the mitral or aortic valves, or both, whether a murmur have been audible during life or not.

The *post-mortem* appearances consequently do not indicate any localisation of the morbid change in the central ganglia. But it is to be remembered that the fatal cases are those in which there is not only extreme violence in the choreic movements, but usually also delirium and other symptoms. There are in fact multiple symptoms just as there are multiple lesions, and we are called upon to distribute the symptoms, and assign them to their respective sources, by such knowledge of the

functions of the different nerve-centres as physiology affords us. The delirium or comparative dementia is thus attributed to the lesions in the convolutions; the loss of speech to lesions in convolutions or in lower centres, according to its character; the impairment or sensation to lesions in the thalami; the choreia to lesions in the corpora striata. The grounds upon which this last localisation—that in which we are immediately concerned—is decided, are as follows. We exclude the cerebral hemispheres and cerebellum, rather arbitrarily perhaps, since there is much to be said in favour of their contributing to excite the movement, now especially that convolitional motor areas have been demonstrated by Hitzig and Ferrier. The important point to be made clear, however, is that choreia has not its seat in the cord. The arguments and evidences against this are: (1) that *tonic* and not *clonic* spasm is characteristic of persistent spinal irritation; (2) the degree of control over the movements retained by the will; (3) their increase under emotion; (4) their cessation during sleep. To those, which were originally advanced by Dr. Russell Reynolds, may be added: (5) the diminished reflex action on tickling; and (6) the phenomena of hemichorea and its relations with hemiplegia. The evidence afforded by hemichorea is so conclusive that other considerations have been merely alluded to. It cannot be supposed, for example, that one lateral half of the entire length of pons, medulla, and cord can be affected without implication of the other half, which would be the case with hemichorea of spinal origin; and still more conclusive is the fact that when in hemichorea there is impairment of sensation it is on the same side with the movements, and not, as in hemiparaplegia (due to the division of one half of the cord), on the opposite side to the motor paralysis. The parallelism between hemichorea and hemiplegia is so perfect as to suggest at once that the two affections represent different conditions of the same nerve-centres, and is made more complete by the very discrepancies, as they may at first sight appear, which have been considered to be objections. In hemiplegia there are certain muscles which more or less completely escape paralysis: the *motores oculorum*, *orbiculares palpebrarum*, and other facial muscles, the muscles of the neck, chest, back, and abdomen. In hemichorea the irregular movements cross the median line and invade the opposite side in these same muscles. This has been explained (rightly or wrongly) by the hypothesis that all these muscles acting in compulsory concert with the corresponding (or other) muscles of the opposite side, the nerve-nuclei of the bilaterally associated muscles will be commissurally associated in the cord, so as to become in effect a single nucleus, and this single nucleus for muscles on each side of the body, being

connected with both corpora striata, is thrown into action by the sound corpus striatum when its fellow of the opposite side is damaged, as in hemiplegia, thus preventing paralysis; and, on the other hand, is reached by the irregular impulses from the corpus striatum affected in hemichorea, thus causing bilateral chorea in the parts enumerated.

In addition to the correspondence between hemichorea and hemiplegia just described, there are transitions from one to the other, and combinations of the two to be mentioned below, under the head of Complications. Hemiplegia may be succeeded by hemichorea (the *post-hemiplegic chorea* of various observers); or chorea may deepen into paralysis; or, as in a case reported by the writer, there may be with chorea of the limbs on one side, first chorea, then paralysis (hemiplegiform), and then again chorea of the same side of the face. The conclusion is obvious, that hemiplegia and hemichorea in these cases are indicative of different degrees of damage in the same centre. Hemichorea and hemianæsthesia have been found very constantly associated with structural lesions in the white fibres just outside the posterior extremity of the thalamus, usually involving also the ganglion itself at this part.

The well-known embolic theory of chorea originated by Kirkes, and improved and ably maintained by Dr. Hughlings Jackson, at once finds its place here. Capillary embolism is of all others the condition which might be expected to induce the instability without abolition of function which exists in chorea, and in almost all cases a fertile source of fibrinous shreds is present, in the form of vegetations on the valves of the heart; the fact of embolism, again, has repeatedly been demonstrated. While, however, giving to capillary embolism a prominent place among the causes of chorea, it cannot be considered as the only cause. The clinical differences between ordinary chorea and the acute and fatal form are of themselves suggestive of a different pathology; and the speedy recovery after delivery in the chorea of pregnancy, or (as in one or two cases on record) after expulsion of intestinal worms, is inconsistent with the existence of embolism. What the precise anatomical condition is can only be matter of conjecture, but it will be some form of inanition; irritability and debility, as the late Dr. C. B. Radcliffe has abundantly demonstrated, going together. Hyperæmia, with capillary blood-stasis, and capillary thrombosis by cohering leucocytes, have been suggested as causes. Prolonged arterial spasm from persistent reflex irritation, uterine or intestinal, or the more brief contraction of the cerebral vessels from fright, may perhaps lower the functional vigour of the ganglia to the degree required. Dr. Octavius Sturges has ably maintained the view that chorea is a purely functional affection, and that the

movements are adequately accounted for by instability of nerve-cells without any structural or vascular change recognisable even by the microscope, comparing it in this respect with epilepsy. A very important consideration is the remarkable limitation of chorea to the period of childhood—the period between infancy and puberty. This is a limitation, if not without parallel, certainly unequalled, and it points to a condition of nerve-centres in childhood which specially favours the occurrence of the disease. This condition may be said with confidence to be the fact that childhood is the period of special activity of the sensori-motor ganglia.

**SYMPTOMS.**—In a slight case of chorea the patient, usually a child, may be perfectly quiet when lying down, and for a short time even when sitting or standing, if not conscious of being under observation; but when walking, or while under examination, there will be various fidgety actions—abrupt flexion of the fingers, a sudden pronation of the forearm, or hitching up of one shoulder, or twist of the body, or there is shuffling of a foot on the floor, or, again, a jerk of the head or twitch of the mouth or eyes. If the patient be told to do anything, the movements will be multiplied and exaggerated in the muscles employed. A small object will be picked up and held, but the hand is brought down upon it hastily and after various irregular excursions. In a more severe case the grimaces, contortions, and jerkings succeed each other without intermission. The gait is now very peculiar, being slow, shuffling, and uneven; the steps of irregular length and unequal time; and the line of progress deviating. In the worst forms of this disease every muscle appears to be thrown in turn into violent contraction, the face is distorted this way and that, the eyes roll to and fro, the teeth are snapped or ground together, the whole body writhes, and the limbs are in unceasing motion. It is to be remarked that, even in extreme cases, the movements, violent as they may be, are in some degree circumscribed; the arms, for example, are not thrown up over the head, nor do the legs go to the full extent of their range of motion; the tongue is rarely bitten, though the lips may be. Deglutition is greatly interfered with in a severe attack, and the evacuations may be discharged involuntarily. In the mildest forms the diaphragm and muscles of the chest and abdomen are affected, causing irregularity in respiration. The action of the heart may also be irregular, but this is probably secondary to the respiratory variations in frequency and depth, and is not attributable to chorea of the heart. There is generally impairment of motor power, and frequently diminution of sensation. This is most readily ascertained in hemichorea, that is, chorea affecting one half of the body only, when the sound side can be

employed for comparison; but in the violent forms of the disease, when the skin is gradually worn through by incessant friction, there is often so little complaint of pain that sensibility must, it would seem, be blunted. Reflex sensibility is also commonly dull.

It has already been stated that the movements cease during sleep; this is a rule to which exceptions are rare though not unknown, even in mild cases, and especially in hemichorea.

Chorea is usually gradual in access, even in the cases which ultimately become severe; it is very commonly one-sided for a time, and occasionally throughout, when the name hemichorea is given. It is not, however, strictly unilateral in these cases, as the movements transgress the median line and affect the corresponding muscles of both sides of the body at those parts where these are bilaterally associated, and where in hemiplegia there is immunity from the paralysis, as, for instance, the oculo-motor muscles, and the muscles of the neck, chest, and abdomen.

**COMPLICATIONS.**—The foregoing description applies more or less to all cases of chorea, but there are often additional symptoms, and it will conduce to clearness if these are considered apart and called complications. Mention has been made of impairment of motor power; at times this amounts to complete paralysis, and the relations and combinations of chorea and paralysis, and especially of hemichorea and hemiplegia, throw much light on the disease. Paralytic chorea has been described as a special variety of the affection, but no line of demarcation exists between this and the common form. Chorea sometimes succeeds hemiplegia in the paralysed parts; more rarely chorea deepens into paralysis. Cases again occur in which with facial hemiplegia there is chorea of the limbs of the same side. Speech is very commonly more or less affected and occasionally completely lost for a time. The difficulty is usually articulatory, chorea of the muscles of respiration, phonation, and articulation interfering with utterance of words; but there is in some cases true aphasia, and when this is so, there is the same tendency to the association of aphasia with right hemichorea as with right hemiplegia. The intellect may suffer; the face has often an idiotic expression, usually from the muscular contortion or atony, but sometimes indicative of temporary imbecility. In the violent and fatal forms there is almost always delirium. Impairment of sensation is common, and hemianæsthesia is almost always associated with hemichorea. For Heart Complications, see *Ætiology*.

**DURATION, TERMINATIONS, AND PROGNOSIS.**—The average duration of chorea is about two months; if prolonged beyond three months it may be exceedingly chronic and go on better and worse for one or two years.

There is a tendency to spontaneous recovery; but on the other hand relapses are common. Chorea is rarely fatal in children; when it is so the case is usually acute and violent from a very early period of the attack, and it is rare for a case to run the usual course for a time, and then take on a very severe character. After puberty, and especially when it supervenes on acute rheumatism, it is very dangerous, but less so when associated with menstrual disorders and pregnancy than in youths or men.

**DIAGNOSIS.**—It is only necessary under this head to warn against the mistake of confounding with the movements of chorea the tremor or jactitation of disseminated sclerosis of the nerve-centres, which, though most common in adults, is not unknown in childhood.

**TREATMENT.**—In a large proportion of cases of chorea, especially such as come into the hospitals of London, rest and food, with perhaps aperients, are all that are required for recovery. But it can scarcely be denied that medicinal treatment often renders important services, especially in cases of a lingering character. The causation and pathological condition being various, it is to be expected that the remedies required will be different, and the attempt should be made to adapt the treatment to the special features of the case, the basis of all being the endeavour to improve the nutrition of the body generally, and of the nervous system, by good food, rest, and warmth. The food may be supplemented by cod-liver oil, and the late Dr. C. B. Radcliffe attached importance to the free administration of wine or other stimulants. Any recognised cause should be removed, such as constipation or worms; irregularities or suspension of the catamenia should receive attention; when there is pregnancy it may perhaps be necessary to induce premature labour. When the chorea is accompanied by rheumatoid pains and feverishness, iodide of potassium with ammonia may have a remarkably good effect. Iron in some form or other is very generally useful, but especially when the patient is anæmic. Another remedy is sulphate of zinc, given in doses gradually increasing from one or two grains three times a day, to six, eight, or ten grains, till sickness is induced, when in some cases the disease appears to be cut short. The remedy which in the writer's experience has been found most generally useful is arsenic. Trousseau sometimes gave strychnine in gradually increasing doses till its physiological effects manifested themselves. On the other hand, conium, recommended by Dr. John Harley, has been extensively employed; the only trustworthy preparation is the Juice, which should be given in gradually increasing doses, beginning with a drachm and going up to one or two ounces if necessary, till its depressing effect on the muscles

becomes evident. It has not, in the writer's hands, given satisfactory results. The same may be said of the application of ether-spray along the spine, which has been strongly recommended, except in acute cases in which the freezing of the skin here has in some cases been followed at once by sleep, and in a few days by alleviation of the violence of the chorea. Baths, warm and cold, especially shower-baths, spinal douches, spinal ice-bags, gymnastics, musical gymnastics, that is, movements timed by music, have advocates, and may no doubt be useful in suitable cases.

In the terrible cases of acute chorea the great indications are to procure rest for the poor sufferer and keep up the strength. Milk, eggs, beef-tea, and other forms of concentrated fluid nourishment, should be given freely, together with wine or brandy. Conium, hyoscyamus, bromide of potassium or ammonium, and chloral hydrate, have been tried separately or in combination, with more or less appearance of success; chloroform, again, may be administered; chloral hydrate by the mouth or rectum, and hypodermic injection of morphine, with free administration of brandy, have, in the writer's judgment, appeared to do much good. It is in these cases that tartar emetic in full doses has been recommended; it is certainly tolerated in an astonishing degree. Restraint of the violent movements is often a great comfort to the patient; the limbs should be carefully bandaged with flannel and bound, the legs together, the arms to the sides, a folded blanket, across the abdomen and hips, keeping down the body. If half-done it only adds to the suffering, but when properly carried out it gives a feeling of relief and favours sleep.

W. H. BROADBENT.

**CHOROIDITIS.**—Inflammation of the choroid. See EYE AND ITS APPENDAGES, Diseases of.

**CHROMIDROSIS** (χρῶμα, colour; and ἰδρῶς, sweat).—Coloured perspiration. See PERSPIRATION, Disorders of.

**CHRONIC** (χρόνος, time).—This word is applied to a disease when its progress is slow, and its duration prolonged. See DISEASE, Duration of.

**CHYLOUS URINE.**—See CHYLURIA.

**CHYLURIA**<sup>1</sup> (χυλός, chyle; and οὔρον, urine).—SYNON.: Galacturia; Chylous Urine;

<sup>1</sup> It has been deemed advisable, in view of recent advances in our knowledge of chyluria and allied subjects, to slightly alter and rearrange the late Dr. Lewis's articles. These advances are, in a great measure, the outcome of Dr. Lewis's own discoveries, and it is, therefore, much to be regretted that his premature death has deprived the Editor of his assistance. Those portions of the article on CHYLURIA which are placed within brackets are new; the unbracketed parts are as they stood originally, and nearly as they were written by Dr. Lewis.

Fr. *Urine laiteuse*; Ger. *Chylurie*; *Milchsaftiger Harnabgang*.

The affection known as chylous or chyloid urine has long remained a puzzle to physicians, not only on account of the very remarkable character assumed by the secretion, but also on account of the very erratic course which the disease runs. Scarcely any two persons affected with this malady give a similar account of its mode of onset, of the duration of the attack, or of the symptoms and seasons of its occurrence. The writer has had the opportunity of studying from thirty to forty cases of the disease in Calcutta; and the variety of symptoms presented, and the numerous causes, of the most opposite character, to which the disease has been attributed, are very perplexing. The histories of the cases published by various observers present a like uncertainty, and Sir William Roberts very aptly describes the course which the disease runs as marked by an irregularity and capriciousness which baffle explanation. It would seem as though the one symptom which may be looked upon as constant is the condition of the urine implied by the designation which was applied to the disorder by Prout.

**DEFINITION.**—[A diseased condition, originating in the great majority of cases in tropical or sub-tropical climates, which manifests itself usually by a more or less milky appearance of the urine, modified at times by an admixture of a variable proportion of a substance resembling blood.]

On standing, the fluid coagulates, so as to present the appearance of size. A microscopic nematoid entozoon (*Filaria sanguinis hominis nocturna*) is generally found in the blood and urine of persons affected with the disease.

**[GEOGRAPHICAL DISTRIBUTION.**—This is peculiar. The vast majority of cases are tropical or sub-tropical in their origin. Nevertheless, from time to time, cases crop up in the persons of individuals who have never been out of the temperate zones, nor in any way exposed to tropical influences.<sup>1</sup> Moreover, the disease is not equally distributed over the zone in which, in a general way, it may be said to be endemic. In most tropical countries it is of more or less frequent occurrence, but there are districts in these same countries which enjoy an immunity similar to that of temperate climates. The explanation of this apparent caprice in the distribution of chyluria is afforded by the peculiarity of the distribution of the *filaria sanguinis hominis nocturna*—its principal ætiological factor.]

<sup>1</sup> Of the four or five cases which have been recorded as having originally occurred in Europe, one is furnished by Sir William Roberts, the patient never having been out of Lancashire; and another by Dr. Beale, in a person who had never resided out of Norfolk.

**SYMPTOMS.**—So far as is at present known, there are no premonitory symptoms of chyluria. Sometimes the only symptom is the milky condition of the urine—a condition which usually comes on very suddenly; generally, however, the patient complains of an uneasiness, scarcely amounting to pain, across the loins, along the ureters, over the bladder, or along the course of the urethra—especially towards the perinæum in the male. There is generally marked debility, with mental depression. Occasionally chylo-serous discharges take place from various parts of the body—the axilla, the surface of the abdomen, the groin, and especially from the scrotum, in that condition of it which is known as elephantiasis lymphangiectodes (Bristowe), nœvoid elephantiasis, or varix lymphaticus. The disease is also sometimes observed associated with true elephantiasis of the limbs and scrotum. It occurs at all ages, from childhood to extreme old age, and in about equal proportion among the sexes—perhaps more frequently in the female than in the male.

With regard to the *urine*, it presents, as already mentioned, a milky appearance, and frequently emits a strong milky or whey-like odour, which is made more evident by warmth. After standing a short time the fluid coagulates, so as to form a more or less semi-solid mass resembling blanc-mange. In the course of a few hours the clot contracts, and the urine becomes rapidly decomposed. In some cases the fluid presents a pink colour, from the admixture of [coloured corpuscles like those of] blood. Commonly—at least, in India—the blood-like admixture, when present, is seen forming a shreddy adherent coagulum at the bottom of the vessel after it has stood for some hours. Not infrequently the flow of urine is suddenly stopped during micturition by the blocking up temporarily of the urethra with one of the clots. The specific gravity varies greatly—may range in the same individual from 1007 to over 1020. Shaken up with ether, the urine loses its milky aspect; and when nitric acid or heat is applied, a precipitate almost invariably results. These characters, and the fact of the coagulability of the fluid, indicate the presence of fat, albumen, and fibrin, all of which are to be considered as abnormal constituents. The proportion, however, in which they exist in different individuals, and even in the same individual at different times, varies greatly. Dr. Beale's analyses show, that though a specimen of urine may contain at one time 1.39 per cent. of fat, another specimen, obtained a few hours later, from the same person, may contain none. In the majority of cases the fatty element is usually scanty in the morning before meals, and so are the other abnormal elements, unless exercise have been taken, or the circulation otherwise accelerated.

Under the latter circumstance, as the late Dr. Bence-Jones has shown, the albumen is increased, without, however, a corresponding increase of the fat.<sup>1</sup> It is evident, therefore, that in order to institute a comparison between the character of the urine and the character of the various nutritive fluids, for the purpose of ascertaining from which of them the abnormal constituents of the urine are derived, the results of analyses of the latter fluids at different times of the day and at different stages of the disease should be taken. Further, as the nutritive fluids themselves undergo constant changes dependent on the quality of the nourishment supplied and the time which has elapsed since partaking of it, it is equally evident that any single analysis would be insufficient. An attempt has been made to bring together in the following table all of what appeared to be the most trustworthy analyses of these fluids which have been published. In order to simplify the table, only the estimates of the albuminoid and fatty matters have been given, these being the most pronounced of the more readily estimated abnormal constituents in the urine:—

Constituents selected.	URINE in chyluria. [Mean of 15 analyses.]	BLOOD in chyluria. [Hoppe-Seyler.]	BLOOD—Normal human. [Bequerel and Rodier.]	CHYLE [Mean of 6 analyses: man, cow, horse, ass, dog, cat.]	LYMPH—human. [Mean of 4 analyses.]
Albuminoids	0.54%	3.35%	7.00%	7.08%	2.96%
Fatty matters	0.80%	0.67%	0.06%	0.92%	0.56%

A glance at this table illustrates the fact that the relative proportion of the albuminoids to the fatty matter in chylous urine does not correspond with the proportion in which they are found in any one of the nutritive fluids of the body. In normal blood, for example, the fatty matter is as 1 to 116 of the albuminoid, whereas in the urine the former exceeds the latter to a very considerable extent. The same discrepancy, but to a less degree, is found to exist when the urine and chyle are compared—the quantity of albumen in the latter being more than seven times greater than that of fat. The proportion of these substances in lymph approaches more closely to what is encountered in the urine—the fat being to the albumen as 1 to 4 very nearly. The specimen of chyluria-blood analysed by Hoppe-Seyler<sup>2</sup> approaches very closely to the average composition of human lymph—the fatty matters being as 1 to 5 of the albuminoid, thus differing in this respect very considerably

<sup>1</sup> *Phil. Trans. of Royal Soc.*, cxl., 1850, p. 651.

<sup>2</sup> *Med.-chem. Untersuchungen*, 1871, s. 551–56. Abstract by Dr. Ferrier in *Journ. Chem. Soc.*, vol. ix., 1871; p. 740.

from Dr. Bence-Jones' analyses of similar blood, where the quantity of fat given is that of normal blood. On the other hand, the urine of the man from whom this blood was obtained yielded a proportion of fat almost identical with what Hoppe-Seyler obtained in the urine of the person whose blood he examined—the figures given by the former writer being 0·74 and by the latter 0·72 per cent. The composition of the blood in this affection must be regarded as hitherto unsettled. Guibort found in a clot of it almost twice, and Hoppe-Seyler about eleven times, as much fat as is found in normal blood; on the other hand, Rayer, Bence-Jones, and Crevaux could detect no change in its composition. It is possible that the discrepancy in the results of these analyses and macroscopic examinations of the blood may be due in part to the particular moment when the blood was abstracted. According to M. Claude Bernard, 'Les urines chyleuses ressemblent au sang d'un animal en digestion, ou plutot à celui des oies que l'engraisse.'<sup>1</sup>

*Microscopical Characters of the Urine.*—[The filaria sanguinis hominis may generally be detected in the urine.] In making a search for it, it is advisable to pick out one of the coagulated shreds found in the urine, transfer it to the glass slide by means of a forceps or pipette, and carefully tease the fragment before applying the cover-glass.

[A still better plan is to break up the coagulum in the urine with a glass rod as soon as it is formed, and then to search the sediment which, after an hour or two, collects at the bottom of the vessel in the same way as is customary in examining for 'casts.' As large a slide as is practicable ought to be examined, and a low power employed in the first instance, as it often happens that the filariæ are present, but only in very small numbers, and might be readily overlooked if a small quantity of sediment is examined or a high power employed.]

[The periodicity characteristic of the filaria when in the blood is not observed by the parasite in the urine. It may be found in chylous urine passed during the day as well as in that passed during the night. In a certain, though small proportion, of cases the parasite cannot be found; but there is every reason to suppose that at one time, even in these instances, the filaria had been present. Cases are on record in which, after its presence had been fully ascertained, the parasite disappeared from the urine while the patient was under observation, the chyluria persisting. In the rare instances of chyluria originating outside the endemic area the filaria will, of course, not be found at any time. It is difficult to define the limits of this area, however, so that in every case of chyluria the parasite ought to be sought for.]

<sup>1</sup> Quoted by Crevaux, *op. cit.*

The other leading microscopical character of the urine is the minutely molecular matter—fat in an emulsified condition—to which the fluid owes its opaline or milky aspect. There are also numerous white, lymphoid corpuscles, together with red corpuscles, numerous or the reverse according to the degree of sanguinolence of the urine. Casts of the tubular structure of the kidney—indicative of organic disease of these organs—are seldom to be seen; they were absent in all the cases that have come under the writer's observation.

With regard to the *microscopical examination of the blood*, the writer has not observed that the corpuscles or serum presented any abnormality indicative of the presence of fatty matter in any form—the serum has seemed as clear and as free from molecular matter as normal blood. As far as his experience goes, the only feature worthy of special note in connexion with microscopical examination of the blood in chyluria is the presence of the hæmatozoon already referred to. [In consequence of the periodicity observed by the filaria in the blood, it is absolutely necessary that this examination be made during the late evening, the night, or the early morning; some time between 9 p.m. and 6 a.m. is the best. Searched for at this time, and as recommended elsewhere (*see* FILARIA SANGUINIS HOMINIS), the filaria is usually found in the blood; if it is absent from the urine it will not be found in the blood. If it is found in the blood in chyluric cases it will also be found in the urine; but it sometimes happens that, though the filaria is present in the urine, yet the most careful and prolonged examination may fail to detect it in the blood.]

*ANATOMICAL CHARACTERS.*—The *post-mortem* examinations of persons who have died whilst affected with chyluria also testify to the freedom of the kidneys from disease. This was the case in two autopsies conducted by Dr. McConnell in Calcutta. The writer had the opportunity of examining the kidneys of the first case, and all the organs of the body of the other case, but could find nothing in any of the organs or tissues suggestive of being a cause of the urinary derangement, except the fact that all the vessels—arteries as well as veins—contained the filaria in their minutest ramifications.

[But, although no evidence of disease of the parenchyma of the kidneys has been discovered in connexion with this disease, two more recent and carefully conducted *post-mortem* examinations have shown that the lymphatic system connected with the urinary tract may be seriously deranged. This is only what might have been expected from the frequency with which chyluria is associated with diseased conditions of the lymphatics in different parts of the body—notably of the

scrotum and the inguinal glands. Dr. Stephen Mackenzie<sup>1</sup> and Ponfick<sup>2</sup> have each put on record details of such examinations, and in both instances thickening and dilatation of the thoracic duct, and marked varicosity of the pelvic and other lymphatics were found. It can be readily understood—as pointed out by Sir William Roberts<sup>3</sup>—how a varicosity, which may have been distinct enough during life, would collapse after death, and be difficult then to demonstrate and readily overlooked; and it is possible that in this circumstance lies the explanation of the negative results of most of the *post-mortem* examinations of cases of chyluria.]

**ÆTIOLGY AND PATHOLOGY.**—Having considered in detail what seem to be the leading pathological features of chyluria, a brief reference may be made to the views which prevail regarding its ætiology. These may be comprised under three heads:—(1) Dr. Vandyke Carter<sup>4</sup> advocates the view that a direct communication exists between the chyle-carrying vessels and the urinary tracts. (2) MM. Claude Bernard and Ch. Robin believe that the condition of the urine is but a symptom of piarrhæmia—fatty blood; the latter condition being but the normal condition of the blood for some time after the partaking of food, aggravated and made permanent by derangement of the digestive organs—notably the liver. One of these distinguished authors (M. Robin) suggests, further, that this derangement is probably induced in the liver and elsewhere by the *filaria sanguinis hominis*.<sup>5</sup> (3) The third view to be noticed is that advanced by Sir William Roberts. This view appears to be based mainly on the history of a very remarkable case, published by him in 1868. It was one in which a coagulable chylolymphous discharge escaped from open vesicles which had formed over the surface of the abdomen; the patient's urine was, moreover, chylous for two days. Sir William Roberts suggests that a condition somewhat similar to that on the surface of the abdomen existed in the urinary tract—a sort of eczema—probably on the front of the bladder. *Post-mortem* examination did not, however, confirm this view, nor could anything be detected in any of the organs suggestive of a cause, but this authority infers that this was probably due 'to the fact that in the last few weeks of life the morbid process had retrograded and had consequently left no appreciable marks on the surface of the bladder.' The examination of the skin in the diseased part showed that the *cutis vera* and the subcutaneous tissue were traversed by short channels or lacunæ from

the width of a crow-quill to that of a hair. A careful study of this and other cases suggested to Sir William Roberts the view that one, at least, of the forms of chyluria may be due to hypertrophy of the lymphatic channels, and subsequent acquisition by them of gland properties. M. Robin's view does not suggest any special anatomical difficulty, but it remains to be demonstrated more conclusively than has hitherto been done that the blood in man ever contains a sufficient quantity of fatty matter to produce such extreme milkiness by admixture with the urine as is observed in chyluria. Sir William Roberts' theory is certainly not open to these particular objections, as, given a certain agent to start the formation of these glandular tissues, there does not appear to be any special anatomical or physiological difficulty to be got over; but the results of all the autopsies as yet recorded do not warrant the inference of the existence of such pathological conditions. Future observation, however, may show that they really do exist.

[Multiplied observations, made during recent years, have made it more than probable that Dr. Carter's view of the pathology of chyluria is the correct one—namely, that somewhere in the urinary tract there is a varicose condition of the lymphatics, similar to that found in elephantiasis lymphangiectodes, varix lymphaticus, or lymph-scrotum—a disease, as pointed out, also commonly associated with the filaria, and very frequently with chyluria. Either the mature parasite itself, or its young, or its prematurely expelled ova, or stenotic conditions of the lymphatic trunks induced by some of these, act as a mechanical obstruction to the onward flow of the lymph, and thus induce a varicose condition of the lymphatics on the distal side of the seat of the obstruction. Rupture of the varicose vessels will then admit the lymph to the urine if the varicosity is anywhere in the urinary tract.]

[For diagnostic purposes, as well as to arrive at a correct idea of the pathology of chyluria, it is necessary to bear in mind that whilst chylous urine, or its coagula, on the one hand, may be clear as the purest gelatine or, on the other hand, white like milk—in most cases from time to time, in others always—they may have a blood-like tinge, often of great intensity. This latter circumstance is likely to lead the unwary to a diagnosis of hæmaturia, and give rise to the idea that blood-vessels as well as lymph-vessels are in some way involved; all the more so as genuine hæmaturia is endemic and very prevalent in many of the countries—Egypt, for example—in which chyluria is also endemic (see ENTOZOA; *Bilharzia*). Some authors, misled by a false idea of the pathology of the disease, describe these cases of red chyluria under the name of hæmaturia, or hæmato-chyluria, assuming, and apparently

<sup>1</sup> *Trans. Path. Soc.*, 1882, vol. xxxiii.

<sup>2</sup> *Berlin. klin. Wochen.* 4 Oct., 1880.

<sup>3</sup> *Urinary and Renal Diseases.*

<sup>4</sup> *Trans. Med. and Phys. Soc. Bombay*, vol. vii.

1861. *Medico-Chir. Trans.*, vol. xlv., 1862.

<sup>5</sup> *Leçons sur les Humeurs*, 2nd edit. 1874, p. 845.

being under the impression, that genuine blood from the blood-vessels is the cause of the red colour of the chylous clots in the urine. It is quite possible for the same individual to be the subject of chyluria and also of hæmaturia, and such a coincidence has been noted; but in an ordinary case of red-tinted chylous urine there is no reason to suppose, further than that suggested by the colour only of the urine, that the term chyluria, applied to these cases, is unscientific, or that the characteristic pathological feature is derived from any set of vessels other than the lymphatics. In the first place, it is very unlikely that both sets of vessels—blood-vessels and lymphatics—could be similarly affected in the same individual and in the same organ; and, in the second place, it is well known that the contents of the thoracic duct, and of some of the larger lymphatics, and also lymph long stagnant in obstructed lymphatics, acquire a red tinge from the normal advance of the development in them of red blood-corpuscles.<sup>1</sup> Hence, undoubtedly, the red colour so frequently seen in the coagula referred to. In the same way may be explained the absence, or gradations, of milkiness occasionally encountered. If the abnormal element in the urine be derived from vessels into which no chyle has regurgitated, if the vessels are filled with a chyle in which the fatty element is defective, either from peculiarities of diet or from its relation as regards time to the period of digestion, it will be less milky and more transparent and jelly-like. The differences in the analyses of chylous urines admit of a similar explanation.]

Of the ætiological significance of the presence of the filaria in the circulation there can, the writer thinks, scarcely be much doubt—more especially when the number of observations recorded within the short period that has elapsed since attention has been drawn to its existence therein, is taken into consideration. These suggest more than a fortuitous connexion; indeed, it might rather be said that chylous effusions may be considered as symptomatic of the parasitism. Filaria have even been detected shortly before chyluria had manifested itself.

[As regards those cases of chyluria apparently unassociated with the filaria, we must bear in mind that anything which could give rise to constriction or blocking of the lymph-channels might act in the same way as the filaria does as regards producing lymphatic varicosity and rupture. Thus pressure by a tumour, constriction from inflammatory thickening in or about the walls of the thoracic duct or pelvic lymphatics, or stricture following ulceration of their walls, might, it is conceivable, bring about this state of

varicosity leading to rupture and possibly to chyluria.]

**PROGNOSIS.**—Persons have been known to suffer off and on from this affection for from one to fifty years. This would suggest that the malady usually runs a chronic course, which, as a rule, it doubtless does; on the other hand, patients apparently in fair health otherwise have been known to die very unexpectedly from no recognised acute disorder. With regard to the prospect of a cure a very guarded opinion should be given, as the probability is that the complaint will return again and again so long as life lasts—even when the disease commences at a very early age, and often after a complete change of climate and avocation.

**TREATMENT.**—This has proved extremely unsatisfactory in almost all the cases recorded; in fact it cannot be distinctly stated that the course of the disease has been materially modified, much less cured, by any known remedy. Iodide of potassium has been tried in large doses, and in some cases appears to have been beneficial; in others the tincture of the perchloride of iron has seemed to be more successful. A decoction of the bark of *Rhizophora racemosa* (mangrove) has a reputation in Guiana, just as a decoction of the seed of *Nigella sativa* (used also as a condiment in curries) has in India. The latter remedy has, however, been known to be powerless in mitigating the malady even in cases where on former occasions it had been resorted to with apparent success. Perhaps the most satisfactory results which have been published are those which have followed the administration of large doses of gallic acid—one or two drachms a day.

[Large doses of benzoic acid given in glycerine have, in some hands, been followed by a cessation for a time of the chyluria. But from what we now know about the pathology of this disease, it is evident that rest in the recumbent position, with elevation of the pelvis, is by far the most important indication to be attended to in the treatment.] See *FILARIA SANGUINIS HOMINIS*.

TIMOTHY LEWIS. PATRICK MANSON.

**CICATRISATION** (*cicatrix*, a scar). Cicatrization is the process by which solutions of continuity in an organ or tissue are repaired. These solutions of continuity may be due to injury, ulceration, extravasation, or the effusion of inflammatory products. The result of the process is the formation of a cicatrix or scar.

**PATHOLOGY.**—Cicatrization as it occurs in superficial parts in surgical practice may be selected as affording a typical illustration of the process. It is most frequently and easily observed, and it corresponds to what is met with in deeper tissues.

Repair may take place either with or without the occurrence of granulation, and the

<sup>1</sup> Gibbon., *Journ. of Anat. and Physiol.*, vol. xx. 1886

process of granulation may or may not be accompanied by suppuration; the existence both of granulation and of suppuration depending on the degrees of abnormal stimulation to which the injured tissues are subjected.

*Healing by first intention.*—In an incised wound favourably circumstanced as to vascularity of tissue, absence of tension, and apposition of edges, epidermic continuity may be re-established in thirty-six to seventy-two hours. The lips of such a wound are temporarily united by a thin layer of lymph and white corpuscles of the blood, and perhaps soon afterwards by cells proliferated from connective-tissue corpuscles. These cells become spindle-shaped, and are ultimately converted into ordinary connective-tissue corpuscles, whilst offsets from the neighbouring capillaries re-establish the circulation throughout the new tissue. Coincidentally with these changes the surface becomes covered with epithelium. The resulting scar at first appears as a red line, which subsequently becomes white from the disappearance of many of its blood-vessels.

*Healing by scabbing.*—When an open wound heals by scabbing, the epidermis spreads over the tissues without the intervention of the process of granulation, owing to the protection afforded them by the crust of blood and lymph which has formed upon the wound.

Recent antiseptic surgery has shown that even large hollow wounds filled with blood-clot, such as are caused, say, by operation for ununited fracture of the femur, may cicatrise completely without suppuration or granulation, if protected from the stimulation of the antiseptic used. In these cases the white corpuscles of the clot become organised directly into fibro-plastic cells and connective-tissue corpuscles, and the new epidermis will probably be formed beneath a thin upper layer of the clot.

*Healing by granulation.*—If a recent wound, too large for scabbing, be simply left exposed to the air, or treated with water dressing, or with an ordinary antiseptic, there will be a discharge at first of serum tinged with blood, then of pale serum, and afterwards of cloudy yellowish serum, replaced in about three days by fully-formed pus. By this time the wound will be studded over with little bright red elevations, termed granulations, which ultimately cover the whole surface. Granulation-tissue consists of nucleated cells, amongst which pass vascular loops with thin walls. After an interval the edges of the skin are seen to be on a level with the granulating surface, and as it were continuous with it. Perhaps already the wound is much smaller, owing to the shrinking of the granulations as their cells assume the spindle-cell type. Then, extending from the cutaneous margin there may be observed a narrow red line, brighter than the rest of

the granulating surface, owing to the presence of a layer of transparent epidermis. Next day this zone will be bluish, from the growing opacity of the epidermic cells, and there will be a fresh red line corresponding with the newest epithelium. A day later the outer layer will have become so opaque as to be whitish, and the second to be bluish; and there will again be a fresh inner red line, which will gradually advance until the scar is complete. The new epidermis quickly separates into a horny and a mucous layer. See also SKIN GRAFTING.

The healing of an evacuated abscess-cavity depends partly upon the contraction, and partly upon the coalescence of the granulations of which its pyogenic membrane consists. This contraction is an essential part of the cicatrising process, and often causes deformities and secondary effects by pressure exerted on vessels and nerves.

Many cases of disease of joints (caries, &c.), and the behaviour of abscesses treated antiseptically and with adequate drainage, may be referred to as instances of the existence of non-suppurating granulations.

JOHN BISHOP. R. J. GODLEE.

**CINCHONISM.**—A condition induced by the administration of quinine, the chief active principle of cinchona. See QUINISM.

**CINCLISIS** (κυκλίζω, I move often). This word signifies agitation or motion, and was formerly applied to involuntary winking or movements of the eyelids; and also to the movements of the chest in dyspnoea.

**CIRCULATION, Disorders of.**—Abnormal excess and deficiency of blood are known as *hyperæmia* and *anæmia* respectively. Each of these may be *general* or *local*. We also recognise as a disorder of circulation stagnation or *local cyanosis*.

**I. Hyperæmia.**—*General hyperæmia* signifies excess of blood in the body, and is also called *plethora*. *Local hyperæmia* means excess of blood in a part. Such excess may be caused either by superabundant supply, or by deficient removal of blood through the agency of the blood-vessels. Dilatation of the arteries, however produced, causes more copious afflux of blood, which fills the capillaries and veins in a corresponding degree, so that there is excess of blood in all the vessels of the part. This condition is called *arterial* or *active hyperæmia*, *active congestion*, or *determination of blood*. If, on the other hand, blood is imperfectly removed by the veins, these vessels, as well as the capillaries, become gorged, and the condition called *venous* or *passive hyperæmia*, or *venous congestion*, results. It is doubtful whether there can be *capillary hyperæmia*, except as the result of one of these conditions.

A. *Arterial or Active Hyperæmia*.—An excessive amount of blood can be conveyed by the arteries only under two conditions:—(1) Enlargement of these vessels by relaxation of their muscular walls; (2) Increased pressure within them, from obstruction of collateral channels with which they communicate, *i.e.* collateral hyperæmia.

(1) Relaxation of the muscular walls may be caused *directly* by violence or by warmth, as is illustrated in the redness of the skin produced by a blow, by heat, or by the reaction after intense cold. Sudden withdrawal of pressure has the same effect, as is sometimes seen on evacuating a hydrocele or fluid-collection in a serous cavity. Dry-cupping produces similar but more complex results, the veins being acted upon as much as the arteries. Relaxation of the muscle-fibres is produced also *indirectly* through the vaso-motor nerves. If these are paralysed, relaxation of the fibres occurs, and the arteries dilate. Experimental section of the cervical sympathetic in animals shows this most clearly; but the same result follows less constantly if other nerves containing vaso-motor fibres are divided or injured, such as the mixed nerves of the limbs, or branches of the trigeminus. Wounds of the brachial plexus have been found to cause hyperæmia of the fingers (*glossy fingers* of Paget). When the section is complete, hyperæmia is only transitory, and is soon followed by a return to the normal condition, or even by undue anæmia, which is permanent. In irritative lesions, on the other hand, such as gunshot wounds, hyperæmia continues as long as the irritation. In such cases it is possible that the lesion is not paralytic, but depends upon stimulation of the actively dilating vaso-motor fibres which physiologists have now shown to exist in many parts of the body, since stimulation of these produces the same result as paralysis of the inhibitory fibres. Hyperæmia often accompanies neuralgia, both depending upon some morbid condition of the nerve. The starting point of the neurosis in all these cases may be, and often is, in the central nervous system, and hence chronic diseases of the spinal cord or brain are often accompanied by general paralytic hyperæmia—that is, flushing, or by congestion of special parts. The same result may come from reflex nervous action, set up by disturbances of the digestive organs, the organs of generation, or of other parts.

(2) Collateral hyperæmia is a consequence of the rise of pressure produced by the blocking-up of arterial channels in the adjoining parts. It is usually effected by the enlargement of existing vessels, and the conversion of small, almost capillary, vessels into pulsating arteries. It occurs not only in the familiar instances of surgical ligature, and the sudden blocking of an artery by a plug (*see* EMBOLISM), but in the gradual obstruc-

tion which accompanies atrophic and sclerotic processes. When the chief afferent blood-channels to an organ become obstructed, its peripheral parts are very liable to become hyperæmic, a principle which when applied to such cases as cirrhosis of the liver, granular kidney, and sclerosis of the brain, will be found fruitful in practical deductions.

SIGNS AND RESULTS.—The colour of parts in a state of active hyperæmia is, during life, bright red, the arteries, large and small, being visibly injected, while the capillaries, filled with arterial blood, produce a diffuse red colour. In experimental hyperæmia the blood may remain bright red even in the veins. The temperature of external parts becomes elevated, though not above that of internal parts. Sometimes there is obvious pulsation or throbbing. There may be swelling, which is due to simple enlargement of the vessels, not to exudation of fluid, since this does not occur from arterial hyperæmia alone. The nerves, both those of common sensation and those of special sense, are more excitable than they are normally. There is usually a subjective sensation of warmth, and there may be pain or itching.

Arterial hyperæmia may last for a long time without producing any change whatever in the part affected, but may, under conditions little understood, give rise to hypertrophy, which sometimes, though rarely, results from section of the cervical sympathetic. Transitory but repeated hyperæmic conditions more regularly produce this result, as is seen in hypertrophy from pressure; in thickening of the skull from excessive exposure of the head to the sun; in hypertrophy of the skin of the face and its glands from frequently recurring hyperæmia (*acne rosacea*). This kind of hyperæmia constantly precedes inflammation, but can hardly be said to produce it. It does, however, render the tissues more vulnerable, bringing them into a condition in which a slight cause will set up inflammation. Unless the vessels are unsound, simple arterial hyperæmia does not lead to hæmorrhage.

B. *Passive Hyperæmia or Venous Congestion*.—This may be due to—(1) Feeble circulation; or (2) Obstruction in the veins.

(1) Blood may be imperfectly removed from a part, owing to the imperfect action of the forces which normally maintain the flow of blood in the veins. These are, besides the action of the heart, the pressure of muscles (combined with the arrangement of the valves in the veins), and the movement of the thorax in inspiration. If these are deficient, the venous current will be everywhere delayed, but notably in those parts where it has to overcome the action of gravity. In the erect posture this will be the case in the lower limbs; and hence venous congestion is common in the legs, ankles, and feet. In decumbent patients, for analogous reasons, the

nates, sacrum, shoulder-blades, and the bases of the lungs behind become the seat of what is called *hypostatic congestion*. Very general obstruction, such as results from imperfection of the heart itself, may lead to the condition called *cyanosis*, which is essentially venous congestion, and to similar congestion of the lungs, liver, kidneys, and other internal organs, with very serious results.

(2) Obstruction of the veins is rarely produced by a morbid condition of the walls of these vessels, but may result from coagulation of blood within them. Another cause is external pressure, such as that of tumours, of the gravid uterus, or of the intestinal contents, as in the case of the hæmorrhoidal veins. Finally, indurative changes in the solid viscera lead to venous obstruction, as is seen in cirrhosis of the liver, which produces congestion of the whole portal system.

**SIGNS AND RESULTS.**—The colour of parts in a state of passive hyperæmia is bluish rather than red, the veins, large and small, being injected with venous blood, and the capillaries, in which the blood is also venous, producing a uniform purple colour. If the congestion is extreme, collateral venous channels are likely to be established, which are sometimes the only evident sign of internal venous obstruction. The surface is usually cooler rather than hotter when compared with corresponding parts of the body; and there is no unusual nervous sensibility or sense of throbbing. Swelling very frequently occurs, and depends on actual serous effusion from the vessels, so that the parts are often anasarcaous, pitting on pressure; while in cavities there is an accumulation of fluid.

Venous congestion produces more important and permanent results than arterial. In experimental venous obstruction, besides engorgement of the vessels, two nearly constant phenomena are seen—copious transudation of serum, and passage of a number of red blood-discs through the walls of the capillaries and smaller veins. Few or no white corpuscles emigrate, and the arterial circulation is unaltered. The absence or occurrence of dropsy depends upon the adequacy or inadequacy of the lymphatics to carry off the superfluous serum. In ordinary pathological venous congestion all these changes are seen to some extent; extravasation of red blood-discs being shown by the pigmentation of parts in chronic congestion, though this is not evident in the acute condition. Chronic venous congestion increases the hardness and density of organs, a change which may, in the first instance, result from simple œdema, but in the end is due to fibroid change (*see* DEGENERATION). Such organs are at first enlarged, but ultimately diminish in size, and suffer fatty atrophy, not only through the general law of fibroid change, but because venous blood is

inadequate to the proper nutrition of tissues. These changes are seen in the liver and kidneys in cases of obstructive heart-disease. External parts, as the skin of the lower part of the leg, show by a tendency to ulceration that they are imperfectly nourished, and are also liable to become inflamed from slight causes (varicose eczema).

**POST-MORTEM CHARACTERS.**—The appearance of hyperæmic parts after death is not necessarily the same as during life. The colour of the blood does not enable us to say whether the hyperæmia was arterial or venous. All blood contained in the body after death, excluded from the air, is dark or venous, but becomes florid when exposed to the air, unless it have previously undergone some *post-mortem* change, or some morbid alteration during life. This change may be watched in the lungs when the chest is opened, especially in the case of children whose lungs have little local colour. The only important point after death is the fulness of the three kinds of vessels. Arteries are usually empty, unless diseased; the larger veins almost always full. If the smaller veins and arteries are conspicuously and brightly injected, the part may be described simply as congested; a uniform colour indicates fulness of the capillaries, which may be confirmed by the microscope. Uncomplicated arterial hyperæmia leaves no trace after death; the appearance of it is produced by inflammation. Simple venous hyperæmia can only be recognised as such after death—by comparison, that is, with the same part under normal conditions. Chronic venous congestion is indicated by many of the same characters as during life. Care should be taken not to mistake for arterial hyperæmia mere staining with blood-pigment of the walls of the vessels; nor for venous congestion mere *post-mortem* hypostasis, or the settling down of the blood, if fluid, after death.

**II. Anæmia.**—*General* anæmia is a morbid condition in which there is a deficiency of blood, or, more correctly, a deficiency of the red corpuscles of the blood, throughout the whole body. It is also called *oligæmia*, or *oligocythæmia* (*see* ANÆMIA). *Local* anæmia, with which we are here concerned, signifies deficiency of blood in a part. It may be complete or partial. Complete local anæmia can only occur when the blood-supply of a part is totally cut off by obstruction of its arteries. The conditions and consequences of such obstruction are discussed elsewhere (*see* EMBOLISM). Partial anæmia or *ischæmia* may be produced by direct pressure, or else by arterial obstruction, permanent or transitory. Permanent anæmia of many parts results from gradual obstruction of arteries by atheromatous change; or, still more strikingly, by a form of endarteritis (*endarteritis obliterans*) generally due to syphilis; or, again, from deposition of fibrin on the diseased

vascular wall. Temporary anæmia results from spasmodic contraction of the annular fibre-cells in the muscular coat of the artery. Such a contraction may be produced experimentally by direct electrical stimulation, or by stimulation of the sympathetic branches distributed to the vessel; and in pathological conditions we find such contraction occurring in consequence of some derangement of the nerve-centres, or from reflex irritation, or even, as it would seem, idiopathically. Neuralgia and migraine are often accompanied or caused by spasm of the arteries, and epilepsy has, with less certainty, been attributed to the same cause. In these cases it is possible, as is held by some authorities, that anæmia of the nerve-tissue is the cause of the disturbed innervation. Hysterical blindness, and probably other hysterical affections, may be explained in the same way. Partial anæmia may, as shown elsewhere, lead to local cyanosis, with the appearance of venous hyperæmia. Total anæmia necessarily causes necrosis of the part; and even partial anæmia produces characteristic changes.

**SIGNS AND RESULTS.**—An anæmic part is pale, its temperature in the case of external parts is diminished, and there is weakened or arrested arterial pulsation. A permanent condition of partial anæmia produces degeneration, ending in atrophy of the affected part. The wasting of the skin, and possibly that of the kidneys in old age, is due to this cause. Transitory anæmia causes necessarily a cessation of functional activity in the part, as is obvious in the nerve-centres and the muscles; but does not, so far as we know, produce any permanent change. Compression or obstruction of the abdominal aorta produces symptoms of temporary paraplegia, from anæmia of the lower part of the spinal cord.

**III. Local Cyanosis.**—It must not be supposed that a venous condition of the blood in a part, with consequent purple colour, low temperature, and deficient vitality, is necessarily due to venous engorgement. It may be due, as is obvious in external parts, to mere stagnation in the capillaries. When blood is stagnant, or nearly so, it becomes venous, having given up its oxygen to the tissues; and the part shows the characters of venous congestion. This is seen in the familiar instance of the effects of external cold on the extremities. A similar condition, which may be called *local cyanosis*, sometimes occurs independently of, or only assisted by, the action of cold, in certain parts, such as the extremities of the fingers and toes, the tips of the ears and the nose. It is possible that similar conditions of internal parts may occur. A part affected with local cyanosis may undergo partial necrosis or inflammation, as in the case of frost-bite or chilblain.

Some persons, especially when young, have a constitutional proclivity to this condition, especially in cold weather, and have been described as having a 'chilblain circulation'; but it is not necessarily associated with chilblains, properly so-called. In other cases the cyanosis is preceded by, or alternates with, a condition of anæmia in the part, and is evidently due to a functional disturbance of circulation, more or less transient. In its extreme form, when occurring suddenly, and affecting deeper parts as well as the skin, and leading to necrosis, this constitutes Raynaud's disease. The condition is nearly always symmetrical, affecting corresponding parts on both sides of the body.

**ÆTIOLOGY.**—The constitutional or habitual form of local cyanosis is generally associated with malnutrition and sluggish circulation; but it does not appear that mere weakness of the heart is sufficient to produce it, or is more than a contributory factor. The fault of circulation appears to lie in the arteries, and to consist either in habitual spasm, or possibly in want of tone. Imperfect nutrition of the walls of the capillaries may also, by retarding the flow, assist in producing stagnation. In the more transient forms there would seem to be spasm of the smaller arteries, causing anæmia, on which capillary stagnation supervenes. The slighter forms occur chiefly in women, accompanying hysteria or menstrual disturbances, or at the climacteric period. Such persons present an alternation of 'dead fingers' with cyanotic circulation. The severe form known as Raynaud's disease is attributable to more complete and sudden spasm of arteries, deep as well as superficial. In both these forms there must be some antecedent derangement of the vaso-motor nerves, dependent probably upon deeper-lying disturbances of the nervous system, probably central, which cannot here be discussed.

**CONSEQUENCES.**—The slighter form may cause no material change, or may lead to the same series of changes as seen in chilblains, namely, necrosis of epidermis, with formation of imperfect bullæ, and inflammation, ending possibly in ulceration. It is difficult to draw the line between these changes and those produced in healthy persons by frost-bite. In Raynaud's disease complete necrosis, causing symmetrical gangrene of deeper parts, may result.

**TREATMENT.**—Young persons, with a tendency to local cyanosis, should be well fed, warmly clothed, and encouraged to take vigorous exercise for short periods. Cod-liver oil is the most useful drug; but iron or arsenic may also have their place. When the condition is paroxysmal, rather than constant, large doses of sulphate of quinine (5 gr. twice or thrice daily) are sometimes extremely useful. In the spasmodic anæmia, followed by cyanosis, of hysterical women, or

at the climacteric period, arsenic has appeared to the writer to be by far the most useful medicine. See RAYNAUD'S DISEASE.

J. F. PAYNE.

**CIRRHOSIS** (*κίρρῶς*, yellow).—SYNON.: Sclerosis; Fibroid Substitution; Fibroid Degeneration; Chronic Interstitial Inflammation; Fr. *Cirrhose*; Ger. *Cirrhose*.

**DEFINITION.**—The term 'cirrhosis,' which was originally invented to describe a particular state of the liver, has now acquired a more extended meaning, and is applied to similar morbid processes affecting other organs, though the name itself, derived as it is from the yellow colour of the liver in this disease, ceases to be properly applicable. Cirrhosis may be regarded as a chronic non-suppurative inflammation affecting the interstitial, connective, and supporting tissues of the different organs, and not those by which the proper physiological function is performed.

**ANATOMICAL CHARACTERS.**—The process begins, after a more or less protracted hyperæmia, by the appearance in the interstitial tissues, between the proper functional elements, of small lymphoid corpuscles or leucocytes, which are arranged in lines or tracts interpenetrating the affected organ. These corpuscles crowd the tunica adventitia of the small vessels, the lymph-spaces, and the cavities in which the connective-tissue corpuscles lie; and when present in considerable amount appear to the naked eye as narrow lines of a slightly translucent greyish material. This condition was formerly termed cellular hyperplasia of the connective tissue, and the corpuscles themselves were supposed to originate from proliferation of the connective-tissue corpuscles. More recent researches have, however, rendered it probable that in inflammation the connective-tissue corpuscles remain passive and take no part in the formation of new cells. The early stage of cirrhosis is perhaps therefore more correctly described as cellular infiltration of the connective tissue, the cells themselves being white blood-globules which have emigrated from the vessels, and their descendants. The amount of this cellular exudation varies very much in different organs in the different forms of the disease. In many cases of cirrhosis of the liver, and in the fibroid induration of the left ventricle of the heart, and of the pylorus, it is very considerable, and causes much increase in the bulk of the affected organ. In other instances, as in some forms of granular kidney, it may be extremely small, and the indurated fibrous tissue consists in great part of the withered remains of the vessels, ducts, &c. of the organ.

The later stages of the process consist in the conversion of these tracts, which may be looked upon as closely analogous to ordinary granulation-tissue, into fibrous tissue. A

more or less fibrillated intercellular substance appears; the corpuscles diminish in number; the remaining ones become in part elongated and oat-shaped, and some may pass into true spindle-cells, or become stellate: in cirrhosis of the lung tracts of true spindle-cell tissue are often met with. In the liver, where much of this new tissue is often formed, numerous blood-vessels become developed in it; they are devoid of distinct walls, and consist merely of channels lined by endothelium. The fibrous tissue which is the final result of the whole process closely resembles ordinary cicatricial tissue; it is usually tough, dense, and imperfectly fibrillated, with a strong tendency to contract. Sometimes, especially in the suprarenal capsules, and less frequently in the liver, portions of it consist of a reticulated connective tissue.

The effect of this series of changes on the proper physiological tissue of the organ is to cause its atrophy. This is partly due to the direct pressure of the new-growth, when it is formed in large quantities, but chiefly to the constriction of the contracting fibrous tissue and the consequent obliteration of the blood-vessels; for even where, as in the liver, new vessels are developed, the original vessels of the organ become obliterated. The atrophy of the proper tissue of the organs appears to take place by a gradual process of granular and fatty degeneration, followed by absorption.

**PATHOLOGY.**—If we now proceed to inquire into the nature and causes of these forms of chronic interstitial inflammation, the question arises whether this irritative hyperplasia of the connective tissue is primary, or whether it is the result of some preceding change in the parenchyma of the affected organ.

In the analogous condition of sclerosis of the central nervous system it can hardly be doubted but that the latter is the case; so also in cirrhosis of the lung, the interstitial changes in most cases are preceded by inflammation of the lining membrane of the bronchial tubes and air-cells. In other organs, as the liver and kidney, the interstitial change is usually considered to be primary, but this cannot be looked upon as certain.

The cause of cirrhosis seems always to be some form of protracted irritation of a low degree of intensity producing a chronic hyperæmia, functional or mechanical. As examples of this may be mentioned cirrhosis of the liver, which results from the congestion produced by spirit-drinking; sclerosis of the grey matter of the cerebral convolutions after protracted maniacal excitement; cirrhosis of the lung, the result of prolonged inhalation of irritating dust in the various forms of grinders' and miners' phthisis; the cirrhotic thickening of the pylorus in chronic catarrh of the stomach; and cirrhotic affections of the lungs and heart extending to these organs in chronic inflammation of their serous coverings. A mere passive congestion, however,

if long-continued, may cause a simple hypertrophy or overgrowth of the interstitial connective tissue, and more or less induration in consequence. Still, it does not appear to have the same tendency to excite active proliferation and the formation of the contracting fibrous tissue characteristic of true cirrhosis. The exception to this rule is that we usually meet with a slight degree of the cirrhotic change in cases of 'nutmeg' atrophy of the liver, due to prolonged passive congestion of the hepatic venous system.

Chronic tuberculosis of the lungs often gives rise to extensive interstitial fibrous induration, the so-called fibroid phthisis. Visceral syphilis also may produce changes which can hardly be distinguished from other forms of cirrhosis. Even the typical gummata originate in and are surrounded by tracts of cirrhotic induration; and in other cases, where true gummata are not formed, the only difference between syphilis and true cirrhosis is that in the former there is a greater accumulation of the new-growth at particular points, and a less general diffusion of it through the affected organ. Lastly, in some cases, as in the cirrhotic liver of hereditary syphilis, the two conditions cannot be distinguished. W. CAYLEY.

**CIRRHOISIS OF LIVER, LUNG, &c.**—See LIVER, LUNG, &c., Diseases of.

**CIVIL INCAPACITY.**—A chief cause of civil incapacity is mental weakness or disease, and it is one of the duties of the physician to aid in determining the existence and nature of such conditions. There is a kind of incapacity which is implied in the restriction of a person's liberty when he is placed under care in an asylum or other special place for treatment. The necessary information regarding this will be found in the article, LUNACY, Laws of. But the question of incapacity is more directly raised when it is proposed that a person should be declared unfit to exercise his civil rights, to require the shield of the law to prevent his being imposed on, and to obtain special protection for his property. Medical evidence must be taken if it becomes necessary for a Commission of Lunacy to be issued by order of the Lord Chancellor. This is a proceeding which ought not to be adopted if it can properly be avoided. But until a person has been found lunatic by inquisition he is, though placed in an asylum under regular certificates, not debarred from exercising his rights in the disposal of his property. The acts of any person either in or out of an asylum may, however, be declared invalid if it can be shown that at the time they were performed the person laboured under such form of insanity as rendered him incapable of performing them rationally and without harmful consequences. On this principle any person may be found to have been incapable

of contracting marriage, of executing a deed, contracting a debt, making a will, or giving credible evidence. The principle, it must be carefully noted, is not that the mere existence of insanity in the person performing them invalidates such actions, but that if the insanity has materially affected the character and quality of the actions they may be thereby invalidated. This is one of the most important principles that a medical jurist has to keep in mind, as it is not an infrequent mistake to suppose that a person is necessarily incapacitated from the performance of every civil act the moment he can be proved to labour under any condition to which the term insanity may be applied. Perhaps the case in which the validity of a civil act is most easily endangered by the existence of any form of insanity is the contract of marriage. This proceeding is supposed to so affect the whole relations of life, that almost any form of unsoundness of mind may be sufficient to interfere with that intelligent and deliberate consideration which is essential to the giving of rational consent.

The different kinds of mental disease will be found described elsewhere (see INSANITY), and it is necessary that the practitioner, when dealing with medico-legal questions, should be fully acquainted with them. But it is chiefly important that he should distinguish the two following classes: (1) diseased perversion of the mental faculties; and (2) weakness or enfeeblement of these faculties, resulting either from defective development, disease, or decay. The first class includes all kinds of insanity which are the result of active disease. These would include the simple forms of delirium, mania, melancholia, and monomania; as well as the similar primary conditions which are found in general paralysis, and other diseases which present maniacal, melancholic, or monomaniacal symptoms. It is in this class that the special knowledge of the physician can be most successfully applied in aiding the administration of justice. In order to establish the incapacity of a person said to labour under any of these forms of disease, it must be necessary that an experienced physician should not only be able to detect their characteristic symptoms, but also to show that the performance of the duties or the exercise of the rights under consideration would be modified or obstructed by the existence of such disease. The second class includes congenital imbecility, and all the forms of what is called chronic dementia—all those enfeeblements of mind which are sometimes the remaining effects of acute disease, sometimes the concomitants of chronic disease, and sometimes only the mental phase of senile decay. Here, again, the information which may be communicated by the physician must be of great importance. But in estimating the extent to which a condition

of mere mental weakness will disable a person from the performance of a certain class of actions, there so much special medical knowledge is not required as is necessary in the consideration of more active disease.

*Marriage.*—As has been already stated, the mere existence of any form of insanity in one of the parties may render a contract of marriage void. In one case which terminated in this manner, a man who had been insane, and when in that state had voluntarily contracted marriage, instituted the suit himself.

*Civil Contracts.*—These may be held binding although made by lunatics. If the person with whom a contract is made had no knowledge that the person contracting was insane, and if no attempt was made to take undue advantage of him, the contract would be held good.

*Wills.*—A person is considered to be of a disposing mind—that is, capable of making a valid will—if he knows the nature of the act which he is performing, and is fully aware of its consequences. It is in regard to the making of wills that the law has carried out most thoroughly the principle, that the validity of an act ought to be maintained in cases of insanity, unless at the time the act is performed the state of mind of the agent can be shown to render him unfit to perform that particular act in a rational manner. Persons have made valid wills while inmates of lunatic asylums. And one will was held to be good though the testator had committed suicide within three days after its execution. The existence of delusion which has been regarded by lawyers as of such importance in cases of alleged insanity does not invalidate a will; for it has been declared to be ‘compatible with the retention of the general powers of the faculties of the mind,’ and to be ‘insufficient to overthrow the will, unless it was calculated to influence the testator in making it.’ On the other hand, a will may be invalidated on account of the existence of mental states which would not be regarded as insanity from either a legal or medical point of view. Drowsiness and stupor resulting from erysipelas or fever, extreme weakness from cholera or other disease, and failure of memory in old age, have been sufficient to render wills void. It frequently happens that a medical man is called on to be witness to a will. On such an occasion it is his duty to satisfy himself as to the testamentary capacity of the testator. His subsequent evidence in regard to this, will, in case of dispute, be of almost decisive influence, if he has taken proper means of forming an opinion. In all cases, therefore, where there may be a possibility of doubt, it is well to require the testator to show that, without extraneous aid, and without referring to the document itself, he remembers and understands all the provisions of the deed.

*Evidence of the Insane.*—Lunacy was, till

a recent date, regarded by the law as incapacitating a patient from giving evidence in court. But according to the much more extended signification which the term lunacy has received, it now includes states of mind which are looked on as compatible with testimonial capacity. Where the judge is satisfied that the lunatic understands the obligation of an oath, and can give a rational account of such things as happen before his eyes, the evidence may be admitted. But the weight to be attached to such evidence will still depend on the extent to which it fulfils the conditions commonly required to constitute credibility. It has been held, however, that when a person has suffered from an attack of insanity between the occurrence of the transaction and the time he tenders his testimony, his evidence cannot be admitted.

*Management of Property.*—Where persons are supposed to be unable, from unsoundness of mind, to undertake the management of their own property, it may be necessary that they should be placed under the protection of the Court of Chancery; but this proceeding is not usually had recourse to unless there is urgent necessity, or a strong probability that the person’s incapacity will be permanent. It is consequently resorted to chiefly in chronic or congenital cases where there is no room for doubt as to the mental condition of the individual; and in cases of recent insanity, where it is necessary to have recourse to an asylum for the protection of the individual, it may also be necessary to obtain protection for his property by the aid of the Court of Chancery. In giving evidence or framing a statement in such a case, it is important, if incapacity is to be proved, to show that the individual has been found, when placed in circumstances requiring such capacity, unable to perform the acts which the management of property necessitates. In cases of active insanity it is especially required to show, not merely that there is delusion or other symptoms of insanity, but that the insanity is of such a nature as specially to disable the person from duly performing the duties which would be required of him. Difficulties most frequently occur in cases of imbecility and dementia; but the verdicts in such cases, when disputed, will generally be found to rest rather upon the impression produced by evidence of the actual behaviour of the individual, than upon the mere medical view of his mental condition. The most effectual aid that the medical witness can render in such cases, is to show whether there are or are not such peculiarities in the conduct of the person under inquiry as are known to be characteristic of imbeciles or demented persons. In undisputed cases, where the duty of the medical man consists merely in making an affidavit, there is no special difficulty to be encountered. Brevity, scrupulous accuracy,

and attention to the fact that such unsoundness of mind as involves incompetency to manage property must be established, are the most important requirements. A person found by the court to be incapable is placed under the control of a 'committee of the person,' and the property under a 'committee of the estate.' In Scotland, proceeding by petition to the Court of Session, or, in the case of small estates, to a sheriff, for the appointment of a *curator bonis*, takes the place of the English inquisition. The chief peculiarities of the Scotch process are that it is cheaper, more easily effected and more easily annulled, and that it does not affect the person of the lunatic. The functions of the curator correspond to those of the committee of the estate in the English court. The Scotch procedure for the appointment of a guardian of the person was virtually in desuetude until the passing of a recent statute (31 & 32 Vict., c. 100). Under this Act a brieve for the cognition of an alleged lunatic is issued from Chancery, and tried before a judge of the Court of Session and a special jury. The procedure is similar to that of jury trials in other civil causes in Scotland, and both medical and other evidence must be produced. If the person so cognosed be found 'furious, fatuous, or labouring under such unsoundness of mind as to render him incapable of managing his affairs,' his person is placed under the guardianship of the nearest male relative found competent.

*Drunkenness.*—This condition is not held to deprive a man of civil capacity unless it has at the time rendered the individual unconscious of what he was doing.

JOHN SIBBALD.

**CLAP.**—A popular name for gonorrhœa. See GONORRHEA.

**CLAREMONT, in Cape Colony.**—See AFRICA, SOUTH.

**CLAVUS HYSTERICUS** (*clavus*, a nail).—An acute pain, often associated with hysteria, but occurring also in other conditions, which is felt in a localised point in the head, and is compared by the sufferer to the sensation that might be produced by a nail being driven into the part. See HYSTERIA.

**CLIMACTERIC** (*κλιμακτήρ*, a step of a ladder).—This word, which properly signifies 'by degrees,' was originally employed to indicate certain epochs or periods in the life of an individual, which were looked upon as critical, and at which the body was supposed to have undergone a complete change, so that it had become entirely renewed in its structural elements. The years in which these epochs terminated were called climacteric years—*anni climacterici*—and their number was variously estimated. Thus, some only recognised three climacterics; the Greek physiologists held that there were

five, ending at the seventh year, the twenty-first ( $7 \times 3$ ), the forty-ninth ( $7 \times 7$ ), the sixty-third ( $7 \times 9$ ), and the eighty-first ( $9 \times 9$ ); others made them multiples of seven or nine, or multiples of seven by an odd number. Most regarded the sixty-third year as the *grand climacteric*, but the Greeks recognised two grand climacterics, terminating respectively at the sixty-third and eighty-first years, and this special denomination was given because there was little, if any, prospect of life being extended beyond these periods. At the present day the word 'climacteric' has lost much of its original meaning, and is generally applied to certain times of life, without any reference to numbers of years, at which marked physiological or developmental changes occur, such as the period of puberty, or that of the cessation of menstruation.

A particular *climacteric disease* has been described, which is said to occur either about or subsequent to the sixty-third year or grand climacteric, and supposed to be distinct from the natural decay and degeneration which takes place in advanced life, inasmuch as recovery often ensues. It is stated that the complaint comes on suddenly, but advances insidiously, the symptoms being at first loss of flesh and weakness, followed by loss of appetite and dyspeptic symptoms with a white tongue, which are regarded as sympathetic, sleeplessness or disturbed and unrefreshing sleep, constipation, pains in the head and chest, a frequent pulse, swelling of the legs, and an emaciated or bloated appearance of the face. The urine does not present any abnormal characters, and most of the viscera seem to perform their functions properly. Whether there is any independent disease deserving this special denomination is, in the writer's opinion, extremely doubtful.

FREDERICK T. ROBERTS.

**CLIMATE.**—Formerly the word climate (from the Greek word *κλίω*, I incline) was a term of astronomical or mathematical geography, which implied a portion or zone of the earth's surface comprised between two lines parallel to the equator, and measured by the length of time during which the sun there appears during the summer solstice, that is, by the sun's inclination. The space between the equator and the pole was divided into half-hour climates, in which the length of each day increased by half an hour; and also into monthly climates. This unequal division of each hemisphere is now replaced by a division of the interval between the equator and the poles into ninety degrees, which constitute what are called degrees of latitude, and the word 'climate' has received a more extended application.

By climate is now understood those conditions of heat, moisture, atmosphere, wind, soil, and electricity, which impress certain

conditions, uniform even when apparently irregular, on given portions of the earth's surface, and which modify, also in a uniform manner, vegetable and animal life.

CLASSIFICATION.—Climate, when thus interpreted, is still principally dependent on astronomical facts, on the sun's position or inclination with regard to the earth, and on the amount of heat it supplies to different portions of the surface of the latter. Climate may be studied generally and locally. The division of the earth's climates is necessarily arbitrary, and many different classifications have been proposed. The most simple is that which recognises three principal kinds of climate, each susceptible of subdivision, viz.: *warm* climates from the equator to  $35^{\circ}$  lat.; *temperate* climates from  $35^{\circ}$  to  $50^{\circ}$  or  $55^{\circ}$  lat.; *cold* climates from  $50^{\circ}$  or  $55^{\circ}$  to the pole. As subdivisions we may recognise equatorial, tropical, sub-tropical, sub-polar, and polar climates; and also *insular* and *maritime* or moist climates, and *continental* and *mountain* or dry climates.

1. *Warm Climates*.—Warm climates, extending from the equator to  $35^{\circ}$  lat., that is,  $12\frac{1}{2}^{\circ}$  beyond the tropics, comprise nearly all Africa and its islands, South Asia, most of the islands of Polynesia, and the portions of North and South America comprised between California and the north of the La Plata territory. In the equatorial regions the medium temperature for the year is from  $80^{\circ}$  to  $84^{\circ}$  F., the min. being  $54^{\circ}$ , the max.  $118^{\circ}$ . Near the equator the annual mean temperature decreases slowly as we recede from it, the decrease not amounting to more than  $2^{\circ}$  F. for the first  $10^{\circ}$  lat. The difference of temperature during the day is slight, but much greater during the night, owing to radiation. The general variations of the barometer are slight, but the periodical or diurnal variations are very marked. It ascends and descends regularly twice in the twenty-four hours. It ascends from 4.13 a.m. to 9.23 a.m., and descends until 4.8 p.m., ascending again until 10.23. Electrical phenomena are very decided. The rainfall is variable, but 40 inches may be given as a mean. It is generally supposed that heat is greatest at the equator, and diminishes as we recede from it; but both observation and astronomical induction lead to the conclusion that not only the maximum of temperature in warm climates is attained at or near the tropics, but also the highest annual mean. The countries in which the highest degree of heat is known to be attained are near the tropic of Cancer, as, for instance, the banks of the Senegal, the Tehama of Arabia, and Mehran in Baluchistan. Moreover, the snow-line, or the line of perpetual snow, is higher at the tropics than at the equator. In the Bolivian Andes, near the tropic, it is 17,000 feet, whereas in the Ecuador Andes, on the equator, it is only 16,000 feet. These facts

are partly explained by the unequal progress of the sun after the equinox in its course towards the tropic. In the first month it passes through  $12^{\circ}$  of latitude, in the second month through  $8^{\circ}$ . At the end of the second month, therefore, it is  $20^{\circ}$  from the equator, and there remain only  $3\frac{1}{2}^{\circ}$  to be traversed in the third month. The sun receding from the tropic at the same rate at all places between  $20^{\circ}$  and  $23\frac{1}{2}^{\circ}$  of latitude, the solar rays during two months fall at noon either perpendicularly or at an angle which deviates from a right only by  $3\frac{1}{2}^{\circ}$  at most.

Another cause which tends to diminish heat in the regions near the equator is the prevalence of rain. For about five degrees north and south of the equator, in the region of the equatorial calms, there are few consecutive days in the year without rain. The principal cause both of the calms and of the rains has been attributed to the meeting in the upper atmospheric regions of the trade winds, north and south. They neutralise each other, and precipitate the vapour they hold in solution.

Regions that lie between  $5^{\circ}$  and  $10^{\circ}$  of latitude have usually two rainy and two dry seasons. The greater rainy season occurs when the sun in its passage to the nearest tropic passes over the zenith, lasting from three to four months. The lesser rainy season occurs when the sun on its return from the nearest tropic approaches the parallel of the place. The rains then only last from six weeks to two months, and are much less abundant and continual. Countries more than  $10^{\circ}$  or  $12^{\circ}$  from the equator have only one rainy and one dry season; the first begins when the sun approaches the nearest tropic, and ends some time after, when in its course from the tropic it has passed the parallel of the place. It lasts from four to six months. Local conditions may modify the course of the dry and wet seasons, as is the case in India, where the dry and rainy seasons depend principally on the monsoons. The amount of rain that falls in a short time within the tropics is very great, much more so than in more northern regions, but these heavy rains do not last continuously, as is supposed. Days of continued rain, even in the rainy season, are rarer than in the north. Still, heavy rains are apt to cause great inundations, and to cover large extents of low or level country with water, producing swamps and marshes, very injurious to health.

In the vicinity of the tropics there is a belt, extending over several degrees of latitude, where it seldom rains. This rainless tract is precisely the region which has been already mentioned as that of greatest heat. These belts of rainless regions, extending around the globe on each side of the equator, may be said to separate the countries which lie on each side of the equator from the

temperate zones. Thus in Africa the rains cease on the southern border of the desert of Sahara at about 16° N., and begin again at 28° N. On the banks of the Nile the rain ceases about 18° or 19°, to begin again between 28° and 29°. The Tehama, or low coast of Arabia, is all but rainless. This rainless tract crosses Asia as far as China, where there is no rainless region, owing, probably, to the fact that all parts of China between 22° and 30° N. lat. are traversed by high mountain chains.

The influence of warm climates impresses certain peculiarities on the people who inhabit them. They are the abode of the Ethiopian and Mongolian races of mankind, and appear to have impressed the same characteristics, in a minor degree, on the Caucasian races that inhabit them: a dark complexion and black hair. The inhabitants of these countries are indolent and apathetic. The functions of the skin and liver are peculiarly active, a circumstance which exposes them to severe disease of these organs. The digestive functions are sluggish, and the nervous system is alternately excited and depressed. Remittent and intermittent fevers, dysentery and yellow fever, are common. During the dry season disease tends to assume the ataxic, during the rainy season the adynamic form. Pulmonary consumption is frequently met with in the towns, in contradiction to received opinions.

2. **Temperate Climates.**—Temperate climates may be said to occupy the zones of the earth's surface comprised between 35° and 50° or 55° lat. They comprise Southern and Central Europe, with its islands; the parts of Asia which extend between the Black Sea and the Mediterranean on the west, and Japan on the east; the greater part of North America; a part of Chili and La Plata and Patagonia in South America. The mean temperature may be stated at from 60° to 50°. The climates in which the mean temperature is from 60° to 68° are often spoken of as temperate, but in reality they approximate closely to warm climates. The four seasons, winter, spring, summer, and autumn, are well-marked, but are very variable both as to barometrical and thermometrical conditions. The mean temperature in the central regions is, for winter 38°, for spring 51°, for summer 68°, and for autumn 53°. The regions which are near the south and north limits of the northern temperate zone approximate to the meteorological characters of the warm and cold climates respectively. The periods of the year when storms, rain, and general versatility of meteorological phenomena are principally observed correspond with the vernal and autumnal equinoxes.

The influence of a temperate climate on the human organisation is salutary, extremes of heat and cold being both trying. Thus the healthiest climates of the world's surface

are found in this zone. Intense heat, or even moderate heat if persistent, throws a physiological strain on the liver, skin, and digestive system, and renders mankind prone to severe and fatal diseases of these organs. Intense cold throws a physiological strain on the lungs and kidneys, and exposes them also to severe and fatal disease. The healthiest temperate climates are those in which the winter is not very cold and the summer is not very warm, and in which, consequently, there is no great or continued strain on any one class of organs. The diseases of temperate regions are those that are the best known, as their study and description constitute the foundation of pathological science, ancient and modern.

The climate which, perhaps, the best deserves the appellation of temperate is that of the Mediterranean basin. The winters are not severe on any part of its north shores, and the summers are not intensely hot on its south shores; at least the heat falls short of that of the tropics. There are many conditions of physical geography which conduce to this result. The north shores are protected from north winds by the ranges of high mountains of Southern Europe which skirt them, and the south shores are in close proximity to the hot, rainless tract of Northern Africa—the desert of Sahara, which favourably modifies winter temperature. Moreover, the Mediterranean is a warm sea, but few cold rivers of considerable size flowing into it from the north, a fact which increases the temperature on its shores and islands.

3. **Cold Climates.**—Cold climates comprise the regions which extend from 50° to 55° lat. to the poles. They may be subdivided into *cold*, with a mean of from 50° to 40°; *very cold*, with a mean of from 40° to 32°; *glacial*, with a mean below the freezing-point. In the austral hemisphere the zone contains but little known land, although the existence of an antarctic continent is suspected; in the northern hemisphere it comprises, in Europe, the north of Scotland, Denmark, Sweden, Norway, Iceland, Finland, Lapland, Northern Russia, Spitzbergen, Nova Zembla; Northern Asia, and some of its large plains below 50° lat., Siberia, and Kamtchatka; in America, Canada, including some regions below 50°, the northern lands and islands of Hudson's and Baffin's Bays, and Greenland. In this zone the decrease of the mean temperature is much more rapid as we recede from the equator, than it is in the tropical regions. Thus from the equator to 20° lat. the variation of the mean temperature is not more than 7° or 8°, whereas the variation between 55° and 75° lat. amounts to from 22° to 27°. The coldest region of the globe is not, it would appear, at or near the pole, but at about 80° lat., or 10° from the pole, north of Behring's Straits: the cold of the glacial climates has been exaggerated. At the lati-

tudes of from 70° to 78°, the extreme limit of human habitation, the mean annual temperature is between 19° and 17°, *i.e.* 13° to 15° below the freezing-point. The extreme of cold registered, however, reaches a hundred degrees or more below the freezing-point. Owing to astronomical conditions there is great disproportion between the length of the nights and of the days at different seasons of the year. In the more northern regions, for several months in the winter the sun never appears above the horizon, and in the summer for several months the sun never disappears below it. Spring, during which the extreme cold is mitigated, lasts but a very short time, and is succeeded by summer, which is in its full strength in June and July. Temperature rises rapidly from 35° to 55° and 60°. In some northern localities it rises to 86° or 90°. Under the influence of the prolonged or persistent days, and of the increased temperature, the vegetation peculiar to each locality passes through all its phases with extreme rapidity. Towards the end of July rain and fog reappear, and are followed by snow and intense cold, the highest expression of which is in January and February. The barometrical changes are the reverse of what obtains in the tropics. Above 60° lat. the diurnal or periodical changes are scarcely perceptible, whereas general or occasional variations become more marked as we approach the pole. Electrical phenomena become less marked, and above 68° lat. they are scarcely perceptible, with the exception of the aurora borealis. The winds which predominate are the N.E. and S.W., and they change rapidly from one point of the horizon to the other, and thus frequently occasion tempests which extend over considerable areas. The quantity of rain that falls in cold climates is much less than in the tropical and temperate, with some exceptions. Between 60° and 90° lat. it only amounts to a few inches, and falls principally in the form of snow.

*The influence of cold climates is shown on the inhabitants of these countries, who vary much in stature, and possess a vigorous constitution, a sanguineous temperament, great muscular development, active digestive functions, and sluggish nervous powers. Notwithstanding the severity of the climate, they generally succeed in preserving health, and live to old age, presenting few diseases referable to climatic influences. They are, however, subject to ophthalmia and amaurosis, owing to the reflexion of light from the snow in the polar regions, and to scrofula and scurvy, the result of a poor and incomplete dietary. Agues and intermittent fevers from marsh influences are rare, and not severe, and disappear entirely as we approach the pole. Continued fevers are met with, but seldom if ever epidemically.*

**4. Insular Climates.**—Insular climates

present important peculiarities. The temperature of the sea is more equable than that of the land. Owing to the action of currents, and to the circulation of its waters under the influence of heat, its superficial temperature is warmer in winter and cooler in summer—more equable—than that of the land. It has thus a tendency to warm in winter and to cool in summer the island which it surrounds. Moreover, there is constantly watery vapour arising from the sea, which extends to island atmospheres, veils the sky more or less, shields the surface from the ardour of the sun in summer, and prevents great radiation both in summer and in winter. Thus it is that the climate of all islands is more equable than that of continents. This fact is more especially recognisable in the climate of the British Isles, which is also modified—rendered warmer and moister—by the waters of the Gulf Stream impinging on their shores. The warm Gulf Stream, commencing in the tropics, in the Gulf of Mexico, passes northwards along the shores of North America, crosses the Atlantic to the south of Newfoundland, and strikes the shores of the British Islands, of Norway up to Cape North, of Holland, and of France; everywhere raising temperature and the annual mean.

**5. Maritime Climates.**—Maritime climates participate in these influences: temperature is more equable, warmer in winter and cooler in summer, on the shores of seas and oceans than it is inland. Owing to this cause most of the winter sanitarium have been chosen in islands or on the coasts of oceans and seas: such as Hastings, Ventnor, Bournemouth, Torquay, Funchal, Malaga, Cannes, Nice, Mentone, Naples, Salerno, and Algiers.

**6. Continental Climates.**—Continental climates exhibit conditions the reverse of those which obtain in maritime climates. The tendency is to cold winters and warm summers, owing principally to the absence of the equalising influence on temperature of large masses of water. A very short distance from the sea is sufficient to establish this difference. Thus the central parts of France are very much warmer in summer and colder in winter than the coasts of Normandy and Brittany. But it is when we reach the centre of continents—Russia, Central Asia, Central America—that the difference is the most marked.

**7. Mountain Climates.**—As we rise above the level of the sea, we meet with two important meteorological conditions. The air becomes more and more rarefied, and the heat diminishes, independently of the more or less obliquity of the sun's rays. The higher we rise above the sea-level the more the air is rarefied, and the more the degree of heat due to the solar rays diminishes. We arrive at last, even at the tropics or the equator, at a height, variable according to

latitude, where the sun's heat is insufficient to melt the snow. This is termed the snow-line. Mountains attract clouds and watery vapour, and the coolness of their atmosphere causes the precipitation of the vapour in the form of rain or snow. Thus, mountains, mountain-ranges, and the glaciers they contain, are the principal cause and origin of rivers.

*The influence of mountain climates, notwithstanding the clouds, fogs, and rain which characterise them, is proverbially a healthy one, owing to the purity of the air, the shelters found in the valleys, and, perhaps, to the sparseness of population. They have of late been much recommended for phthisis. The conditions above mentioned, together with sunshine to a limited extent, are quite sufficient to account for the rarity of phthisis amongst their inhabitants, and for the improvement of the phthisical who resort to them.*

*Soil modifies climate considerably. Wet and marshy soils are cold, engendering fog and mist. Sandy, dry, well-drained soils are comparatively warm. All sandy plains are warm in summer and cold in winter.*

Such are the features which characterise the climates of the terrestrial globe, generally. Each locality, however, each mountain, plain, and valley, each city, village, and house has a climate of its own, modified by all the meteorological elements which we have successively considered. To discover what each climate is, we must study carefully the meteorological conditions and influences which we have rapidly surveyed in their application to it.

*Isothermal Climates.*—Owing to the unequal influence of the different elements that constitute climate, the annual mean temperature of regions occupying the same latitude on the earth's surface is very variable. Hence the above name has been given to the regions in which the annual mean is the same. The study of a map on which the isothermal lines are marked is an instructive illustration of the facts above described. Thus it will be seen at a glance that the limit of constantly frozen ground in the central plains of Asia and in the northern plains of America is below  $54^{\circ}$ , which is the latitude of York!

**THE GENERAL INFLUENCE OF CLIMATE.**—The vegetable and animal worlds, including man himself, have been modified in essential characters by climatic conditions. The study of its influence on their vitality and organisation opens out a wide field to the observer. This study leads to the conclusion that geological periods of time have been necessary to impress on terrestrial life, be it vegetable or animal, the characteristics observed now or formerly in the climates of different regions of the earth. The current of modern research is strongly directed to the elucidation of the

influence of climate on life during such geological periods. The investigations of Darwin and his followers may be named as the most remarkable and important illustration of this fact. We may also mention the researches of modern philologists, which have proved, by the study of languages and their roots, that most of the nations of modern Europe have descended from the same Aryan parents as the inhabitants of the Indian peninsula. Climate, in the course of time, has so modified them as apparently to produce different races. For such a change to take place geological lapses of time are certainly required. Our earliest records, dating back several thousand years, show these races, such as they are now, quite as distinct. In these days the Aryan races of Europe cannot rear their children in the climate of India, where their Hindoo relatives thrive and propagate their species. In Palestine and Egypt the biblical records, those of the Pharaohs, and those of Nineveh and Babylon, show these regions to have been inhabited, several thousand years ago, by nations and tribes presenting precisely the same race characteristics as those that now inhabit them. During the historical period, the races of Europe have in vain endeavoured to colonise the valley of the Nile; for they have not been able to propagate their species, and have died away, leaving the valley of the Nile to its ancient inhabitants. Their children cannot withstand the heat of summer. On the north-eastern shore of Africa, the Algeria of the present, history presents the same record. The Romans and the Visigoths occupied its plains for centuries, continually recruiting their colonies from the mother-countries; and yet, except in the mountains, all trace of their presence has disappeared. They could not rear their children so as to occupy the land of the Arabs. What lengthened periods of time must have elapsed to so profoundly modify races deriving their origin from a common parentage, that they can no longer live and propagate their species in the same climates!

**THERAPEUTICAL APPLICATIONS.**—The therapeutical application of many of the above facts is contained in the facts themselves. Reasoning suffices to deduce the therapeutical law, and experience proves the correctness of the deduction. Only a very brief summary can here be given. See also **CLIMATE, Treatment of Disease by.**

Firstly, it is clear that if a local climate produces injurious effects on the health of its inhabitants, or of visitors, the latter should leave it, and the regular inhabitants, who cannot leave it, should endeavour by every possible means to modify the pernicious climatic influence to which they are exposed, and to partially escape from its action. This rule may be illustrated by the influence of confined mountain valleys in producing

goitre, and of marshy districts in producing intermittent fevers. The principle applies to all local climates which exercise a pernicious influence of any kind on the human organisation. When possible the climate should be abandoned; if that is impossible, its pernicious influence must be combated by every possible hygienic and therapeutical means.

The injurious effects which extreme climates exercise on the human economy—warm climates on the abdominal and cerebral organs, cold climates on the thoracic—point to change of residence as an important therapeutical agent, the value of which is only beginning to be understood. Our naval and military surgeons have done much to clear up this branch of therapeutics, as regards the diseases of tropical regions. Thus chronic affections of the liver and intestines, incurable in a warm climate, often become quite curable if the patient is transferred to a temperate region or to a mountain elevation in the tropics, which reproduces a temperate climate.

Inversely, persons suffering from diseases of the respiratory organs, so common in damp temperate climates like those of France, England, and Holland, find relief by migrating, especially during winter, to warmer regions of the earth's surface, where they escape from the influences which have proved so detrimental to them. Thence the yearly increasing exodus of persons suffering from chronic laryngitis and bronchitis, from bronchial asthma and from phthisis, from the north of Europe to the south.

The increased facilities of locomotion, by rail and by steam, have thus opened out, as it were, a new and important branch of therapeutics, that of the application of climate to the treatment of disease.

J. HENRY BENNET.

**CLIMATE, Ætiology of.**—See DISEASE, Causes of.

**CLIMATE, The Treatment of Disease by.**—Though we can scarcely say with accuracy that change of climate is a specific for disease, yet much can be effected by it in relieving symptoms, and in assisting the recuperative powers of the organism by thus improving the general health. The diseases in which change of climate has been found of value will be enumerated below, with a short notice of various climates. Here we may notice the *rationale* of the benefit to be derived from such change.

Change of climate, we must premise, is only a relative term. It does not necessarily involve the idea of removal to a great distance from the patient's home. A few miles' journey from the town to the country, from inland to the seashore, from the plain to the mountain, often suffices to produce marked results. One use of climate being to expose

the organism to the effects of *contrast*, the element of distance comes in most when we wish to make the contrast greater; for instance, in ordering change from a cold to a warm climate or *vice versa*.

The therapeutical elements of most importance in any climate are: (1) pure air, free from dust and organic particles, or excessive damp; (2) abundance of sunshine, without excessive heat, so that much time can be spent in the open air; (3) a temperature without extremes, so that the body is not exposed to the risk of great variations of heat and cold—equability; (4) absence of violent, very cold, or very hot winds, at any rate of long duration (in this is involved the element of local shelter).

These four elements should be present in each of the subdivisions of climates which a therapeutical classification renders necessary, namely, climates of: (1) *the seashore*; (2) *mountains*; (3) *inland wooded districts*; and (4) *the open sea*.

The epithets 'moist' and 'dry,' which are applied to climates, are merely relative, and depend on local peculiarities of rainfall, soil, &c., as well as to some extent on season; and the essential differences between the climate of the seashore, the woodland, and the mountain remain everywhere the same. We shall say a few words about each of these, with the indications for their use. The climate of the open sea will be referred to in speaking of sea voyages.

**1. Climate of the Seashore.**—The special peculiarities of this variety of climate are that the air is saturated with moisture, except when dry land winds prevail; it is dense, and, as a rule, therefore, bulk for bulk, contains more oxygen than air of any higher level; its density is liable to great and frequent but regular variations, which increase the activity of the circulatory and respiratory organs, and thus favour their functional activity; it is more equable; and, lastly, it contains saline particles in suspension.

According to Beneke, sea air cools the body relatively more quickly than mountain air, and thus hastens more the processes of tissue-change. Hence, the seaside should be ordered where we wish for a highly stimulating effect, as in persons of scrofulous tendency, in chronic succeeding acute diseases, or in the later stages of convalescence from the latter, in convalescence from surgical operations, or in some surgical diseases where we wish to accelerate tissue-change, without exertion on the patient's part. On account of the equability of the climate, some patients who cannot bear great changes of temperature do well at the seaside. Persons suffering from overstrain, mental or bodily, with a fair digestive power, and not liable to nervous irritability, may also be sent there.

**2. Mountain Climates.**—Mountain climates are distinguished from seaside climates by the lower density of their atmosphere; their lower and less equable temperature; by less humidity, though, owing to local winds, mist and cloud often form; and by relatively lower night-temperatures in clear weather, owing to the dryness of the air, and consequent great radiation. They are cooler also than the inland climates of level districts, and this coolness tends to some extent to diminish the rarefaction and increase the density of their air.

The general action of mountain air is to produce a freer circulation of the blood and greater vascularity of the lungs, owing to deeper and more frequent inspirations and greater ease of bodily movement. Owing to the cooling of the body by the lowered temperature more food is required. The appetite improves, and the body becomes better nourished and gains weight.

The intensity of the effect is, broadly speaking, directly as the height. The term 'mountain climate' is applied in medical parlance to elevations in Europe of from 1,500 to 6,000 feet, though in South America patients have been sent as high as 10,000 feet, or higher.

Mountain climates are indicated: (1) in cases of hereditary tendency to phthisis in young persons with narrow, shallow chests, who are growing too fast; also in young scrofulous patients. (2) In chronic phthisis and pneumonia; remembering, however, that phthisis occurs at all elevations. The coolness of the mountain air in the height of summer is an important element in phthisical cases, which always suffer from great heat. (3) As a tonic and restorative in persons suffering from over-work in business or literary pursuits, and who have no real organic disease. (4) Generally to complete the convalescence from acute diseases of individuals not past middle life, with a fair amount of muscular power and bodily activity. (5) As a prophylactic against hay-fever, cholera, and other infectious diseases. Mountain climates are not advisable in cases of chronic bronchitis, heart-disease, emphysema, Bright's disease, chronic rheumatism, hæmoptysis, nor for aged persons.

**3. Climate of Inland Wooded Districts.**—The climate of wooded districts (elevations above 1,500 feet are not here referred to) is peculiar in several respects. It has a temperature lower than that of the surrounding country—on the average 3° F.—during the hours of daylight; the temperature is also more equable. The relative humidity is higher (9·3 per cent.) in summer than in the less wooded country, and hence there is greater liability to rain and mist. It also affords greater protection and shelter against winds than other climates.

The general effect of woodland climates, as

may be deduced from the above, is sedative and tonic. They may be advised in chronic bronchitis, emphysema, heart-disease, and in hypochondriasis, hysteria, and other nervous affections where tranquillity and subdued light are of importance; also in the earlier stages of convalescence from acute disease when sea or mountain air is too stimulating. In bronchitis pine-woods should be selected, and in heart-cases level walks are essential, unless exercise be deliberately ordered for certain kinds of cardiac disease in the form of graduated hill-climbing.

**4 Ocean Climate: Sea Voyages.**—Voyages have been much recommended in the treatment of phthisis in its early stages, with a view to enable the invalid to spend much time in an exceedingly pure and fairly equable atmosphere, and to secure a sufficient amount of bodily movement without great fatigue.

The drawbacks to the sea ought to be provided against as far as possible, although certain of them from their very nature must be endured. The principal of them are the impossibility of escape from bad weather, and the confinement, perhaps, to ill-ventilated cabins, when such occurs; the absence of sufficient light and air below decks, the latter being felt very much at night; want of variety in the diet after a certain time, and in many instances of fresh food, milk, &c.; monotony in society and occupations; and, lastly, the inconveniences arising from crowding of the main-deck with hen-coops, sheep-pens, &c., and in steamers from the smoke of the engines, and the smell and vibration of the machinery.

The routes generally recommended to invalids are chiefly: (1) to Australia, 90 days in a sailing vessel, 45 in a steamer; (2) to the Cape of Good Hope, 20 days; (3) to the West Indies, 17 days; and (4) to the United States or Canada (in summer), 6 to 7 days. Short cruises in the Mediterranean, or to the latitudes of the Canaries and Azores, are suitable for certain cases where expense is no object. Of routes (3) and (4) we may say that they are too short for the full benefit of the sea to be obtained, as improvement does not generally begin for a week or two after sea-sickness has subsided, and the patient can remain comfortably on deck. Route (2) does not allow him to get the bracing effect of high South latitudes. Hence where a long sea voyage is indicated, route (1) is decidedly the best. England is quitted in the beginning of October, Australia (Sydney or Melbourne), or New Zealand (Wellington), is reached early in January, and the return voyage is begun not later than the end of February.

Patients should not remain in the coast-towns of Australia in summer on account of the heat and the dust. They should go to the table-land of New South Wales, or to the Darling Downs in Queensland, or else they

should cruise from port to port, or cross to New Zealand or Tasmania.

In returning, the route round Cape Horn should be *avoided*, on account of the great risk of the climate of the South Pacific Ocean, and the chance of encountering icebergs, fogs, and unfavourable winds. Either the patient should come back round the Cape of Good Hope; or, if his strength permit, he may cross to California, travel overland to New York and thence by steamer; or, lastly, he may come by the Red Sea, Suez Canal, and Mediterranean.

The invalid must expect about 20 wet days on the voyage out. The temperatures met with range from 40° to 80° F., the coldest and most uncertain weather occurring in the North Atlantic and South Pacific Oceans.

*Choice of Vessel.*—The following considerations may be useful in deciding between steam and sailing vessels:—

For a steamer there is the greater certainty in predicting the length of the voyage, and the calms of the tropics are sooner passed.

Against steamers there is the nuisance of steam and smoke on deck; much space is taken up by the engines, stokers' rooms, &c., and hence other parts of the ship are more crowded; the bilge water is tainted with the engine grease; there is the wearying grind of the screw by day and by night; while more seas are shipped, because a steamer can run against a head-wind.

For sailing vessels there are the advantages of more room, light, and air in the cabins, and the absence of the above-mentioned disagreeable conditions.

Against them there is the longer and more uncertain duration of the voyage, the necessity of shaping their course by the prevailing winds, and the possibility by route No. 1 of being becalmed at the equator for days or weeks, in a humid atmosphere of 80°–85° F.

The vessel selected should not be under 1,000 tons, and her age, class of passengers, the character, temper, and standing of her captain, as well as the reputation of the owners, and the size of her cabins, should be carefully ascertained. Of course it is a *sine qua non* that she should carry a duly qualified surgeon.

The cabin for the outward voyage to Australia should be on the port side, so as to get the breeze in hot weather, and *vice versa* in returning.

The cabin outfit should include a spring mattress, with hair (not wool) mattress over, a folding easy chair, chest of drawers, carpet, curtains, and sponge bath (Faber). Clothes of various degrees of thickness are essential, both for body and head, and a waterproof suit is necessary for bad weather.

Plenty of linen must be taken, as washing is difficult on board, and there should be a supply of preserved milk, meat or essence of meat, fruit, and light wines.

The *indications* for a sea voyage are hereditary tendency to phthisis, or the presence of actual but uncomplicated disease in a very early stage, in persons not past middle life, with a fair digestion, absence of severe pyrexia, and general health not much impaired. Patients with a tendency to hæmoptysis should not be sent, nor should those of a desponding disposition, who would thus be likely to suffer by the long absence from home, or from fears of their personal safety. Of course a tendency to protracted sea-sickness is a distinct contra-indication.

The invalid should be careful not to overtax his digestion too much; he should take regular daily exercise on deck to the extent of his strength, have some definite occupation to beguile the time, and, if possible, be accompanied by a personal friend (Faber).

*Choice of a Climate—General Hints.*—It is a good plan, if possible, to order a patient a climate with that mean temperature and relative humidity which he is known to tolerate well (Sigmund). The patient's disposition must be considered, and a lively or a quiet place chosen according to his temperament. We should not send a poor man to a place beyond his means, otherwise he has to grudge himself many comforts, and loses much, if not all, the benefit of the change. In sending patients to the South of Europe this rule is too often neglected.

The special indications for the climates of particular places can only be understood by studying their local aspect. Generally speaking, as far as Great Britain is concerned, the climate of the east coast is colder and drier than that of the west and south coasts. In Europe the north and west coasts are moister and cooler than the shores of the Mediterranean. As to season, mountain climates include some very important winter stations; woodland climates are almost exclusively indicated from May to the middle of July. Certain parts of the sea-coast are adapted for invalids at all seasons of the year; but as a rule the northern coasts of Europe and the eastern or south-eastern coasts of Great Britain are best suited for summer, and the south, west, and south-western for winter residence. The Mediterranean coast is only to be recommended from mid-October to the middle or end of May, and Egypt should be quitted not later than April. A word may be added as to the advantages of wintering in the South of Europe. It is incontestable that the invalid gets a milder winter, a longer autumn, and an earlier spring. Although there is no place where some days of bad weather do not occur, or where uninterrupted calms are met with, yet the number of rainy days is fewer, there is more sun, little or no fog, and, except in the neighbourhood of the Pyrenees, little or no snow or ice. The scenery is picturesque and attractive, and the invalid is able to spend much time in the open air, and to sit out of

doors on many days, even in mid-winter. The drawbacks to the South are the risk of chills, owing to the difference between sun and shade temperatures, especially at first, when persons are unaccustomed to the climate, and fail to take sufficient care; the occasional occurrence of high winds, especially in spring; the more limited accommodation, owing to the expense of rooms and living; and the absence of many so-called 'home comforts.'

Those who visit the South must remember that the curative value of the climate consists in its *allowing much time to be spent in the open air*, and in its milder temperature and drier air, which protect the respiratory organs from fresh inflammatory attacks. A south room and warm clothing of the texture usually worn in England in autumn are essential, and a coat or wrap should always be carried out of doors in mid-winter to put on in passing from sun to shade. The invalid should strictly avoid the hot atmosphere of gaslit salons at night.

Patients with acute diseases of the respiratory organs should not be sent to the South; and high fever, excessive weakness, or the necessity of remaining in bed, are also contra-indications, owing to the fatigue and risks of the journey and the need of home comforts. Cases of mental disease with excitement, where rest and protection of the mind and body are of primary importance, should likewise not be sent.

In the convalescence from acute diseases occurring in autumn, where a cold northern winter would prevent open-air exercise, and probably set up fresh exacerbations, southern winter climates are of great value.

In ordering change of climate the accommodation, food and water supply, soil and drainage of the locality chosen, should be carefully considered, especially if the distance be a long one. The best climate may be unavailable for the invalid, owing to defects in one or more of these particulars.

Lastly, the patient's own feelings should be carefully consulted before he is sent far away from home. In some cases all the benefits of climate are counteracted by 'homesickness.' *Cælum non animum mutant qui trans mare currunt.*

**Enumeration of Climates.**—We shall now enumerate various climates and regions suitable for the treatment of cases that can be thus benefited.

**1. Of the Nervous System.**—In neuralgia:—Arcachon (for the calm, sedative, yet tonic atmosphere of the pine-woods), Cannes (the districts away from the sea), Upper Egypt (Cairo, Luxor, Helwan), Hastings, Hyères, the Engadine, the Bernese Oberland, Pau, Pisa, Rome, the Salzkammergut (Ischl, Aussee, Berchtesgaden). Some of the above climates will also be found suitable to cases of hemicrania and sciatica.

In hysteria, hypochondriasis, spinal irritation, and in some cases of protracted chorea:—Cannes, Ischl, Aussee, Meran, Mentone, Montpellier, Morocco (Tangiers), Nice, Naples, the Bernese Oberland (Gründelwald, Müren), Palermo, Pau, Seville, Spezia, and Valencia; the effect produced being chiefly due to diversion of the attention by the change of scene, although the bracing influence exerted on the system at large must be taken into account.

In chronic softening of the brain and spinal cord, in paralysis of cerebral origin, and in some cases of locomotor ataxy, the South of France may be advantageously ordered in winter, and Alpine climates of moderate height in summer. As a rule hot climates, or those where the sun has considerable power, are contra-indicated where there is a tendency to apoplexy or hyperæmia of the brain.

Temperate and bracing climates are, as a rule, to be recommended in nervous diseases, to restore the general tone of the system.

The immediate neighbourhood of the sea not infrequently causes nervous excitement, neuralgia, and sleeplessness.

**2. Of the Respiratory and Circulatory Systems.**—In chronic bronchitis, emphysema, bronchial and spasmodic asthma, as well as in chronic pharyngeal and laryngeal catarrh and laryngeal ulceration, the following climates may be recommended:—Africa (South), Algiers, Australia, the Azores, Bordighera, Bournemouth, the Canaries, Upper Egypt, Glengariff, Hastings, Hyères, Lisbon, Madeira, Malaga, Mentone, Meran, Montreux, Nervi, Nice, Palermo, Pau, Penzance, Pisa, Queenstown, Rome, San Remo, Santa Barbara, St. Leonards, Torquay, and Ventnor.

Change of climate is of great value in convalescence from the acute, and as a prophylactic and curative measure in the chronic forms of bronchitis; but we must remember that where there is copious expectoration a dry climate is indicated, while in the irritative forms with scanty sputa (*bronchitis sicca*) a moderately moist mild climate is generally suitable. In emphysema we should choose a mild and not too dry climate, if possible in the neighbourhood of pine-woods, such as Arcachon on the west coast of France, or Bournemouth. In spasmodic asthma the choice of climate must be partly a matter of personal experience.

In the early active, and in the quiescent forms of the later stages of phthisis, as well as in chronic pleurisy, and in convalescence from pneumonia, the following (chiefly winter) health resorts and climates have been favourably spoken of, and many of them will be found described in this work under their own or associated headings:—Africa (South), Algiers; Australia, Bordighera, Bournemouth, Canaries, Cannes, Colorado Springs, Denver, Davos, Upper Egypt, Upper Engadine, South

of France, Falmouth, Hastings, Hyères, Queenstown, Ischl (in summer), Madeira, Malaga, Mentone, Natal, Nervi, Nice, the Oberland (in summer), Palermo, Pau, Pisa, St. Paul's in Minnesota, San Remo, Santa Fé, Sicily, Spezia, Torquay, and the Undercliff (Isle of Wight).

In chronic endocarditis, pericarditis, and in heart-disease generally, a rather bracing climate, without extremes and of the character suited to chronic bronchitis, is usually indicated. Here both the tonic effects of climatic change, and the prevention of pulmonary complications, and fresh rheumatic attacks, must be taken into account. Mountain resorts of moderate height, well-protected from sudden changes of temperature, may be prescribed in summer.

In the neuroses of the heart, including (1) angina pectoris, (2) palpitation associated with chlorosis, hysteria, or hypochondriasis, and (3) exophthalmic goitre, moderately bracing climates are indicated. In angina pectoris long journeys involving fatigue or exertion must be rigorously forbidden, as must also sightseeing or exciting amusements.

**3. Of the Abdominal Organs.**—In the various forms of chronic dyspepsia and intestinal catarrh, in chronic hepatic disease, in chronic dysentery (after removal from a malarial district or tropical climate), in diabetes, and in chronic endometritis, pelvic cellulitis, and other diseases of the uterus and its surroundings, the following climates may be selected from:—Cannes, the Engadine, Hastings, Hyères, Lisbon, Malta, Mentone, Montpellier, Morocco, Naples, Nice, the Nile, the Bernese Oberland, Pontresina, the Pyrenees (in summer), Queenstown, Schwalbach, Seville, Spezia, St. Moritz, Valencia, and Ventnor.

In convalescence from acute nephritis, and in all forms of chronic Bright's disease, but especially *catarrhal* nephritis, warm dry climates are indicated. Among the best are Upper Egypt, the Riviera, the Cape of Good Hope (inland), Bombay; and, in England, Brighton, Folkestone, Hastings, and Ventnor.

In renal calculus, removal from particular districts in which stone is known to be prevalent may possibly be of use in some cases.

**4. Of the System at large.**—Change of climate is here nearly always indicated:—

(1) In convalescence from typhus and typhoid fevers, scarlet fever (at the end of the desquamative stage), measles, diphtheria, and acute rheumatism; also in the third stage of protracted whooping-cough.

(2) As a prophylactic against all infectious diseases, and especially cholera, yellow fever, hay fever, influenza, and malaria; also against rheumatism and phthisis by withdrawal from damp districts, and goitre and cretinism by removal from the *ensemble* of conditions to which the latter are due.

(3) In rickets, scrofula, chlorosis, general

anæmia, and functional debility. Here, where a pure air and a bracing sunny atmosphere are the chief indications, the climates enumerated in section 1 are suitable; as are also Algiers, Biarritz (in autumn), the Cape of Good Hope, Ischl, Malaga, Rome, Sicily, St. Moritz; and, in Great Britain, a number of inland and seaside places (Malvern, Scarborough, &c.) which it is unnecessary to mention.

EDWARD I. SPARKS. M. CHARTERIS.

**CLINICAL** (κλίνη, a bed).—This word literally signifies 'of or belonging to a bed'; but it has been especially applied to the practical study and teaching of disease at the bedside; and has more recently been extended to all that relates to the practical study of disease in the living subject generally.

**CLONIC** (κλονος, tumultuous movement). This word is applied to spasmodic movements which are of short duration, and alternate with periods of relaxation. See CONVULSIONS; and SPASM.

**CLONUS** (κλονος, tumultuous movement). The term now applied to the movement of a portion of a limb by striking or forcibly stretching one of its tendons. See SPINAL CORD, Diseases of; § 5. SPINAL REFLEXES.

**CLOT.**—A clot, or coagulum, is the product of the formation of fibrin (see BLOOD, Morbid Conditions of). Coagulation of the blood within the blood-vessels is described under THROMBOSIS; the coagulation of extravasated blood under HÆMORRHAGE, and BRAIN, Hæmorrhage into; and the coagulation of inflammatory exudations under INFLAMMATION.

**CLOTHING.**—See DISEASE, Causes of; and HEALTH, Personal.

**COAGULUM** (*coagulo*, I curdle).—See CLOT.

**COAL GAS, Poisoning by.**—Coal gas, so largely employed for illuminating purposes, is a compound containing—in addition to olefiant gas and analogous hydrocarbons, on which the luminosity principally depends—certain so-called diluents, which burn with a non-luminous flame, namely, hydrogen, marsh-gas, and carbonic oxide, along with what are termed impurities, of which the chief are carbonic acid, sulphuretted hydrogen, and bisulphide of carbon. On these impurities the characteristic odour mainly depends. This odour, which is perceptible even to the extent of 1 in 10,000, is a valuable safeguard against accidents from escape of gas. The recently introduced water gas, made by decomposing aqueous vapour over burning coke, while it is more dangerous—only 1 per cent. being necessary to produce a fatal result—has not even this safeguard, for it is quite odourless.

A mixture of coal gas with the air inhaled exerts a deleterious effect on the system, and proves fatal when it reaches a certain percentage. In addition to the danger from inhalation, fatal accidents frequently occur from the explosive nature of the compound which is formed when the gas reaches the proportion of 1 to 10 of the atmosphere. Much less than this, however (a non-explosive mixture, therefore), proves fatal if long inhaled.

It is difficult to determine the exact proportion of the gas present in atmospheres in which fatal accidents have occurred, but we derive important information on this point from experiments on animals. Many such have been made. M. Tourdes, who has carefully investigated the subject, finds that pure gas is almost instantaneously fatal;  $\frac{1}{4}$ th kills rabbits in five minutes, and dogs in twelve minutes;  $\frac{1}{3}$ th kills rabbits in from ten to fifteen minutes;  $\frac{1}{6}$ th still proves fatal after a longer period; and evident signs of distress are caused in rabbits by an atmosphere containing only  $\frac{1}{10}$ th of the gas. Dr. Taylor (*Edin. Med. Journ.*, July 1874) has estimated the proportion of gas existing in a room in which a fatal case occurred at three per cent.

**SOURCES OF POISONING.**—Poisoning by coal gas is only known of as an accident. Occasionally suddenly fatal consequences ensue among workmen from exposure to a sudden rush of undiluted gas from gasometers and mains. More commonly slowly fatal cases result from the gas tap in a bedroom being left open carelessly, from accidental extinction of the light, or from leakage of gas-pipes in a house or at a distance. The gas gains access to the house in the latter case through cellars, walls, and more especially by means of drains and sewer-pipes, most commonly during winter and severe frosts, presumably from the ground surface being frozen and preventing escape into the streets. After passing underground, coal gas loses its odour, which is such a safeguard, most of the carburetted hydrogen and marsh gas is absorbed, and the amount of carbonic oxide is increased.

**SYMPTOMS.**—Gas, even when in comparatively small proportion and just sufficient to cause an unpleasant odour, acts deleteriously if long breathed, and gives rise to headache and general depression of health. A very slight escape of coal gas—just sufficient to produce a close smell, not like that of gas, and therefore not recognisable as such—will cause relaxed or ulcerated throat, probably from the irritation of the sulphur compounds.

In severe and fatal cases the symptoms which have been noted are headache; nausea or vomiting; vertigo; and loss of consciousness, passing into deep coma and muscular prostration, which resembles the apoplectic state, the individual lying insensible and incapable of being roused, with livid features, stertorous breathing, and froth at the mouth.

Death usually occurs quietly, in this state of coma, but occasionally with convulsions.

The state of the pupils does not seem to be constant, though they are generally dilated before death.

In Dr. Taylor's case (*sup. cit.*) the teeth were firmly clenched, and the eyes were in a constant state of lateral oscillation.

**FATAL PERIOD.**—The fatal period of poisoning by coal gas is extremely variable, and a remittent character of the symptoms sometimes gives rise to fallacious hopes of recovery in cases which ultimately prove fatal.

**DIAGNOSIS.**—The smell of gas in the clothes, breath, and perspiration, which continues for a considerable time after removal from the polluted atmosphere, is the best indication of the cause of the coma.

**ANATOMICAL CHARACTERS.**—The smell of gas is often very marked. M. Tourdes found the blood as a rule dark, but in some recent cases it has been noted as being of a bright red colour. Other signs are: a bright coloration of the pulmonary tissue; froth in the air-passages; congestion of the mucous membrane at the base of the tongue, the mucous membrane of the trachea being in some cases a bright rose colour; engorgement of the cerebral and spinal venous system; and rose-coloured patches on the thighs.

**MODE OF ACTION.**—It is obviously impossible to differentiate between the effects of the various constituents of coal gas, but we have good reason for believing that the most active agent is the carbonic oxide, which exists in the proportion of from five per cent. (English gas) to twenty-five per cent. The active agent is the same in water gas, which contains forty-four per cent. of carbonic oxide.

The symptoms in the main agree with those caused by carbonic oxide (*see* CARBONIC OXIDE, Poisoning by), and the effects, therefore, would be chiefly due to the action of the carbonic oxide on hæmoglobin. The blood of one patient after sixty hours gave the spectrum of CO-hæmoglobin, and Grehaut calculates that 100 c.c. of blood from an animal poisoned with gas give 20 c.c. of CO.

**TREATMENT.**—Instant removal from the polluted atmosphere is the first thing to be attended to. Attempts must then be made to cause oxygenation of the blood, by artificial respiration and excitation of the respiratory centres, by reflex stimulation of the face, chest, &c. Pure oxygen gas may be administered. As, however, the compound which carbonic oxide makes with the blood colouring-matter is a very stable one, and not easily broken up by the introduction of atmospheric air or oxygen, it not infrequently happens that these measures prove of no avail. In such cases it would be highly advisable to perform venesection, and then transfuse fresh blood, a plan of treatment which has been found successful in poisoning by carbonic oxide.

D. FERRIER.

**COARCTATION** (*coarcto*, I straiten). A pressing together, narrowing, or stricture of any hollow tube, such as the aorta, intestine, or urethra.

**COCYGOODYNIA** (*κόκυξ*, the cuckoo; and *δδύνη*, pain).—**SYNON.**: Fr. *Coccygodynie*; Ger. *Steissbeinschmerz*.

**DESCRIPTION.**—This name has been given to a peculiarly severe and obstinate neuralgic pain in the neighbourhood of the coccyx, incited by such actions as defæcation, walking, sitting down, or rising from a recumbent position. It may arise from injury, such as fracture or luxation or horse exercise; or it may be the result of inflammatory affections of the sacro-coccygeal joint or of the pericoecygeal fibrous structures. Coccygodinia is also a concomitant symptom of many rectal, vulval, vaginal, and uterine disorders, being excited whenever any of the muscles which have their insertion in or near to the coccyx are brought into play. It not infrequently is a sequela to a difficult instrumental parturition, or may be a neurosal condition associated with gout or rheumatism.

**TREATMENT.**—In cases where the affection is primary, recourse must be had to rest in the lateral posture, anodyne suppositories of extract of belladonna and opium, and to the careful regulation of the bowels. If the pain be very severe, a local hypodermic injection of morphine may have to be administered. To be successful in our treatment we must, however, always endeavour to differentiate by means of a carefully conducted systematic examination the cause or causes which may be at work, and to attend to the proper indications. Tenotomy and excision of the coccyx have not proved to be uniformly successful, and should therefore only be had recourse to in specially selected cases. Attention must be paid particularly to the nervous system, and the general nutritive processes improved by the administration of arsenic, quinine, and iron.

JOHN HAROLD.

**COLD, A.**—A popular name for Catarrh. See CATARRH.

**COLD, Ætiology of.**—See DISEASE, Causes of.

**COLD, Effects of Severe or Extreme.**—**INTRODUCTORY.**—The general effect of exposure to severe or extreme cold is to lower, even to extinction, all vital activity. The blood-vessels, especially the smaller arteries and capillaries, after a brief period of congestion, become contracted, the latter to such an extent as no longer to permit the passage of the red corpuscles; the normal condition, composition, and structural integrity of the various tissues are more or less impaired, or altogether destroyed; the red corpuscles become first crenated and later disorganised and broken up, the watery elements of the blood thicken, and when

frozen become more or less separated from the saline and other constituents; and those processes of chemical and physiological change which are essential to every manifestation of life, being only possible within certain very narrow limits of temperature, are hindered or absolutely prevented.

The more *special effects* vary in degree and kind:—1st, with the degree of cold, the duration of the exposure, and the medium or manner of application; 2nd, with the part, and extent of surface exposed; and, 3rd, with the general constitution and physiological condition of the sufferer.

Moderate cold, acting during a short time, or even severe cold during a still shorter time, followed by the glow of speedy reaction, exercises a tonic and stimulating influence. If, however, the cold is very severe, or the exposure too long, the glow of reaction does not occur, but a sense of depression is experienced, from which, at best, recovery takes place but slowly. Continued exposure to such a degree of cold as is yet not incompatible with the maintenance of life, nevertheless keeps at low ebb activity of nutrition and function alike. Extreme cold and long exposure lead to congelation and consolidation of the various tissues of the body. After complete congelation of the body recovery is impossible.

Dry cold is much less readily injurious in its influence than cold associated with wet. The better conductor of heat the medium is, the more speedily and completely does it reduce the temperature of the part with which it is in contact. Immersion in water cools more rapidly than exposure to air of the same temperature; and contact with wool, wood, or metal, of the same degree of coldness, excites in each case a different sensation, and leads to a different result, or to similar result but with very different rapidity. Constant renewal of the medium in contact hastens the cooling effect; and a continuous draught of only moderately cold air may do more to chill than temporary exposure to an intensely cold but still atmosphere. If some external part, and a comparatively small extent of surface only, be acted upon, the effect may be simply local, and the general disturbance of the system scarcely appreciable. But if the whole body, or a considerable extent of surface, or any important internal organs be acted upon, a proportionately serious general effect is produced.

The young (infants especially) and the aged alike ill sustain exposure to cold, and are most liable to suffer, not only from its direct effects, but also from the various maladies to which it gives rise. The feeble, ill-nourished, and broken in health, especially the subjects of organic disease, or of degeneration due to habitual intemperance, readily succumb, or only slowly and imperfectly recover. Among the healthy and otherwise vigorous, hunger, fatigue, sleep, anxiety of mind, fear, and

mental depression of whatever kind, lower—too often even to fatal issue—the power of resistance to the deadly influence of cold.

**LOCAL EFFECTS.**—For local effects of exposure to cold, see CHILBLAIN; FROST-BITE; and GANGRENE.

**GENERAL EFFECTS.**—The general effects and symptoms produced by exposure to severe or extreme cold vary in different cases. Temporary exposure produces, first, a sense of coldness or chilliness, associated with paleness and corrugation of the skin (the so-called *cutis anserina*), then shivering and tingling sensations, followed by numbness and diminution of muscular activity and power. Healthy reaction restores more or less quickly the normal condition. Prolonged exposure to extreme cold gives rise to a series of symptoms, graphically described by Beaupré somewhat as follows:—Reaction has a limit, and a moment arrives when the powers are exhausted. Shiverings, puckerings, paleness and coldness of the skin, livid spots, muscular flutterings, are symptoms of the shock given to the vital forces; syncope approaches; the stiff muscles contract irregularly; the body bends and shrinks; the limbs are half-bent; lassitude and languor invite to repose; a feeling of weight and numbness retards the steps; the knees bend; the sufferer sinks down or falls; the propensity to sleep becomes irresistible; everything grows strange; the senses are confused; the mind grows dull, the ideas incoherent, and the speech stammering or raving; respiration, at first interrupted, becomes slow; the heart's action is feeble, quick, hard, irregular, and sometimes painful, and the pulse progressively smaller; the pupils dilate; the brain becomes stupefied; and finally deep coma indicates the approach of inevitable death.

Other and somewhat different effects and symptoms, attributable to differences of circumstances and condition, have been from time to time observed. Distressing and almost intolerable thirst, with loss of appetite for food, is often experienced; and the attempt to obtain relief, by sucking snow or ice, only adds to the suffering. Somnolence is by no means so constant an effect as is commonly supposed—at any rate, in the earlier stages, and less extreme cases. On the other hand, inability to sleep has proved a common cause of suffering and consequent loss of strength. The manifestations of brain-disturbance due to exposure to cold, varying as they do from dulness, incoherence, wandering, and thickness of speech, to even raving delirium, are especially worthy of note, inasmuch as they resemble, and are liable to be mistaken for, the effects of alcoholic intoxication.

Death from the direct and immediate effects of cold is rare in the British Islands; but it is estimated that in the Russian Empire, on an average, 694 deaths occur annually

from this cause. The length of time during which exposure can be sustained varies greatly with the condition of the individual and with surrounding circumstances, as well as with the degree of cold. Under ordinary circumstances, an hour's exposure to intense cold, without due protection against the loss of natural heat, often suffices to determine a fatal result. At the same time, well-authenticated cases are on record, in which persons buried, for days even, in snow, have nevertheless survived and ultimately recovered with little permanent damage.

**MODE OF DEATH.**—The immediate cause or mode of death occurring directly from exposure to cold seems in some cases to be principally shock; in some, syncope; in some, apoplexy; in others, asphyxia; and in others, again, coma. In most cases it is probable that these several conditions, with others less readily specified, combine to produce the fatal result.

**ANATOMICAL CHARACTERS.**—The appearances presented on *post-mortem* examination are somewhat differently described and estimated by different observers; but none of them are absolutely pathognomonic, and some are as likely to be produced by exposure of the body after death as during the process of extinction of life. Among the more noteworthy are the following:—strong cadaveric rigidity; paleness or waxy whiteness of skin, with patches of more or less bright redness about the face, neck, and limbs, especially on exposed or prominent parts; a contracted and shrunken condition of the male genital organs; comparative bloodlessness of superficial and external parts; accumulation of blood in and about the thoracic and abdominal viscera; great distension of all the cavities of the heart, with more or less clotted and often bright-coloured blood; the blood in other parts also sometimes of brighter colour than usually seen on *post-mortem* inspection; hyperæmia and congestion of the lungs; hyperæmia of the brain, overfulness of the sinuses, and excess of serous fluid in the ventricles, and at the base, in some cases; in others, comparative bloodlessness of the surface of the brain, and no distension of the sinuses; excessive fulness of the urinary bladder; and, lastly, separation of the cranial bones along the coronal and sagittal sutures. The lines of reddish or brownish staining along the course of the superficial blood-vessels, relied on by some as pathognomonic, are certainly not so, inasmuch as they depend upon exosmosis of the blood colouring-matter set free by disruption of the corpuscles, which may be effected by freezing after death, as well as before.

**TREATMENT.**—The treatment of sufferers from the effects of cold consists in the restoration of warmth, and the rekindling of those processes by which the natural heat of the body is maintained. But this must be done

gradually, and with great care. As in the treatment of a frost-bitten part, so in the treatment of the body generally—all sudden or rapid elevation of temperature must be avoided. The sufferer, divested of the clothing previously worn, and wrapped in blankets, should be placed in the recumbent position in a room, the air of which is dry, still, and cold, but capable of being gradually warmed. Gentle but continuous friction should be made over the trunk and limbs, care being taken that rigid or frozen parts be not damaged by rough manipulation. At first, ice or ice-cold water may be used; afterwards dry rubbing with flannel or with the hands is better; later still some stimulating liniment may be employed. Some recommend immersion of the body in a bath of cold—at first ice-cold—water, the temperature of which can be gradually raised. This method would seem easy and advantageous, if means are at command. When the sufferer can swallow, warm, gently stimulating drinks—as tea, coffee, aromatic infusions, beef-tea, or soup—may be given, at first without, but later with some wine or spirit. Alcohol, though useless or injurious if taken to fortify against cold, is useful and beneficial when judiciously administered as a restorative after exposure. In all cases of insensibility, and even apparent death from cold, every effort must be made to restore animation; and the attempt must be persevered in for a considerable time before being given up as hopeless. It is often difficult, sometimes impossible, to judge whether life is absolutely extinct or not. And while, on the one hand, it is important that the temperature be not raised too quickly, lest reaction should be too strong or dangerously irregular; on the other hand, it is equally if not more important that the needful measures be adopted without delay, and carried out not too slowly, lest the chance of revival should be lost. In the less severe cases, restoration of warmth may be comparatively quickly accomplished. The state of the bladder should always be examined, and relief afforded, if needful, by aid of the catheter. Attention to the general health is often requisite for long after recovery from the more immediate effects of exposure has taken place. Rest, good nourishment, and tonics are indicated.

**Cold as a Cause of Disease.**—As a predisposing and exciting cause of disease, cold proves, in the British Islands, year by year, more fatal in its effects probably than any other single condition or influence. Any considerable fall in the thermometer below the average standard during the colder months of the year is constantly followed by a corresponding rise in the death-rate, and an increase in still greater proportion in the amount and extent of sickness and suffering. The Reports of the Registrar-General clearly prove this, so far as the death-rate is concerned. A

striking instance may be quoted. In the week ending December 19, 1863, in the London district, 1,291 deaths were registered. Severe frost set in, and in the week ending January 9, 1864, the number rose to 1,798. The week following, ending January 16, no fewer than 2,427 deaths were registered. This enormous increase could be attributed to no other cause than the effects of the severe cold which prevailed. The Registrar-General also shows that after the age of from twenty to forty the mortality from cold increases in something like a definite ratio with increasing years.

General depression of the vital powers, congestion and functional derangement of various internal organs—the lungs, liver, and kidneys—catarrhal and other forms of inflammation of the mucous membranes, especially of the respiratory tract, but also of the intestinal canal and bladder, paralysis from central or peripheral lesion, together with rheumatism, chilblain, frost-bite, and gangrene, constitute the list of maladies most commonly caused and fostered by exposure to the influence of cold.

ARTHUR E. DURHAM.

**COLD, Therapeutics of.**—The therapeutic uses of cold are various and extensive. Cold may be applied as moist cold, by means of wet compresses and cold lotions or baths; and it may also be used as dry cold in the form of ice enclosed in a receptacle of metal or india-rubber, or as a current of iced water circulating through a soft metal coil, as in Leiter's tubes. The flat coil of tube can be pressed to fit any part of the body. Each of these methods has its special advantages and adaptations. Furthermore, cold may be made use of by the mouth, and by injection into the mucous canals of the body.

**GENERAL PRINCIPLES.**—The general effects of cold, however applied, are to lower temperature, to diminish sensibility and fluidity, to contract the tissues and vessels, and so to reduce the volume of parts. The cold bath and cold sponging alike have the effect of lowering the temperature of the body. The fall of temperature sometimes is but transient, reaction setting in and heat of surface returning when the body is withdrawn from the cooling medium. At other times the temperature continues to fall after the individual is removed from the bath. If the action of the cold bath be prolonged, then a thermometer, introduced within the rectum, shows a great depression of temperature, and much pain is experienced, similar to the severe pain which is felt in the hand and arm when the former is held for some time in water at a temperature of 41° F., and which soon compels the withdrawal of the hand from the vessel. Cold baths and their uses are treated of in another article (*see BATHS*), but attention may here be drawn to

the practice of cold sponging over the surface as an efficacious means of lowering preternatural heat and relieving acridity and dryness of the skin during fever. The addition of enough solution of potassium permanganate to give a slight purple tinge to the water, makes it especially refreshing to the patient. Reduction of hyperpyrexia by means of cold is now a well-established therapeutic method. See FEVER; and TEMPERATURE.

**METHODS OF APPLICATION AND USES.**—There are several methods of applying cold therapeutically:—

**1. Cold Affusion and Wet Packing.** In the practice of cold affusion, introduced by Dr. Currie, in 1797, the patient is unclothed, seated in a tub, and four or five gallons of cold water thrown over him. Thus, Dr. Currie said, a commencing fever might be 'extinguished.' In cases where the skin was burning hot and dry, it was observed that after the cold affusion temperature fell, perspiration broke out, and the patient usually dropped into a refreshing sleep. If the body-heat did not exceed 98.4° F., or if the patient was damp and chilly, with or without delirium, the cold affusion was regarded as dangerous and by all means to be avoided. Drs. Strauss and Hirtz speak most highly of the marvellous effect of cold affusion in cases of collapse during fever. This kind of collapse appears due to a paralysis of the nervous centres: the heart's action fails, as does also the respiration; but while the surface of the body is cold, the temperature, taken in the rectum, still remains abnormally high. The affusion is applied by pouring a pitcherful of water, at a temperature of, or a little above, 10° C. (50° F.), over the patient seated in a bath or on a waterproof cloth. The patient, plunged in stupor, is suddenly roused by the shock; he draws a long breath; the respiration becomes fuller; the cardiac ataxy ceases, and the pulse, which, traced by the sphygmograph during the collapse, presented a scarcely broken horizontal line, now regains its normal line of ascension; heat of surface returns, and the temperature in the rectum falls. The action of the cold affusion, as thus applied, is to excite immediate and energetic reflex action.

In many nervous affections, such as maniacal delirium, chorea, and hysteria, cold douches, shower baths, and affusions are valuable as restorative and curative agents. In the convulsions of robust children, a stream of cold water directed over the head from a height of two or three feet often has a speedily beneficial effect. Cold affusion has been tried in tetanus, but it has in some instances killed the patient (Elliotson).

Other cases in which cold affusions are of service are those where respiration fails, and it is necessary to appeal powerfully to the reflex excitability of the nervous centres. To resuscitate those who are in danger of death

from a narcotic such as chloroform or opium, slapping the patient severely with a cold wet towel is an efficient method. In sunstroke, cold affusion over the head and neck may be resorted to, provided the skin be not cold and clammy, and the patient in a synopal state. In conditions of nervous spasm—of the larynx, for example—cold douches over the neck may prove useful. Cold affusion to the feet was much commended by Cullen as a means of promoting action of the bowels in cases of obstinate constipation. Spasmodic retention of urine has been relieved by a cold douche over the perineum and thighs (Currie); and Mr. Erichsen mentions the case of an old man who found his power of micturition increased by sitting on the cold marble top of his commode. In cases of extreme debility, with damp, cool skin, low muttering delirium, and very feeble pulse, cold affusions are dangerous.

In some cases of fever, where for any cause a cold bath is objectionable, the patient may be wrapped in a wet sheet and then covered with a few blankets. The sheet as it becomes heated may be changed for one fresh and cold, or very cold water may be squeezed from a sponge over the sheet as the patient lies rolled up in it on a waterproof cloth. Allowing fragments of ice slowly to melt on the sheet which covers the patient is, in the author's experience, a successful way of treating the hyperpyrexia of acute rheumatism. In scarlet fever of malignant type, where the rash does not readily appear, this form of cooling pack has been found most valuable. The late Dr. Hillier and Dr. Gee have both added their testimony to its utility. In Dr. Gee's cases the patient remained packed in the wet sheet for one hour and was then removed to bed. The cases best suited for treatment by the wet pack are those where the skin is very dry and hot, and the patient exceedingly restless and delirious.

**2. Cold Compresses, Ice-Bags, Irrigations, Lotions, and Injections.**—Cold may be continuously applied with a view to abating undue heat of a part of the body. Iced-water rags or compresses may be placed over an inflamed throat, or on the head in inflammation of the brain. A narrow bladder of ice laid over each carotid artery in the neck is an excellent way of cooling the blood as it flows to the brain. In acute pneumonia Niemeyer has commended strongly the use of cloths dipped in cold water, well wrung, and then applied so as to cover the chest, and especially the affected side. These compresses are repeated every five minutes. Pain and dyspnoea are much relieved; sometimes the temperature falls an entire degree; and if the cold appliances do not arrest the actual attack of pneumonia, they may shorten its duration and promote convalescence. The necessity of so often having to change the compress, and thus disturb the patient,

is a great objection to this mode of applying cold. Dr. Lees, in the *Lancet*, Nov. 2, 1889, records several instances of arrest of the pyrexial process in pneumonia by means of a bag of ice applied over the inflamed lung. The development of broncho-pneumonia in children may be cut short by the ice-bag. A frozen compress over the cæcum is useful to stay peristalsis and check hæmorrhage in typhoid fever.

A powerful sedative and antiphlogistic effect of cold can be obtained by *irrigation*; i.e. allowing cold water to fall drop by drop on a cloth, so as to keep it continually wet with fresh supplies of water. This may be done by suspending over the part to be irrigated a bottle of water, in which a few pieces of ice may be put; one end of a skein of cotton, well wetted, is then allowed to hang in the water, while the other end is brought over the side of the bottle. This, acting as a syphon, causes a continual dropping upon the part to be irrigated. In injuries of joints, where it is of much consequence to check inflammation, this process, which abstracts heat gradually and without disturbance of the part, is most valuable. Irrigation of the shaven scalp in cases of meningitis is a very powerful cooling and sedative appliance, requiring care and watchfulness. A cap of india-rubber or coil of tubing over the head and back of neck, so arranged that a current of cooled water may flow continuously through it, will act as a general reducer of temperature. Where pounded ice is applied to the head in a bladder, this should be suspended by a string from the bedstead, so that the head of the patient may not have to sustain the weight of the bag and its contents. Five ounces of sal ammoniac and five ounces of nitre in a pint of water will form a frigorific mixture, which can be applied in a bladder when ice is not at hand. The ice-bag, and cold-water compresses renewed every three minutes, have been used as an appliance to strangulated hernia, and to prolapsed rectum, to reduce the volume of the part and so facilitate reduction. Care must be taken that the cold application be not continued so long as to cause gangrene. Cold wet compresses should not be applied over dry bandages, with which wounded or broken parts are secured. Several cases are recorded where a hand or arm has become gangrenous, in consequence of having been bound up with dry bandages, and then treated with cold-water compresses. The dry bandages, as they become wet, contract tightly on the limb and stop the circulation.

A mixture made of spirit of wine and water, or of eau de Cologne and water, is a simple form of *cooling lotion*. The spirit evaporates and so carries off heat from the surface. 1 fl. oz. of rectified spirit to 15 of water makes a good spirit lotion, and the addition of 4 drachms of nitrate of potassium, or

chloride of ammonium, will add to its cooling and sedative effect. 4 drachms of the chloride of ammonium with half a fluid ounce of diluted acetic acid, and the same quantity of rectified spirit, in 15 fl. oz. of camphor water, is another form for a very serviceable lotion. These lotions, applied by means of a piece of soft rag or lint over the skin, act as refrigerants, cooling the head when it is hot or painful; reducing heat and arterial excitement in tumours or contusions; and tending in the latter to promote the absorption of effused blood. A lotion of 1 part alcohol and 3 parts camphor water, at a temperature of 75° to 85° F., was highly praised by Scudamore in cases of gout. Cold water, and cold lotions of vinegar and water, are familiar means for stopping hæmorrhage.

In cases of severe uterine hæmorrhage, *injections* of ice-cold water into the vagina, or into the rectum, frequently succeed in checking the bleeding. If the cold injection fail, hot water (110°-120° F.) may be tried in its place. In cases of bleeding internal piles, an injection of cold water, after the action of the bowels, braces the parts and constricts the bleeding vessels. Iced water has been used as antipyretic enemata.

3. *Dry Cold. Uses of Ice.*—Heat may be continuously abstracted from an inflamed part in a safe way, and without undue risk, by applying dry cold by means of a waterproof bag of vulcanised india-rubber filled with ice, snow, or a freezing mixture made of equal parts of salt, nitrate of potassium, and chloride of ammonium. Moisture from the air will condense on the exterior of the cold bag, but a piece of lint interposed will protect the skin from damp. The india-rubber is a bad conductor, and too great abstraction of heat need not be feared. The walls of an animal bladder conduct heat much better than the india-rubber, and it is necessary to watch carefully over the application of ice-bladders, for when continuously applied they have been known to cause severe frost-bite of the part. If, when cold is being applied, the patient persists in complaining of severe pain, it is right to examine carefully and see how the part is affected by the cold. Professor Esmarch, in cases of fracture, and in various forms of traumatic inflammation, has applied ice for periods of twenty or thirty days with the best results. In cases of commencing disease of the vertebræ this surgeon has used cold water placed in a tin vessel, so made as to adapt itself to the part to be treated. Minor cases of bruise with inflammation may be treated by cold employed in the form of a common bottle filled with cold water and kept pressed against the part. After operations upon the eye, the extraction of cataract for example, a small ice-bag is very useful in relieving pain and keeping down inflammation. For the same purpose an ice-

bag is sometimes applied to the chest after the operation of paracentesis.

Ice-bags placed along the course of the spine have been found effectual remedies in many forms of nervous disorder. In cases of epilepsy, where the circulation is sluggish, the hands and feet being always clammy and cold, an india-rubber bag of ice applied along the spine has been found to restore warmth, at the same time relieving headache and symptoms of incipient paralysis. Cold to the spine is asserted by Dr. Chapman to lessen the excito-motor power of the cord. In the severe pain of an inflamed ovary or testicle ice in a bag may often be employed beneficially as an anodyne. The long-continued application of bags of ice to the chest, for the arrest of hæmorrhage from the lungs, has been observed by Sir Richard Quain and others to be very apt to cause bronchitis.

Lumps of ice swallowed are invaluable in arresting hæmorrhage from the throat and stomach. In tonsillitis and diphtheria this same treatment tends to reduce inflammation and cool the throat of the patient. Obstinate vomiting can often be checked by swallowing fragments of ice.

**4. Cold as an Anæsthetic.**—Dr. James Arnott, in 1849, brought forward the use of a freezing mixture of ice and salt as a means of producing local anæsthesia, by freezing the part to which the mixture was applied either in a bag or in a metallic spoon. For small superficial operations this method of anæsthesia by congelation answers very well. The part becomes white and hardened to the cut of the surgeon's knife, there is very little hæmorrhage, and the wound made usually heals well by primary adhesion.

Subsequently Dr. Richardson indicated a very convenient way of inducing local anæsthesia, by the volatilisation of ether in the form of spray, by means of the hand-ball spray atomiser. Ether sprayed on the bulb of a thermometer, held about an inch from the jet, brought down the mercury to within 10° of zero F. When the jet was turned on to the skin, a marked degree of local anæsthesia was produced, but not enough for surgical purposes. By driving over the ether under atmospheric pressure, instead of trusting simply to capillary action—or to suction, as in Siegle's apparatus—one may bring the thermometer to 4° below zero within thirty seconds. By the use of this apparatus, at any season or temperature, the surgeon can produce cold even 6° below zero; and by directing the spray upon a half-inch test-tube containing water, he can produce a column of ice in two minutes. For local anæsthesia by cold, the ether spray answers well. Such operations as the removal of small tumours, opening abscesses, and inserting sutures may be painlessly performed.

JOHN C. THOROWGOOD.

**COLIC** (κόλον, the large intestine).—Originally colic signified a painful affection due to spasm of the bowel, but, though still retaining this application, it has now come to be further associated with other complaints which are attended with severe pain of a spasmodic character, a qualifying adjective indicating the nature and seat of each particular form. Thus *renal colic* is applied to the group of symptoms due to the passage of a stone from the kidney to the bladder; *hepatic colic* to those accompanying the escape of a gall-stone. See COLIC, Intestinal.

**COLIC, INTESTINAL.**—SYNON.: Fr. *Colique*; Ger. *Die Kolik*.

**DEFINITION.**—Painful and irregular contraction of the muscular fibres of the intestines, without fever.

**ÆTIOLOGY.**—*Predisposing causes.*—These include the nervous (as hysteria and hypochondriasis), lymphatic, and bilious temperaments; sedentary occupations; the female sex; and the period of youth or adult age. *Exciting causes.*—These may be grouped as follows:—

1. Irritation—from lodgment of gas due to fermentation of undigested food, and decomposition of feces long retained within the large intestine; from feces, or intestinal concretions, undigested or partly digested food, such as pork, shell-fish, salt meats, unripe fruit, or septic game; from cold drinks or ices; from excessive or morbid secretions, especially bile; from gall-stones; or from worms—a bundle of round-worms or coiled up tape-worms. 2. Morbid states of the bowel, including obstruction from intussusception, twisting, strangulation, &c.; ulceration (typhoid, tubercular, dysenteric); inflammation (enteritis, typhlitis, &c.) 3. Reflex nervous disturbance, due to anxiety, fright, anger, or other emotional disorder; to disease of the ovaries or uterus; to calculus (hepatic or renal); to dentition; or to exposure, especially of the feet and abdomen, to cold. 4. Blood-poisoning, as from lead, copper, gout, rheumatism.

**SYMPTOMS.**—The characteristic or essential symptom of intestinal colic is pain in the abdomen, without febrile disturbance. It usually begins, and is most severe, in the umbilical region, then spreads to other parts, or to the whole abdomen, and is apt to travel from one part to another. It is almost always relieved by firm pressure, and by expulsion of flatus; and is paroxysmal in character, remitting, or exacerbating, or completely subsiding at intervals. The suffering is usually severe, often agonising, and to relieve it the patient bends forwards, pressing the abdomen firmly with his hands or against some hard surface, or rolls about. As a rule the abdomen is distended by flatus; in lead-colic it is, however, firmly retracted towards the spine, and the movements of the inflated intestines affected by spasmodic contraction,

producing loud borborygmi, may frequently be seen and may be felt by the hand applied to the abdomen. The muscles of the abdominal wall and the bladder usually participate in the internal spasm: the abdomen becomes rigid or knotted, and the recti muscles become contracted into round balls, while frequently the navel is retracted; micturition is frequent or suppressed. Usually there is constipation, and the pain disappears when the bowels are freely relieved; sometimes, however, it persists for a time. Constipation stands in a twofold relation to colic, either as cause or as effect of the spasm.

The countenance expresses great suffering, anxiety, and depression, and the features may be pinched. The surface of the body is cold, especially the feet, and the pale skin is covered by a cold perspiration. The pulse is commonly of normal frequency, or is infrequent and feeble.

The symptoms vary somewhat with the cause. When due to irritating ingesta, the prominent symptoms are vomiting and diarrhoea, sometimes ending in catarrhal dysentery. In children the legs are drawn up upon the abdomen; the bowels are often at first confined; and the evacuations are greenish, offensive, and very acid, afterwards becoming loose.

In nervous and hypochondriacal subjects, and especially females, severe pain in the intestines, resembling that of spasmodic colic, is apt to occur (*see* ENTERALGIA). Flatulence plays a varying part in different cases; it is often a prominent symptom, and the form of colic thus characterised has been termed *Colica flatulenta*.

Vomiting is generally in proportion to the severity of spasm and the degree of intestinal obstruction.

**DURATION.**—The duration of the attack varies greatly, from a few minutes to several days. The spasm usually ceases abruptly, leaving a feeling of soreness in the abdomen, while there is enjoyable relief from suffering. Colic, when violent or intractable, may terminate in enteritis, in peritonitis, and—especially with children—in intussusception.

**DIAGNOSIS.**—A pain moving from place to place, relieved by firm pressure and unattended by fever, separates colic from other affections, more particularly from those due to inflammation, in which pain is always aggravated by pressure.

Distension with spasm of the stomach may be distinguished from a similar condition of the colon, by the pain occupying a higher position in the abdomen (at or around the ensiform cartilage instead of—as in colic—the umbilical or hypochondriac regions), and by the percussion note elicited being deeper-toned and more prolonged than that which is produced by a distended colon. Besides, in colic the spasmodic contraction of the colon, producing borborygmi, may be traced by the

hand, or may be even seen, and there is tenesmus.

It should be borne in mind that enteritis or peritonitis may follow colic, when the pulse may become frequent, hard, and small, or frequent and soft, the temperature increased, and the seat of inflammation becomes tender.

**PROGNOSIS.**—Colic almost always ends in recovery, preceded by free evacuation of the bowels. Unfavourable signs are those arising from inflammation or intestinal obstruction.

**TREATMENT.**—In the first place the etiological indications should be met. The irritating contents of the bowels should be dislodged by purgatives combined with sedatives, such as calomel (five or eight grains) or rhubarb (twenty grains), with opium (one grain), followed by repeated doses of some saline aperient, such as sulphate of magnesium or potassium, with tincture of belladonna, henbane or opium, and spirit of chloroform, until free action of the bowels is obtained.

A suppository containing half a grain each of hydrochlorate of morphine and extract of belladonna, or a subcutaneous injection of morphine, may secure immediate relief from pain before aperients have time to act. Large warm enemata often relieve quickly. Other suitable measures are—the warm bath, friction with warm oil or stimulating liniments, hot-water fomentations, steamed flannels, mustard or turpentine stupes, flannel bags containing hot chamomile flowers or heated sand, the stomach-warmer filled with hot water, and large linseed and mustard poultices. The diet should be liquid.

In the prophylactic treatment the diet should be strictly regulated; lodgment of irritating solids and gases within the bowels should be prevented (*see* CONSTIPATION; FLATULENCE; and STOOLS); and the abdomen and feet should be kept warm by a flannel roller or belt and thick woollen stockings, or by a foot warmer. GEORGE OLIVER.

**COLLAPSE.**—**DEFINITION.**—Collapse is a condition of nervous prostration. When it is extreme, the vital nervous functions are in a state of partial, sometimes nearly complete, abeyance. It may terminate in death, or be followed by gradual reaction and complete recovery.

Collapse and shock have usually been classed together, but it is not accurate to do so. It is true that the ganglionic centres of the medulla oblongata are more or less profoundly involved in both, and that the two conditions possess many symptoms in common, dependent upon the derangement of function of one or more of these centres. Some confusion is attributable to the fact that shock is a term applied not only to a state or morbid condition, but to the cause which most frequently produces that condition—a violent impression or 'shock' to the nervous centres (*see* SHOCK). Collapse arises from many

different causes, shock being one, of which collapse may be regarded as a final and extreme degree, and into which it often imperceptibly passes. Collapse may occur under conditions where there has been no antecedent state of shock. Collapse presupposes previous nervous exhaustion, while shock may instantly appear in a healthy individual.

**ÆTIOLOGY.**—Any severe injury, especially if attended by profuse and sudden hæmorrhage, may terminate in collapse, such, for instance, as the rupture of one of the abdominal viscera, a penetrating wound of the chest or abdomen, or a wound of the heart or of a large artery. Extensive burns or scalds frequently give rise to typical collapse; and severe and prolonged pain is capable of causing it. Rupture of the heart or of an internal aneurysm, if the patient do not immediately die, causes extreme collapse. Certain poisons, as tobacco and arsenic, will also produce this condition. It is an advanced stage of some diseases, as, for instance, of Asiatic cholera; severe drastic purgation also, or prolonged vomiting, from whatever cause, may occasion it. The pernicious malarious fevers and yellow fever often end in collapse. In acute yellow atrophy of the liver symptoms of severe nervous disturbance, resulting in a species of collapse, sometimes suddenly supervene. Pyæmia, septicæmia, prolonged narcosis, frequently terminate in collapse.

**SYMPTOMS.**—The severity of collapse depends on the nature of the cause, and on the age and physical and mental vigour of the individual. It may vary from a moderate to an extreme degree. A moderate amount is seen in the course of fevers and some other diseases, but this neither modifies the progress of the malady nor attracts the notice of the patient, causing chiefly peripheral coldness. From this trifling amount collapse may pass to the most intense form, where the patient scarcely differs in outward semblance from a dead body. A superficial inspection will fail to detect the existence of the functions of respiration and circulation. Vitality may be said to have reached its minimum. In well-marked collapse from severe injury or loss of blood, the pulse at the wrist may be almost or wholly imperceptible; the heart's action scarcely audible, or very rapid, fluttering, and irregular; the surface of the body, the face especially, is deadly pale and cold, and the skin moist with clammy sweat; the respirations are very feeble, slow, and irregular, accompanied by sighing or gasping inspirations at intervals; the expression and character of the face are lost; the features are sunken and relaxed; the eyes are dull, glassy, staring, or languidly rolling about, and the conjunctivæ perhaps insensible to the touch; the nostrils are dilated; the sensibility of the whole body is diminished; and the muscular debility is

extreme. The patient lies on his back, without a trace of voluntary effort. If a limb is lifted it falls back again as if dead. Yet the consciousness and senses may in many cases be almost unimpaired; if roused by repeated questions, the sufferer will with visible effort make a coherent though, probably, inadequate reply.

If relief be not given, the respirations may become slower and slower till each one appears the last, when a sudden sigh shows that life is still present; finally the pulse and heart's action become more and more faint, and death results from pure asthenia. A condition closely resembling traumatic collapse is often witnessed in the last stage of cholera, when in an extreme degree the patient almost resembles a corpse save for the convulsive movements induced from time to time by the painful cramps. The surface is pale or bluish and covered with profuse sweat, but is at the same time cold to the touch. The hands and extremities are icy cold; the tongue is cold, and so also is the breath, which is gasping and paroxysmal; no pulse can be felt at the wrist; the eyes and features are sunken; the mind is apathetic, but nevertheless the consciousness may be perfect, and the patient able to respond to questions with a strong voice. Severe purging and tobacco-poisoning produce a condition very like that described as traumatic collapse.

Some cases of malignant fever terminate in collapse, which is characterised by extreme anxiety, by pallor or lividity of the face and surface, coldness of the skin, sweating, and a small, frequent, and irregular pulse.

A decrease of animal heat, especially in the external parts, is characteristic of collapse. The temperature of the internal organs varies; and there may be collapse with high internal temperature. This occurs in cholera, intense fevers, and some forms of septicæmia; or the converse may obtain, as is frequently witnessed in the collapse of dissolution. Collapse from loss of blood differs from syncope, although the latter may be described as an acute and transient form of collapse. In syncope the prominent symptom is loss of consciousness, which in collapse may be almost or quite perfect.

For the **PATHOLOGY** and **TREATMENT** of collapse, see **SHOCK**.

WILLIAM MAC CORMAC.

**COLLAPSE, Pulmonary.**—A condition in which the lung is simply more or less devoid of air. See **LUNG**, Collapse of.

**COLLIQUATIVE** (*colliqueo*, I melt). A term which originated in the belief that in certain conditions the solid parts of the body melted away, and were carried off as liquid discharges. The word is now generally applied to the copious sweats and diarrhœa which occur in certain wasting diseases, such as phthisis.

**COLLOID** (κόλλα, glue; and εἶδος, like). A peculiar morbid product resembling in its characters glue or jelly, and found associated with cancer and other forms of new-growth. See CANCER; and DEGENERATION.

**COLON, Diseases of.**—**SYNON.**: Fr. *Maladies du Colon*; Ger. *Dickdarmkrankheiten*.—The colon participates to a varying extent in the lesions and derangements of dysentery, typhoid fever, enteritis, peritonitis, and other affections. The special disorders to which it is most liable are intimately connected with its anatomical and physiological peculiarities. The colon is a distensible membranous tube, of large capacity, with chiefly solid contents, which are propelled slowly onwards by the muscular contractions of the walls. The moving force and the resistance offered to it are often too finely balanced, so that whenever the energy of the former is somewhat reduced, an accumulation of excretory products is apt to be determined. Hence arise retention of feces and gases, constipation, and consecutive evils, such as colic, colo-enteritis, or ulceration of the colon.

**I. Atony and Dilatation.**—**DEFINITION.** Loss of contractility of the walls of the colon, leading to accumulations, dilatation, and other sequelæ.

**ÆTIOLOGY.**—The causes of torpor or atony of the colon are mainly those of constipation—sedentary, indolent, and luxurious habits, over-stimulation of the bowels by coarse foods, purgatives, and enemata, a sluggish and lymphatic temperament, old age, and general debility or exhaustion, as after a long and tedious illness (see CONSTIPATION). Whenever the walls of the colon are distended by solid or gaseous accumulations, the contractile power is apt to be enfeebled, leading to further retention, loss of tone, and dilatation.

Atony of the colon is an important element in the pathology of constipation. The colon is apt to become greatly dilated throughout, or mainly so in certain portions, such as the cæcum and sigmoid flexure, behind some cause of obstruction—stricture, misplacement, elongated meso-colon, lodgment of feces, &c.; but instances of even excessive dilatation are recorded without the *post-mortem* discovery of a mechanical cause. When, however, dilatation results from obstruction, there will be found hypertrophy of the muscular coat of the bowel. Tympanitic distension of the colon from paralysis of the sympathetic occurs in peritonitis and in certain fevers, such as typhoid.

**SYMPTOMS.**—Torpor of the colon may be indicated only by constipation. There are usually the ordinary signs of retention of flatus or feces. In hysteria, and in inflammation of the bowels or peritoneum, flatus is apt to accumulate rapidly, and to produce great distension of the colon. Fæces

may collect and form large tumours in any part of the large intestines, but especially in the cæcum and sigmoid flexure. See STROOLS.

Fæcal and gaseous accumulations in the colon resulting from atony may produce the following effects, directly or remotely connected with them:—

(a) *Local.*—1. Colic. 2. Inflammation of the walls of the colon, or of the mucous and submucous coats, and ulceration. 3. Disturbances from pressure. Thus flatulent distension and fæcal accumulation encroaching on the cavity of the thorax, and impeding the descent of the diaphragm, may cause feeble respiration, dyspnoea or short and rapid respiration, palpitation and irregular action of the heart or upward displacement of this organ, with remote effects arising from a disturbed circulation in the brain, such as giddiness and headache. On the other hand, a distended cæcum or sigmoid flexure, pressing on veins and nerves, may induce œdema, numbness, and cramps of the right or left lower extremity. 4. Retarded digestion, derangement of the stomach and liver, and intestinal obstruction.

(b) *General.*—The absorption of excrementitious matters may lead to widespread general effects, indicated by a sallow, earthy, or dirty complexion, lassitude, debility, offensive breath, loaded urine, and other symptoms. Chlorosis has been attributed to this cause.

**TREATMENT.**—Atony of the colon is usually a chronic disorder demanding prolonged treatment. The hygienic and dietetic rules laid down in the article CONSTIPATION require in most cases to be supplemented by medicines. The most satisfactory results follow a course of tonics combined with aperients, such as iron, quinine, strychnine, and belladonna, with aloes, colocynth, or rhubarb. The purgative should be adjusted to each case, so as to secure no more and no less than a regular and efficient evacuation; and while the loaded colon continues to be thus relieved, the dose should be very gradually reduced. This tonic-aperient course may be greatly aided by local stimulation of the colon, as by massage, electricity, and cold-water compresses or abdominal douches (see CONSTIPATION). The abdomen should be supported by a belt or roller. Bretonneau and Trousseau strongly advised a course of belladonna, giving gr.  $\frac{1}{7}$  of the extract, or of the powdered leaf, as a pill in the early morning—the stomach being empty; then two such pills, if in four or five days the bowels do not respond; and increasing the dose, but not beyond that contained in four or five pills in twenty-four hours. A teaspoonful of castor oil may be given twice a week to aid this course of treatment. Flatulent distension of the colon in the elderly, and in females at the climacteric period, is often greatly relieved by the prolonged use of a pill containing the compound asafetida pill and extract of nuxvomica, after meals.

In excessive gaseous dilatation of the colon (as in volvulus of the sigmoid flexure), producing much distress from dyspnœa, pain, and vomiting, puncture by a fine trochar or aspirator-needle has been successfully resorted to as a palliative measure.

II. **Stricture.**—Stricture of the colon may occur in any part, but is most common in the sigmoid flexure and descending colon. It is limited to adults. It more commonly arises from cancer than from the cicatrising of an ulcer (simple stenosis). In non-cancerous stricture a history of dysentery is important—though a long interval (even years) may have elapsed since the attack. The stricture declares itself by setting up symptoms of intestinal obstruction—there being at varying intervals attacks of paroxysmal, shifting, colicky pain across the navel, with constipation (or constipation alternating with diarrhœa) aggravated by purgatives, becoming more and more frequent and associated with vomiting—when, as a rule, the difficulty ceases to be intermittent. Large fecal masses may often be felt above the site of obstruction. In stenosis of the sigmoid flexure there is sometimes tenesmus, and the motions are flattened; the stricture can be reached when the whole hand is introduced into the rectum. The motions are not, as a rule, altered in shape when the stricture is situate in the higher part of the colon. The site of obstruction may sometimes be determined by auscultating the abdomen while water is being gently and intermittently injected into the rectum—the observer failing to hear the stream above the stricture. The duration of the cancerous as well as of the simple forms averages from five to six months.

**TREATMENT.**—The treatment of stricture of the colon is further referred to in the article on **INTESTINAL OBSTRUCTION.**

III. **Inflammation.**—**SYNON.:** Colitis; Colonitis; Colo-enteritis; Fr. *Colite*; Ger. *Entzündung der Schleimhaut des Kolons.*

An inflammation, leading to ulceration of the mucous membrane and submucous connective tissue of the colon, producing lesions indistinguishable from those of dysentery, has been pointed out by Copland and Parkes. Colitis is said to be a non-specific local affection, commencing in the submucous tissue, and subsequently attacking the mucous membrane with its glandular structures—the primary seat of dysenteric inflammation. As in dysentery, the inflammation induces gangrenous destruction and ulceration of the mucous membrane and underlying cellular tissue.

A *catarrhal* form of colitis is apt to occur in measles. It often happens that the morbillous catarrh of the intestines exhausts itself by attacking the large intestine, producing that special form of colitis characterized by tenesmus and glairy bloody stools.<sup>1</sup>

<sup>1</sup> Trousseau, *Clinique Médicale.*

Inflammatory diarrhœa, particularly in children, often terminates similarly.

Colitis arising from *retained excreta* may involve the entire wall of a circumscribed portion of the colon, commonly the ascending colon and sigmoid flexure, or may be confined to the mucous and submucous tissues. In fecal retention the mucous follicles of the colon may become obstructed, and the distension resulting therefrom may lead to inflammation and ulceration. Irritation of the mucous lining of the colon from the lodgment of feces may extend to the lymphatic vessels and glands. The glandular enlargement cannot, however, as a rule, be recognised during life. In colitis there are usually colicky pains, intestinal distension, and diarrhœa alternating with constipation; and the stools are often coated with masses of mucus.

**TREATMENT.**—The treatment of colitis consists in the local application of fomentations, poultices, opium enemata, or morphine suppositories; and in the use of gentle laxatives, such as castor oil, combined with sedatives, such as opium, belladonna, or henbane. The catarrhal form generally terminates in spontaneous recovery. Trousseau advised the use of albuminous injections, or of injections containing about half an ounce of water and nitrate of silver—from  $\frac{3}{4}$  to  $1\frac{1}{2}$  grains; or sulphate of copper or sulphate of zinc—from  $3\frac{1}{2}$  to  $4\frac{1}{2}$  grains. The diet must be very carefully ordered.

IV. **Displacements.**—The parts of the colon most liable to displacement are the transverse colon and sigmoid flexure. The former may descend as low as the pubes; the latter may occupy any position between the left iliac region and the right side of the abdomen. Usually the mesocolon is elongated; there is adhesion between the displaced part and the new site; and, the longitudinal bands being elongated, the loculi are obliterated. Displacements are most apt to occur in those who have long suffered from constipation, retention of feces, chronic dysentery, hernia, or from encysted or other tumours. They may lead to complete obstruction, and cannot usually be recognised with certainty during life.

V. **Diverticula.**—A *loculus* of the colon from repeated accumulation may become so distended as to form a lateral appendix. Such a diverticulum when loaded with feces may be felt through the abdominal wall as a distinct tumour, which may collapse when pressed between the fingers.

GEORGE OLIVER.

**COLORADO SPRINGS, in Colorado, U.S.A.**—A clear, cold, and very bracing climate, with great dryness and diathermancy of the atmosphere; 6,028 feet above the sea. No snow in any quantity, and consequently no snow-melting season. Dry

and dusty winds in spring. Camping out at nearly all seasons tolerably safe. A high-altitude station, useful in asthenia and in chronic phthisis, especially with limited lesions and no pyrexia. The name of 'springs' is added from the proximity of Colorado City to Manitou Springs, six miles distant. See CLIMATE, 'Treatment of Disease by.

**COLOUR-BLINDNESS.**—A defect of vision, the subject of which is unable to distinguish certain colours. See VISION, Disorders of.

**COMA** (κῶμα, deep sleep).—A condition of profound insensibility. See CONSCIOUSNESS, Disorders of.

**COMA-VIGIL** (κῶμα, deep sleep; and *vigil*, wakeful).

**DEFINITION.**—A symptom, or set of symptoms, where continuous sleeplessness is associated with partial unconsciousness.

Coma-vigil occurs towards the end of diseases in which the nervous system is involved either directly or indirectly, especially where sleeplessness has been a symptom in the earlier part of the disease. Thus it frequently appears towards the end of an attack of typhus or of delirium tremens, when these are about to terminate fatally.

**SYMPTOMS.**—The patient lies quiet with his eyes half-closed, inattentive to everything around, but not absolutely unconscious. If the eyelids are touched, they are closed, and perhaps the head is slowly turned away. The eyes have a dull, half-glazed look, and slowly follow any moving object near them. The pupils are neither much dilated nor contracted, and they move under the influence of light, but very sluggishly. The mouth is generally somewhat open and dry, as are also the lips. The power of swallowing is much impaired: if a small quantity of fluid be put into the mouth, an effort is made after a short time to swallow it, and this effort is for a time successful; but after the symptoms have been present for some time, the effort is so feeble that no result follows. The patient lies mostly on the back; if turned on the side, he either remains as placed, or often slowly turns to the former position. The limbs are occasionally moved a little; and if the hand or arm be raised, a slight resistance is offered. If the bladder or the rectum be emptied, there is slight consciousness of the act, as if a feeling of discomfort preceded it. The pulse is quick and weak. The respirations are weak, but otherwise normal. The symptoms continue unbroken throughout, nothing like natural sleep occurring.

**PATHOLOGY.**—As being little more than a symptom, coma-vigil has strictly speaking no pathology. It seems to coincide with the gradual suspension through exhaustion of the functions of the nervous centres; the cerebral hemispheres being nearly if not quite inactive, while the action of the rest of the centres

is kept up weakly but continuously, till the little remaining nervous power is exhausted, when death ensues. It differs from coma inasmuch as in the latter the medulla oblongata is the only centre left active, the functions of the rest being entirely suspended. It differs from concussion, inasmuch as the symptoms of the latter attending the temporary unconsciousness are more those of irritation than of pure suspension of function.

**DIAGNOSIS.**—Coma-vigil is distinguished from coma by the presence of a certain amount of consciousness, by the quick pulse, and by the absence of stertorous breathing. It is distinguished from concussion of the brain by the pupil not being contracted, by the history of the case, and by the absence of coldness of the skin, and of any sign of shock.

**PROGNOSIS.**—The prognosis is unfavourable: coma-vigil is almost invariably a fatal symptom. It may last from a few hours to three or four days; from twenty-four to forty-eight hours being the most common duration. It may deepen into actual coma; but more usually the symptoms change but little, save that the pulse becomes quicker and weaker, and the respiration more feeble, and death by asthenia then results.

**TREATMENT.**—Coma-vigil does not call for treatment directly itself, but is an urgent indication for nutritive and stimulant measures calculated to relieve the primary disease.

R. BEVERIDGE. JOHN BANKS.

**COMEDONES** (*comedo*, a glutton).—**SYNON.**: Grubs.—This is the name applied to the little cylinders of sebaceous and epithelial substance which are apt to accumulate in the follicles of the skin, and to appear on the surface as small round black spots. When squeezed out they have the appearance of minute maggots or grubs with black heads, and thence have derived their name. They may occur in all parts of the body where sebaceous follicles exist, but are most common on the face, the nose, the neck and shoulders, the breast, and within the concha of the ears, in the latter situation often attaining a considerable size. The accumulation of this substance is due to want of expulsive power of the skin, and to the slight impediment which is afforded by the aperture of the follicle to its exit. When squeezed out, a comedo is found to vary in colour, in figure, and in density, according to the period of its detention. When recent, the comedones are soft and white, and modelled into an exact cylinder by compression through the mouth of the follicle. If impacted for a considerable time, they acquire the yellow tint, the transparency and hardness of horn; they assume a bulbous figure from the dilatation of the follicle below the constricted orifice of the epidermis; and by their bulk they sometimes stretch the hair-follicle so far as to obliterate it completely. Besides

their usual composition of sebaceous substance and epithelial cells, they frequently contain lanuginous hairs, and not rarely the entozoon folliculorum in its different phases of development (*see* ACARUS). When they raise the pore into a minute pimple they have a similitude to acne punctata, and might be mistaken for that affection; whilst the black spot on the summit of conical aene is due to a comedo.

**TREATMENT.**—Comedones are most frequently met with in young persons in whom the powers of the constitution are not yet established, and who are benefited by generous diet and tonic treatment. Locally, soap and water with plentiful friction and abluion will be found of great service; and, as an astringent, a lotion of perchloride of mercury in emulsion of bitter almonds (two grains to an ounce) and spirits of wine; or a lotion of carbolic acid.

ERASMUS WILSON.

**COMPLICATION** (*con*, with; and *plico*, I fold).—It is difficult to give a strict definition of what ought to be included under the term *complication*; but the word signifies the occurrence during the course of a disease of some other affection, or of some symptom or group of symptoms not usually observed, by which its progress is therefore 'complicated,' and not uncommonly more or less seriously modified. The difficulty lies in determining what should be looked upon as essentially part of the original disease, and what as a mere accidental occurrence. For instance, many regard the cardiac affections which so often arise during the progress of acute rheumatism as a part of the complaint, others as complications. The same remark applies to the relationship of renal disease to scarlatina, as well as to numerous other cases.

Complications arise in different ways. They may, as just indicated, be considered as developments of the original morbid condition, resulting from the same cause and being more or less allied; or they are independent and accidental, of which an illustration is to be found in the association of ague with scurvy or dysentery, or in the co-existence of two or more of the exanthemata. The most important class of complications, however, are those which follow the primary disease as more or less direct consequences. These may further be induced in various ways. Thus, for example, in febrile diseases secondary lesions are liable to arise as a result of changes in the blood; a mechanical act, such as cough, may lead to complications in the course of phthisis and other pulmonary affections; cardiac diseases frequently bring about consecutive changes in other organs, by inducing obstruction of the venous circulation; or emboli may originate under certain conditions and produce their usual consequences. It is of great practical importance to be acquainted with the complications which are

liable to be met with in various diseases, and especially in those which are of an acute nature, in order that measures may be taken for their prevention, and that they may be recognised and treated at the earliest possible period, if they should occur.

FREDERICK T. ROBERTS.

**COMPOUND GRANULAR CORPUSCLES.**—Formerly these microscopic objects were regarded as of inflammatory origin, and as affording positive evidence of the occurrence of inflammation. Hence they were termed 'compound inflammation globules' (Gluge). Almost all pathologists now, however, recognise the fact that they are not products of an inflammatory process, but result either from the degeneration of pre-existing cells, in which protein and fatty granules accumulate, or, perhaps, from the aggregation of granules originally distinct, which are present in abundance in degenerating tissues. They may even be formed out of the cells of morbid products, such as cancer. These compound granular corpuscles derive their name from the fact that they consist of a large number of minute granules aggregated together, and they either present a delicate cell-wall, or this cannot be detected. Occasionally there is an appearance of a nucleus in the centre. *See* CELL.

FREDERICK T. ROBERTS.

**COMPRESS.**—A compress is made of folds of lint or other material, and is used for the purpose of producing pressure, or as a pad by which hot or cold water or medicinal agents may be applied to the surface. In the latter case the compress may be rendered waterproof by being covered with a piece of gutta-percha tissue or mackintosh-cloth. *See* HYDROPATHY.

**COMPRESSIBLE.**—A term implying comparatively slight resistance, and applied specially to the pulse when it yields readily under the finger. *See* PULSE, The.

**COMPRESSION of the Brain.**—*See* BRAIN, Compression of.

**COMPRESSION of the Lung.**—*See* LUNGS, Compression of.

**CONCRETION** (*con*, together; and *cresco*, I grow).—**SYNON.**: Calculus; **Fr.** *Concrétion*; **Calcul**; **Ger.** *Concrement*.

**DEFINITION.**—An unorganised body, formed either in one of the natural cavities or canals, or in the substance of an organ, by the deposit of certain solid constituents of the fluids of the part. In the widest sense of the term, concretions comprehend calculi.

**ENUMERATION AND CLASSIFICATION.**—The following classified list includes the principal varieties of concretions:—

1. In *glandular structures*: lacrymal, salivary, pancreatic, prostatic, seminal, urinary, hepatic, sebaceous, and mammary.

2. In the *circulatory system*: cardiac, and venous (phleboliths).

3. In *closed sacs*: peritoneal, and articular.

4. In *culs-de-sac*: bronchial, pulmonary, nasal, tonsillar, laryngeal, gastric, intestinal, præputial, uterine, and vaginal.

5. In the *substance of tissues* and new formations, especially in the nervous system: corpora amylacea.

6. *Various*: such as the concretions on the teeth known as 'tartar.'

**GENERAL CHARACTERS AND NUMBER.**—Concretions are generally firm or even of stony hardness; but they may be soft and friable. Their colour varies from white to black through shades of yellow and red. Concretions occur either singly or in groups; and their shape and size, as well as the characters of their surface, vary considerably with their number: single concretions are more frequently larger, rounded, and less smooth than multiple specimens, which often present facets and polished surfaces. Many concretions are composed of concentric laminae.

**COMPOSITION.**—The chief constituents of concretions are inorganic, that is, mineral salts, in a basis of organic matter. The bulk of the salts are carbonate and phosphate of calcium and magnesium, with smaller quantities of alkaline compounds. The organic basis is composed of albuminous substances, mucus, cholesterin, and colouring matters.

**MODE OF FORMATION.**—Concretions are generally derived from the solid constituents of vital fluids, whether physiological or pathological. In most instances the fluids are delayed in the natural passages by some abnormal obstruction or dilatation; and under such circumstances a chronic inflammatory condition of the walls contributes greatly to the probability of mineral deposit. Most frequently—as in the formation of the salivary and biliary concretions—the fluid portions of the secretion escape by the natural outlet or are absorbed, while the solid constituents are deposited; the particles being either agglomerated around a nucleus, or deposited in centripetal layers upon the surface of the cavity. In other instances—intracardiac, peritoneal, and articular—a nucleus is furnished by a particle of fibrin, blood-clot, or growth, on which fresh deposits take place, while calcification proceeds in the interior. In a third series, examples of which are found in the alimentary canal, the basis of the concretion consists of foreign or indigestible matter, such as hair, a gall-stone, inspissated fæces, and masses of magnesium or iron salts. See CALCULI.

**EFFECTS AND SYMPTOMS.**—The functions of a part occupied by a concretion are generally more or less impaired; the neighbouring tissues frequently atrophy; and inflammation and ulceration are common results, ending probably in the escape of the body. The

concretion may be passed along a duct, and this process is generally attended with great pain; but concretions may remain where formed without causing symptoms. Occasionally they are spontaneously disintegrated or dissolved.

**TREATMENT.**—The treatment of concretions will be found discussed under the heads of the diseases of the organs where they respectively occur.

J. MITCHELL BRUCE.

**CONCUSSION** (*concutio*, I shake together).—This term is used to indicate a condition induced by a more or less violent shaking or physical commotion of the general system, or of some particular organ, whereby serious symptoms may be induced, but no definite lesion can be detected to account for them. The nerve-centres are the parts most liable to be thus affected, concussion of the brain or spinal cord being of considerable moment, giving rise to more or less complete abolition of their functions, though this effect is usually only temporary (see BRAIN, Concussion of; and SPINAL CORD, Diseases of). General concussion of the body is highly important at the present day, in connexion with railway accidents, after which persons seem to be uninjured, or only to be slightly shaken, but subsequently more or less grave symptoms, associated with the nervous system, set in. See RAILWAY ACCIDENTS, Results of.

FREDERICK T. ROBERTS.

**CONCUSSION OF BRAIN, SPINE, &c.**—See BRAIN, Concussion of; and SPINAL CORD, Diseases of.

**CONDYLOMA** (*κονδύλωμα*, a knob).—**SYNON.**: Fr. *Condylome*; Ger. *Feigwarze*.

This vague term has been used to describe at least two different things—namely, (a) papillomata, or warty growths in the neighbourhood of the anus and genital organs; and (b) the syphilitic lesions called mucous patches or mucous tubercles. Of these the former only will be dealt with here. The latter are described under MUCOUS PATCH.

**DESCRIPTION.**—Condyломата are commonly found in connexion with the constant moisture and irritation due to acrid secretions, whether natural or morbid, especially the discharges of venereal affections. The growths occur most frequently and attain the greatest size in dirty persons, in whom also they are most liable to become inflamed, ulcerated, and fissured. The enormous masses sometimes seen in pregnant women are often called 'cauliflower' growths.

**TREATMENT.**—Frequent washing, followed by the free application of some absorbent powder, such as oxide of zinc, is essential. When the growths are very large, or fail to disappear under caustics, removal by scissors, knife, or cautery will be necessary.

ARTHUR COOPER.

**CONFLUENT** (*confluo*, I run together). Applied chiefly to a variety of small-pox and other exanthemata, in which the eruption runs together or coalesces.

**CONGENITAL** (*con*, together; and *genitus*, begotten).—Existing at birth: a term generally applied to diseases such as *congenital syphilis*, or malformations such as of the heart, and *congenital clubfoot*.

**CONGESTION** (*congero*, I accumulate). Overfulness of vessels, caused by accumulation of their contents: generally applied to blood-vessels. See CIRCULATION, Disorders of.

**CONIUM, Poisoning by.**—SYNON.: Fr. *Empoisonnement par la Ciguë*; Ger. *Schierlingsvergiftung*.—All parts of the hemlock plant (*Conium maculatum*; Greek, *κόνειον*) are poisonous. Both the leaves and fruit are used in medicine. Its toxic properties were known in ancient times; the plant was used for the destruction of criminals by the ancient Greeks, and there is no doubt that Socrates was poisoned by it. Death from conium in this country has perhaps always been the result of misadventure or suicide; but on the Continent the active principle of the plant, *conine*, an alkaloid, has been administered for the purpose of wilful homicide, death resulting from a dose of 10 to 15 drops.

**ANATOMICAL CHARACTERS.**—The signs of asphyxia, engorgement of lungs and of the right heart, and a general venous condition of the blood, appear to be constant after death from conium. There is nothing else specially noticeable.

**SYMPTOMS.**—Preparations of conium, as well as the alkaloid, or mixture of alkaloids, known as *conine*, when taken in toxic doses, produce excessive muscular weakness, beginning in the lower limbs, and extending gradually upwards, with giddiness and disordered vision. These symptoms are in some cases preceded by nausea and vomiting, with dryness or burning pain in the mouth and fauces. There is a desire to remain quiet, and a peculiar heaviness or drooping of the eyelids, the patient lying with his eyes shut. This, and the impairment of vision, appear to be due to paralysis of the ocular muscles. The pupils may be natural, but later they become dilated. The pulse is slow till death is actually impending. The paralysis progresses gradually upwards, till eventually heart and respiration are affected, more especially the former. Convulsions and impairment of the mental faculties—hitherto intact—now set in; finally sensation is impaired, and death ensues from asphyxia.

**DIAGNOSIS.**—The paralysis of motion, progressing gradually upwards, with sensation long unimpaired, and the peculiar drooping of the eyelids, are perhaps diagnostic of the nature of the poison.

**PROGNOSIS.**—As no antidote is known

which counteracts the effects of conine, the prognosis must always be a guarded one, and will depend entirely upon the general condition of the patient.

**TREATMENT.**—The stomach must be emptied by the stomach-pump or syphon-tube, and well washed out. Emetics may also be used to evacuate the stomach. Tannin and other astringents must be freely administered, to precipitate the active alkaloid, and prevent its absorption. Castor oil, administered by the mouth or rectum, may aid the removal of the alkaloid when it has been rendered insoluble by tannin. Strong coffee, brandy, ammonia, and stimulants generally are serviceable, as may also be the hypodermic injection of ether. Hypodermic injections of 1-40th grain of sulphate of atropine are very promising, especially in the later stages; atropine acting as a respiratory and cardiac stimulant. Artificial respiration, and stimulation of the respiratory and cardiac functions by the use of electricity, ought not to be neglected when these are affected.

THOMAS STEVENSON.

**CONJUNCTIVITIS.**—Inflammation of the conjunctiva. See EYE AND ITS APPENDAGES, Diseases of.

### CONSCIOUSNESS, Disorders of.—

The disorders of consciousness are so numerous as to make it desirable briefly to consider them in one article, with a view to their classification and the better comprehension of their mutual relations. We shall, therefore, here group and arrange the varied morbid conscious states, not aiming to produce a strictly scientific classification so much as one which will be practically useful.

1. **Exaltation.**—Under this head may be ranged certain states of consciousness more or less distinctly bordering upon the unnatural, to be met with in persons under the influence of 'mental excitement' from various causes, as from sudden good news, or generally pleasant surroundings; also from a slight degree of poisoning by alcohol, opium, hashisch, or other drugs; or from an early stage of some forms of insanity, or of delirium. In this state of mental exaltation the individual's powers of perception, apprehension, recollection, thought, emotion, and volition, would seem to be all more or less intensified, just as in that of hebetude or dementia they are diminished, and consciousness is proportionately dwarfed.

2. **Perversions.**—Many of the various defects here to be referred to are very partial in the extent to which they implicate consciousness, though others are general. In what is known as an *illusion* some object of sense is not correctly perceived; or, in other words, some sensorial impression is quite wrongly interpreted—as when a feverish or a maniacal patient, looking at some inanimate object, declares that it is a cat or a dog.

about to fly at him, or, hearing even the slightest noise in any part of his room, interprets it to be the voice of some friend or imagined enemy. In the case of an *hallucination*, however, forms are declared to be seen, or voices heard (by a patient suffering from delirium tremens, for instance), where no appreciable external realities could have started the notion. And in these cases, it is not that the patient sees or hears without believing; he implicitly believes that the visions or voices, which have been conjured up subjectively by the mere disordered working of his own brain, have a real existence in the outside world. It is necessary to make this distinction, because it is by no means uncommon, in regard to the olfactory sense (especially in some epileptics), for odours or smells to be perceived which the patient soon comes to know are purely subjective or devoid of any external correlative.

Hallucinations and illusions, moreover, though occasionally existing alone, are quite commonly associated with a very important and more general derangement of consciousness, namely, *delirium*. This is a symptom very common in many fevers, in certain low states of the system, after severe frights, in inflammatory or other lesions of the brain and its membranes, as a result of some narcotico-irritant poison, or occasionally in a person who is recovering from an epileptic attack, or from the stupor sequential to a series of convulsive attacks. The state itself varies much in intensity. Three fairly distinct types exist. In (*a*) *low or muttering delirium* the patient lies still and more or, less heedless of what is occurring around; or, if heeding at all, the impressions which he receives give rise to erroneous perceptions (illusions), which are woven into the incoherent fabric of his rambling thought. In (*b*) *delirium tremens* the patient is more restless; tremors of the limbs and of the muscles of the face are often easily induced; hallucinations of sight and hearing are common; and the character of the delirium reveals that the patient is, to an unusual extent, possessed by fears, terrors, and other emotions of a depressing type. In (*c*) *wild or raving delirium* we have to do with a much more active state. The patient raves loudly and incoherently, more in regard to his fleeting dream-like thoughts than in connexion with external impressions, of which he is more or less heedless. He is often violent in demeanour, and difficult to be restrained, persons in this state being capable of great and prolonged muscular exertion. The bodily activity accompanying this form of delirium is, in fact, just as characteristic as the great intensity of the mental processes. It is met with occasionally in some fevers, but more commonly in meningitis and in acute mania.

In its early stages delirium is principally

noticed during the transition-period between waking and sleeping—at times, that is, when the nervous system most needs the re-invigorating influence of sleep. It is in these cases, too, that beef-tea or stimulants may for a time dispel all traces of the wandering thought. Whilst illusions and hallucinations enter largely into the mental activity of a delirious patient, *delusions* also are generally well-marked components. That is to say, the person becomes for a time possessed by an idea, notion, or fancy, for which there is no real warranty, though he believes and wishes to act as though it were true.

Somewhat allied to delirium in nature, though much lower in intensity as a mental process, is that *incoherence* of thought which is met with in many chronic maniacs, or in non-febrile patients suffering from various organic brain-diseases. In its slighter degrees this incoherence displays itself as mere 'rambling' talk; the patient has not sufficient brain-power to follow up the main subject of thought, and is frequently diverted into collateral channels. This, which is a natural state with some persons, may be distinctly indicative of disease in others whose mental power has previously been of a more vigorous type. At times the incoherence is seen to be governed principally by mere verbal suggestion, the patient being led away from point to point in new directions, owing to the associations of some word which has been used becoming for the time dominant. This state is often well seen in the sub-acute exacerbations of chronic mania, though it may occur also where multiple softenings or indurations of the brain exist. At other times the incoherence is more absolute—wayward transitions from subject to subject, connected by no discoverable bond, rapidly following one another. The result in such a case is a mere unmeaning jumble of words, interspersed here and there with brief propositions having a limited significance of their own, though often wholly unrelated to that which precedes or follows.

*Hypochondriasis* is a perverted state of consciousness, having some resemblance to that of illusion, but in which some internal or visceral state becomes the starting-point of impressions (possibly not actually painful) which, when magnified and perverted as they are in the mind of the patient, fill him with false and gloomy apprehensions of various kinds. This perversion of consciousness is more generalised than that which exists in the case of illusion; and also, instead of being a more or less temporary defect, it is one that may last for weeks, months, or even years. The state of mind of an hysterical patient is often not altogether different from that of the hypochondriac.

3. **Partial Loss.**—Defects of this order are numerous and may exist in great variety. They may implicate almost equally nearly all

the varieties of conscious mental activity, or some more than others. They may be either congenital, or acquired during the life of the individual.

In *idiocy* we may have from birth defect in the power of concentrating the attention, a defective power of apprehension and of thinking, and a defective volition, shown alike by an inability to guide or control thought, and by a deficient vigour of bodily movement. Again, as a result of epilepsy, of organic brain-disease, or of injuries to the head, the patient may gradually lapse into such a condition from one of health, so as to become, as it is termed, 'demented.' Whilst this state of *dementia* may supervene at any age, it is much more common as a consequence of the brain-diseases frequent in advanced life. There is, moreover, a form known as *senile dementia*, in which without any typical disease, but as a consequence of impaired tissue-vitality and diffused degenerative changes throughout the nervous system, the mental faculties undergo a more marked degradation than is usually met with in old age. This condition in its minor degrees goes by the name of *hebetude*. In all such states or grades of idiocy and dementia, we meet with an undue tendency to sleep in the daytime as a result of the listless and languid mental condition. This is but another sign of the general lowering of conscious vigour.

Here we must include, also, a peculiar group of conditions, having some alliance with one another, and which are all characterised by loss of consciousness to some extent, either partial in range or general. They are—*reverie*, *somnambulism*, *ecstasy*, *coma-vigil*, *catalepsy*, *hypnotism*, and *trance*. They are merely enumerated here, but are defined or described in their several places. In the last of these conditions the loss of consciousness, in the ordinary acceptation of the term (namely, loss of perceptive power), is so absolute, that some may think it ought rather to be included in the next section. Loss of perceptive power, however, would not seem to be absolutely synonymous with loss of consciousness. There is good reason to believe, for instance, that where the influence of chloroform and other anæsthetics is not pushed to the fullest extent, a condition of *anæsthesia* intermediate between slight and profound is produced, in which, whilst there is absolutely no consciousness for external impressions, so that pain is altogether unfelt, there is still a certain amount of cerebral activity—as evidenced by rambling and indistinct speech on subjects altogether apart from what the surgeon may be doing. There is mental activity clearly, though the nature of this, as revealed by the patient's speech, may preclude the notion that pain is at the time being felt. Sensorial consciousness is blotted out, whilst a kind of ideational con-

sciousness remains. We have an approximation to such a condition, also, in the case of *sleep* when dreams are rife. But here sensorial consciousness is not completely in abeyance. Again, in certain rare and anomalous epileptiform attacks we may find the patients, after the first paroxysms, bereft of some senses, though not of others. They may hear what is said by those around them, though they continue for a time quite unable to see or speak.

**4. Complete Loss.**—In very profound sleep (*sopor*), in that prolonged form of it in which the person, if he can be momentarily roused, drops off again immediately (*lethargy*), and also in profound *anæsthesia*, there is complete loss of consciousness. The terms *sopor* and *lethargy* are now rarely used, and authors are not even agreed as to the precise state which would be designated by the latter word. It is sometimes regarded as a synonym for 'trance.'

In *syncope* we have insensibility resulting from a cutting off of the proper supply of blood to the brain; whilst in *asphyxia* we have a like result following upon an interference with respiration, and a consequent engorgement with impure blood.

A condition of *narcosis* or profound insensibility may result from opium or other drugs and poisons, amongst which alcohol is to be included as one of the most common producers of such a state. It may also be due to the deficient elimination of waste products by the kidneys, when 'uramic coma' is produced; or to the abnormal production of chemical compounds in the system, which lead on to blood-poisoning, as in 'diabetic coma,' and in the final stages of septic poisoning.

Complete loss of consciousness exists for some time during the ordinary form of *epileptic fit*, or during an attack of convulsions; though in other epileptiform fits, not infrequently met with—having some of the characters of hysterical convulsions—there seems to be a loss of sensorial consciousness only (loss of perception), whilst a certain amount of ideational consciousness remains. In *apoplexy* also there may be for hours or days a more or less profound loss of consciousness. In the less profound attacks, as well as after an epileptic fit or an attack of convulsions, the loss of consciousness is not complete, and we have a condition now commonly known as *stupor*. This state is also frequent as a result of concussion or other injuries of the brain, and it occasionally follows a severe fit of hysterical convulsions. It may last for hours, days, or even weeks in some cases. In it the patient lies with his eyes closed, taking no heed of what is passing around, though he may show obvious signs of feeling when touched or pinched, and may be capable of being momentarily roused, so as to give a short monosyllabic answer if slightly shaken or spoken to in a loud voice.

On these occasions, signs of impatience are often shown. Though such a patient will not ask for food, he will often drink freely when it is offered. He will of his own accord, when his bladder is full, sometimes get out of bed, find the chamber-pot, use it, and return to bed without saying a word—and then speedily relapse into his previous state of stupor. When the insensibility is more profound, both urine and fæces are passed incontinently.

The state just spoken of is referred to in this section because it is so intimately allied to and connected by all sorts of transition conditions with another, known as *coma*, in which the loss of consciousness is more complete and absolute. There are different degrees of stupor and there are different degrees of coma; the former is commonly spoken of as slight or deep, whilst a comatose condition, coma, and profound coma (the latter being what the older writers termed *carus*) are the phrases ordinarily used to denote the increasing insensibility of the graver state, which is more especially characteristic of the apoplectic condition. Coma may result from long-continued exposure to cold, from sunstroke, from poisons of various kinds, from erysipelas of the head and face, from inflammations of the meninges, multiple embolisms, the effects of hyperpyrexia, or from cerebral hæmorrhage. The most common cause of very profound coma is cerebral hæmorrhage (apoplexy). In this condition the breathing is often loud and stertorous, and consciousness is entirely obliterated, so that there is an utter absence of reflex movements when a limb is pinched or when the conjunctiva is touched. The patient in the deeper forms of coma often cannot be roused at all, even for a moment; and if this state does not terminate in one way or another before the expiration of twenty-four hours, or if it does not gradually pass into one of mere stupor, a fatal result may be considered imminent.

H. CHARLTON BASTIAN.

**CONSTIPATION** (*con*, together; and *stipo*, I cram).—**DEFINITION.**—Slow passage of the fæces from the cæcum to the anus, leading to infrequent or incomplete alvine evacuation, and to retention of fæces.

**ÆTIOLGY.**—The causes of constipation may be *local*—an impediment to the onward movement of the fæces in the large intestine or from the rectum; or *general*—pertaining to habits, diet, and other conditions.

**Local.**—The local causes of constipation include: (a) Lesions inducing narrowing of some part of the large intestine, as from cicatrising of an ulcer or displacement of the bowel. (b) Collections of scybala, intestinal concretions, &c., in the cæcum, sigmoid flexure, or rectum. Fæces are more especially apt to collect when too solid or when deficient in bile. (c) Pressure on the rectum, by

uterine fibroids or ovarian tumours, uterine displacements, the gravid uterus, or an enlarged prostate. (d) Defæcation thwarted, as when the expiratory abdominal muscles are enfeebled, as in pregnancy—especially when repeated or after twins, obesity, old age, or in some painful affection of the abdomen, such as rheumatism of the abdominal walls and diaphragm, chronic dysentery, piles, fissure, or the evacuation of a hard mass of fæces. (e) Feeble contraction of the intestinal muscular fibres, as in distension of the large intestine or a portion of it by gas, fæces, or lumbrici, inflammatory affections (chronic intestinal catarrh, chronic peritonitis), lead-poisoning, senile atrophy, or in delicate females with lax muscular fibre (*see COLON, Diseases of*). (f) Pain in the pelvic viscera, and probably elsewhere, may induce paralysis of the sympathetic nerves supplied to the intestinal walls; thus may be explained obstinate constipation in painful uterine and ovarian diseases, which cannot be accounted for by pressure on the bowels or otherwise.

**General.**—The general causes of constipation are: (a) Sluggishness of function—lymphatic temperament, hereditary influence, anæmia, especially with amenorrhœa; or, on the other hand, disposition to great activity of the muscular and nervous system. (b) Certain habits, such as sedentariness; railway travelling—long journeys; too great muscular activity; mental application, especially when excessive or prolonged; the continued and *apparently* necessary use of aperients or enemata after the relief of temporary constipation—falling under the tyranny of aperients; habitual disregard of defæcation, or hurry in the act; prolonged hours of sleep; the excessive or even moderate use of alcohol, tea, tobacco, or opiates. (c) Dietetic errors: the diet too nutritious—leaving little intestinal residue, or poor and insufficient; improper feeding, especially in infants and children; and the use of indigestible substances, such as cheese, nuts, or uncooked vegetables.

Constipation is frequently a prominent symptom in diseases of the stomach; of the liver; of the heart, inducing congestion of the portal system; and of the nervous system: as well as in connexion with diabetes, excessive perspiration, prolonged lactation, and morbid discharges.

The causes of constipation are such as evidently induce one or both of the following conditions, namely, (1) dryness and hardness of the contents of the large intestine from deficient secretion, or too active absorption of fluid from the intestinal tract; and (2) impaired contraction of the muscular fibres of the large intestine.

**DESCRIPTION.**—In constipation the evacuations are infrequent, solid, deficient in quantity, and sometimes unusually offensive; they often consist of dry, hard, dark or clay-

coloured masses or scybala. Defæcation is generally difficult or even painful. As a rule the depth of colour, and the scybalous character of the motions, are in proportion to the duration of the lodgment of fæces in the large intestine. Infrequency of defæcation regarded alone is an untrustworthy sign of constipation, or of constipation demanding medicinal or other treatment, inasmuch as it often depends on individual peculiarity. Good health may be consistent with departures from the ordinary rule—a daily evacuation; not infrequently there is no relief from the bowels for several days or even for a week, and yet without inconvenience, so long as the infrequent defæcation is habitual, or can be ascribed to idiosyncrasy.

The disturbances of function usually associated with constipation may be local, or may extend to distant parts.

The *immediate* or *local* effects are such as may arise from retention of fæces:—signs of fæcal collections in the cæcum, colon, sigmoid flexure, or rectum; irritation of portions of the intestine, indicated by colic, inflammation, dilatation, ulceration, and perforation of the intestines, followed by peritonitis; intestinal obstruction—a portion of the bowel loaded and distended with fæces being no longer capable of peristaltic movement; pressure of fæcal accumulations on the intrapelvic vessels and nerves, inducing menorrhagia, uterine catarrh, seminal emissions, varicocele, hæmorrhoids, cold feet, neuralgia, numbness and even œdema of the legs (*see FÆCES, Retention of*). Constipation frequently exerts a pernicious influence on primary digestion, indicated by foul tongue, fetid breath, anorexia, acidity, flatulence, biliary disturbance—even jaundice, and urine loaded with lithates.

The *remote* or *general* effects of constipation are lassitude of body and mind; headache, flushing, heat of head, and vertigo; palpitation; anæmia, and wasting. Headache and nausea depending on retention of fæces may be either pretty frequent without loaded tongue; or may recur with vomiting and much coating of tongue every three or more weeks—these periodical attacks resembling migraine, but always cleared up by an aperient.

**TREATMENT.**—Constipation depending on individual peculiarity is rarely relieved permanently by treatment. The bowels, having acquired from early life the habit of infrequent evacuation, may be stimulated for a time, and are then apt to become more sluggish than before. In all cases the habit of the patient in this respect from childhood should first be determined, either as a caution against active or prolonged treatment, which may prove injurious, or as a guide to the adjustment of directions and remedies—affording as it does a limit which should not be overstepped. A healthy daily discharge

of fæces should not fall far short of five ounces, and should form a coherent cylindrical-shaped mass of five or six inches in length, which should float; and it is an essential condition of healthy and efficient defæcation that the colon should always be moderately full of retained fæces. The practitioner should bear in mind that many persons acquire the erroneous belief that they are not discharging a sufficiency of fæces, and in consequence betake themselves to aperients, which daily remove the normal residuum of fæces that should exist in the colon—healthy peristalsis and defæcation being thus out of question. This apparent constipation can only be met by completely suspending the use of aperients.

1. *Ætiological, hygienic, and dietetic treatment.*—In treating constipation the causes should be met. Local causes, such as those inducing contraction of or pressure on some part of the large intestine, or feeble or ineffectual contraction of the intestinal muscular fibres or of the expiratory muscles, should first be eliminated. When defæcation is obstructed by the sphincter and either remaining firmly contracted, or failing to relax sufficiently, it may be effectually relieved by forcibly dilating the anus under ether. Habits disposing to constipation should also be corrected. Persons who are much preoccupied or careless are apt either to disregard the call to stool, or to perform the act of defæcation hurriedly, incompletely, and at irregular intervals. Such persons should be strictly enjoined to direct their attention, expectancy, and will to the attainment of complete and regular evacuation. The sensibility of the nerves of the rectum becomes blunted by the constant contact of fæces, and the promptings of nature at last cease. Hence the periodical removal of collections in the lower part of the large intestine is an essential element of the treatment. This is best secured if possible by well-timed and well-directed and sustained natural efforts. The patient should be told to attempt defæcation every day after breakfast, and to persevere in this habit even though the result be occasionally or frequently unsuccessful, and in order to develop his solicitation into the daily habit of evacuation he should continue to practise it always at the same hour, and should disregard any prompting that may arise at other times. While straining to relieve the bowels, he may facilitate evacuation by pressing firmly the fingers in front and on each side of the coccyx, thus supporting the levator ani during contraction, reducing the concavity of the rectal pouch, and causing the mass of fæces to glide forwards through the sphincter, which should be smeared with vaseline; or he may attain the same end by alternately contracting or relaxing the anal sphincter. Failing to obtain relief on the second day,

a small cold-water enema should be used to prevent further accumulation of fæces in the rectum, and to restore tone and sensibility to the blunted nerves. The enema should never be larger than is required to dislodge the motion from the pouch of the rectum, nor should it be warm; at first it may, however, be tepid, but should afterwards be cold. When evacuation is obstructed by the lower part of the fæcal mass becoming dry, relief may be obtained from a small emollient enema administered on rising (such as infusion of linseed, glycerine in small quantities, solution of white of eggs, olive oil alone or in oatmeal gruel) and allowed to remain until after breakfast; or by the use of suppositories at bedtime, consisting of cacao-butter, or honey hardened by heat, either alone or combined with extract of belladonna. The clothing should not constrict the abdomen or waist, and should be warm—especially about the feet and legs. Sedentary habits should be broken into. Exercise on foot or on horseback, or on a cycle, is especially to be commended, and carriage exercise to be avoided. While studying or reading, the patient should walk about, and stand rather than sit at the desk. Gymnastics and out-of-door games (such as lawn-tennis, cricket, football, golf) are useful when a limited time only can be devoted to exercise. Excessive and exhausting exertion should be avoided. It is generally advisable to recommend early rising and cold bathing, followed by brisk general towelling in the morning. In different cases one or other of the following may be found serviceable: a shower or sponge bath containing vinegar or bay-salt, or consisting of sea-water, or a cold sitz-bath; douches directed to the abdomen; a cold-water compress applied to the abdomen during the day or night, or for three or four hours in the morning; friction or kneading, or slapping with the half-closed fist in the course of the colon every morning, and when at stool; an abdominal belt (flannel or elastic)—especially if the abdomen be pendulous.

*Diet.*—The diet should be varied, should not consist too exclusively of animal food, but should contain a good proportion of fresh vegetables, and especially green vegetables (cabbage, lettuce, spinach). Fresh and ripe fruit (such as grapes, apples, and oranges) should also find a prominent place, and are most effective when taken in the early morning, or at breakfast. When fruit induces acidity a tumblerful of alkaline (Vichy or Vals) water should be taken at the same time. Oils and fats are generally serviceable when they do not disturb the digestion, such as the addition of a dessert-spoonful of olive oil to green vegetables or to potatoes. A tea-spoonful of glycerine with each meal is often helpful. The diet should not be too dry. As to beverages, much must be left to individual experience: sometimes coffee, or beer, or cider answers best. A tumblerful of cold water

should be taken night and morning on awaking; sometimes carbonic-acid water in the early morning is found preferable to plain water. Coarse articles of food—such as bran or wheatmeal bread, oatmeal porridge, &c.—are often commended, and now and then they do favour the continuance of more efficient evacuation; more frequently, however, they fail—especially after a time—and they then aggravate the evil by further adding to the undue collection of residue in the large bowel, and are apt to set up catarrhal irritation. Acidity and other symptoms of dyspepsia contra-indicate the use of these indigestible foods. Honey or treacle with bread, gingerbread, and Spanish or Portugal onions, plainly boiled, may be found useful in certain cases. Dujardin-Beaumez recommends infusion of linseed (a little water let stand on a dessert-spoonful of linseed for an hour) immediately before a meal.<sup>1</sup> As a rule, eggs, milk, cheese, farinacea, astringent wines, and tea increase constipation; and besides these articles it is best to avoid pickles, salted meats, nuts, and pastry in any form.

2. *Treatment by massage and electricity.* Many observers in recent years have reported the complete cure of constipation by a course of massage alone. The manipulations consist of kneading (*pétrissage*), stroking (*effleurage*), and slapping (*tapotement*), each having its special effect: the first loosening impacted fæcal masses, the second favouring their passage along the bowel, and the third improving peristaltic action. In some instances the forms of massage best adapted to the case should be applied to the whole of the large bowel from the cæcum onwards; in others it should be addressed specially to certain portions, as the cæcum, transverse colon, or sigmoid flexure. Sometimes all the manipulations, in varying proportions and degrees, are required in succession, or only one or two kinds—for instance, in atony of the bowel depending on sedentary habits, gentle and then deep stroking should give place to strong slapping with the half-closed hand; and in cases marked by flabbiness of the abdominal muscles, the recti should be forcibly separated by the thumbs and fingers, and then strong transverse strokings from the median line should be made, and the patient should take deep inspirations to strengthen the diaphragm. Each case should therefore be treated with discrimination, and a routine method should be avoided. The massage should be applied for from twenty to thirty minutes every or every other morning, and should be continued without interruption for from five to eight weeks.<sup>2</sup> *Electricity,*

<sup>1</sup> *L'Hygiène Alimentaire.*

<sup>2</sup> A rude form of massage has been suggested in the form of a cannon-ball, or other similar round metallic body, weighing 3 or 4 lbs., to be rolled up and down the uncovered abdomen for from five to ten minutes daily at the same hour, the patient being recumbent.

either alone or with massage, has also proved successful in certain cases; a mild galvanic (continuous) current being applied by inserting the negative pole well within the sphincter ani and drawing the positive pole along the colon, and the faradic (interrupted) current being passed through the abdominal muscles.

3. *Medicinal treatment.*—Should the foregoing directions fail to establish the habit of a regular action of the bowels—either daily or on alternate days, they may be supplemented by the prescription of medicinal agents, to compass the twofold aim of (1) relieving the large bowel of the excess of fæces and gases which, by over-distending it, opposes peristalsis, and (2) toning its walls. To this end care should be taken to so adjust the dose (for it varies considerably in different cases) as to secure if possible efficient assistance to the discharge of fæces, without going so far as to clear away or to considerably reduce the normal residuum which should always exist in the colon. If, therefore, any prescribed dose has exceeded this limit it should be reduced, and should not be given next day, but after the lapse of a day or two. Repeated purging exhausts torpid bowels, and perpetuates constipation. Nor should the bowels be pushed to more frequent relief than has been habitual with the patient from early life. If the evacuations habitually contain scybala, and fæces are generally found, on examination, in the rectum, the injection of a tea-spoonful of glycerine, or the insertion of a glycerine suppository, twenty minutes or so before the time of the desired evacuation, may alone suffice, or may enable a smaller dose of medicine to deal effectually with the difficulty. If gentle assistance only is required, and if griping and other signs of irritation are readily set up by the ordinary aperients, one or other of the following may be tried:—a tea-spoonful of castor oil alone, or with an equal part of glycerine, beaten together and then added to a table-spoonful or two of milk, or with olive oil or almond oil, at bedtime or in the early morning; the official sulphur lozenges (suggested by Sir Alfred Garrod) or tabloids at bedtime; rhubarb and dried carbonate of sodium (gr. ij. or iij. of each), or a small piece of rhubarb-root before the midday meal; pills of dried ox-gall and soap. Or, if a more decided laxative is necessary, a preference may be given to the compound liquorice powder, or confection of senna (with or without confection of sulphur or the compound sulphur tabloids), or the infusion of a few senna-pods in half a tumbler of water let stand over night, or extract of cascara sagrada. During late years cascara sagrada has acquired a high position as a tonic aperient, and is prescribed either in the form of solid extract (as in tabloids or pills, alone or variously combined), or in that of the liquid extract—the dose of the latter varying from ℥v. to ℥xxx. alone or asso-

ciated with tincture of nux vomica, of belladonna, or of podophyllum, in a tea-spoonful of glycerine, or along with euonymin in capsules. Combinations on the lines of the favourite formula of Sir Andrew Clark also meet the indications well in the majority of cases (aloin, extract of nux vomica, extract of belladonna, sulphate of iron, powdered myrrh, powdered soap, of each half a grain; and, if the fæces are dry and hard and there is no special weakness of the heart, add powdered ipecacuanha half a grain). Some prefer a pill of Socotrine aloes with henbane and sulphate of iron and quinine. In a well-organised course of medicinal treatment the form of the aperient should be varied every now and then, and the dose very gradually reduced until at last it is almost withdrawn, while the intestinal tonics (belladonna, nux vomica, and iron) should be continued. The time when the dose should be taken is important, for it will vary—be it before lunch, or dinner, or at bedtime—with the slowness or quickness of the response of the bowels to the attainment of relief after breakfast. Bretonneau's treatment by belladonna, greatly prized by Trousseau, is described in the article COLON, Diseases of.

In anæmic cases a firm and prolonged course of iron should be aided by aloes, nux vomica, and arsenic. In hæmorrhoidal complications aloes should, as a rule, be avoided, and laxative electuaries should be prescribed; in some cases, however, it gives tone without irritating the rectum. In obstinate cases colocynth—either alone or variously combined—is most useful; such as the tincture of the Prussian Pharmacopœia, ℥v. or more on sugar or in extract of liquorice (to disguise the bitterness) three or four times a day, or 10 to 20 minims an hour before breakfast; or the extract or compound pill with small doses of croton oil, or with gamboge, elaterium, or other combinations. A full dose of opium may liberate the bowels after the failure of the strongest purgatives, and constipation depending on inhibition of the sympathetic nerve from pain will be relieved by opium with belladonna. When constipation is associated with a deficient flow of bile, chologogues (such as an occasional mercurial, euonymin, or podophyllum) should be prescribed with the other remedies; and dried ox-bile in capsules or pills, and nitric acid or nitro-hydrochloric acid often prove useful. Sometimes enemata answer better than purgatives; then, when frequently required, they should be small in quantity, and at first tepid, afterwards cold; for occasional use, for the purpose of clearing away fæces loading the large bowel, they should be large (from two to six pints) and warm (*see* FÆCES, Retention of). The frequent use of large warm injections perpetuates constipation. Purgative waters, such as the Rubinat, Friedrichshall, Pullna, Hunyadi János, or Carlsbad, given in warm water

in the early morning or over night, are often valuable adjuncts to other treatment. A combination of the sulphates of iron, magnesium, and sodium with strychnine and belladonna, after food twice a day, is often efficacious; especially in the constipation of children; and in children under two years of age carbonate of magnesium (gr. v. to xx.) in milk is preferable to other aperients.

GEORGE OLIVER.

**CONSTITUTION.**—SYNON.: Diathesis; Habit; Conformation of Body; Fr. *Constitution*; Ger. *Leibesbeschaffenheit*.

The constitution may be sound or unsound. A sound constitution may be defined as the harmonious development and maintenance of the tissues and organs of which the body is made up. It originates with the union of a healthy sperm and germ cell, continues with the growth of the product under the most favourable conditions to adult life, and becomes gradually enfeebled with advancing age by the process of natural decay.

The constitution may be unsound in consequence of *deficient vitality*. This deficient vitality may be general, as is sometimes observed in the children of parents one or both of whom are in advanced life, or whose vitality on one side or the other has been reduced by excesses, such as alcoholic or venereal. Exhausted vitality from prolonged disease, e.g. phthisis or tertiary syphilis, affecting either parent, may determine the death of the offspring at an early period, from mere failure of nutrition, or may cause it to succumb to acute disease not necessarily associated with any inherited tendency of a special kind. The deficient vitality may be restricted to certain tissues or organs, namely, those concerned in the nervous, vascular, respiratory, or digestive systems. Thus amongst the most strikingly hereditary of diseases are those of degeneracy, such as emphysema, structural heart-diseases, atheroma of vessels, and certain kidney-diseases. Rightly interpreted, these diseases are of the nature of premature senility, attacking certain tissues or organs—as it may be seen to attack the hair or the cornea—from some inherent defect in their vitality.

The constitution may, in the second place, be unsound from some definite *inherited* form of disease. Although the constitution of an individual begins with his life, it is nevertheless the resultant of the constitutional peculiarities of many antecedents. This being so, tendencies to disease may date far back in the pedigree, to be called forth from time to time by favouring circumstances. We need, however, practically only go back a few generations in inquiring for those diseases which are well recognised as being hereditary. These form one section of the group of *constitutional diseases*.—Congenital syphilis, gout, scrofula, tuberculosis, cancer, asthma, and certain neuroses, are all diseases which

are apt to appear at certain periods of the life of the offspring, in consequence of some specific inherent defect of blood or tissue derived from his progenitors.

The constitution may, thirdly, become unsound at any period *subsequent to birth*. (a) This may be due to the surrounding conditions of life being evil. Deficient or impure air, insufficient or improper food, defective sunlight, over-work, intemperance, &c., may injure the constitution, and give rise to diseases whose constitutional nature is sometimes strikingly shown in the tendency of some of them to become hereditary. Rickets, phthisis, and scrofula are examples. (b) The introduction of certain poisons into the system affects the constitution profoundly, and in some cases permanently, after the more obvious effects of the poisons have passed away. All the acute specific zymotic diseases, including vaccinia, would come under this category. They render the organism, for a long period or for life, proof against subsequent attacks of the same disease. Only in certain cases, however, can the soundness of the constitution be said to be *impaired* by such diseases, and then it is usually through the occurrence of sequelæ.

R. DOUGLAS POWELL.

#### CONSTITUTIONAL DISEASES.

These may be regarded as diseases generated from within, in the course of the wear and tear, nutrition and waste, of the body, in consequence of inherent or acquired weakness in its construction.

The applicability of the term 'constitutional' to disease is sufficiently explained in the preceding article on CONSTITUTION. The term may, however, be associated with a group of so-called 'general' diseases, in opposition to that which includes 'zymotic' or 'specific' diseases, which are generated by the introduction of some definite poison from without.

In our present state of knowledge, however, no very rigid lines can be drawn to separate local, general, constitutional, and specific diseases from one another. See DISEASE, Causes of.

R. DOUGLAS POWELL.

**CONSTRICION** (*constringo*, I bind together).—A narrowing, to a limited extent, of a canal or hollow organ, due either to a textural change in its walls, or to the pressure of a band surrounding it.

**CONSUMPTION** (*consumo*, I waste).—A term for any wasting disease, but generally applied to pulmonary phthisis. See PHTHISIS.

**CONTAGION.**—The word *contagion* is applied in pathology to the property and process by which, in certain sorts of disease, the affected body or part causes a disease like its own to arise in other bodies or other parts; and the Latin word *contagium* is conveniently

used to denote in each such case the specific material, shown or presumed, in which the infective power ultimately resides. See ZYME; and ZYMOSES.

The property of CONTAGIOUSNESS belongs to a very large number of the diseases which affect the *human* body. And in more than this direct way the property is of great interest to mankind. Contagiousness of disease is a fact not only for man, but apparently for all living nature; and the influence of contagion in spreading destructive diseases among *domestic animals*, and among those parts of the *vegetable kingdom* which contribute to the nourishment of man, is such as to make it of immense social importance that the laws of contagion should be well studied and understood. Further, just as contagion in the case of *living* bodies and their parts spreads disease from one to another, so, to an immense extent, in the case of certain matters which, though of organic origin, are *not living*, it spreads various processes of decay. The so-called 'fermentations' which yield alcohol and vinegar, as well as that in which putrefaction consists, are contagious affections of the respective matters in which they occur: every cheesemonger knows that moulds of different kinds spread by inoculation, each in its own kind, from cheese to cheese; and if the Greek proverb 'grape mellows to grape' is true of the living fruit, the apple-loft gives analogous experiences of contagion among the fruit which is garnered.

The RATIONALE of the word 'contagion' as now used, is that the property is understood to attach itself essentially to a material *contact*; not necessarily that, when infection is spread from individual to individual, the contact of the individuals must have been *immediate*; but that in all cases there must have been such passage of material from the one to the other, as was in itself at least a *mediate contact* between them. And similarly, in those very instructive illustrations of the process of contagion which are furnished within the limits of a single diseased body by the propagations of disease from part to part of it, we can in general easily see that infection advances from part to part, either in proportion as part touches part, or in proportion as the one receives from the other the outflow of lymph or blood or secretion.

The various SPECIFIC MATTERS which effect contagion in the living body, the respective 'contagia' of the given diseases, seem all to have in common this one characteristic: that in appropriate media (among which must evidently be counted any living bodily texture or fluid which they can infect) they show themselves capable of *self-multiplication*; and it is in virtue of this property that, although at the moment of their entering the body they in general do not attract notice, either as objects of sense or as causes of

bodily change, they gradually get to be recognisable in both of these respects. Now, the faculty of self-multiplication is eminently one of the characters which we call *vital*; and when it is said that all contagia are self-multiplying things, this is at least very strongly to suggest that perhaps all contagia are things endowed with life.

In order to any general consideration of the question thus suggested, contagia may conveniently (even if but provisionally) be distinguished as of two main CLASSES, differing, or at present seeming to differ, from each other in their mode of action on the organisms which they infect: one class, namely, that of *Parasites*; and the other class, that of the true or *Metabolic Contagia*. Of this separation, so far as present knowledge seems to justify it, the assumed grounds are: that each true Contagium, in proportion as it multiplies in the body, *transforms*, in a way which is specific to itself, and is different from the ways of other contagia, the bodily material with which it has contact; while, on the contrary, the Parasite, however much it may grow or multiply in the body, produces no qualitative effects specific to itself, but only such effects as are of common kind to it and all other parasites—indications, namely, of its *mechanical intrusiveness* in the parts which it occupies, and sometimes of the *drain* which it makes on its host's general nutritive resources.

A.—Of PARASITES, in relation to processes of contagion, little needs be said in the present article. When an organism or part of it is, in greater or less amount, inhabited by other organisms, animal or vegetable, which subsist on it, or its food or refuse, it of course may be a centre of infection to other (if susceptible) bodies or parts, to which it can transmit live parasites or their germs or seeds: for, when this transmission takes place, growth and self-multiplication, as in a colony, are the natural results which have to be expected; and in proportion as these occur, the newly-infected body or part gradually gets to suffer, like the old, from those particular derangements which make the type of parasitic disease. Some parasitic diseases, especially some of those of the skin, spread actively by direct contagion in ordinary intercourse; as, for instance, scabies by the migration of its acari, and porrigo (among children) by the spores of its microphyte; and the spreading of such diseases where they exist may of course be to any extent facilitated by aggregation of persons and uncleanliness of personal habits. There are cases in which parasitic disease spreads from animal to animal only in proportion as the one feeds on the other, and eats it with parasites still living in it; or in proportion as live parasite-eggs or larvae, discharged from the body of one animal, get conveyed with food

(especially on raw herbage and in water) into the bowels of another. Considerable epidemics of trichiniasis in the human subject have been traced, chiefly in Germany, to infection from the pig; in cases where pork, abounding with trichinæ, has been eaten, as sausage-meat or otherwise, in a raw or imperfectly-cooked state: and in Iceland the very great sufferings of the human subject and the cattle from echinococcus have been traced to the influence of the dogs in spreading contagion from the slaughter-house, where they eat hydatidised offal, to the kitchen-gardens and water-sources and pastures, where they discharge tape-worm eggs from their bowels. *See* PORRIGO; SCABIES; ENTOZOA.

The DISEASED STATES which consist in being colonised by parasites are diseases of indefinite duration, tending in some cases to indefinite increase. In cases where the disease consists in the presence of swarms of blood-sucking or otherwise exhausting animal parasites, symptoms of the blood-drain will of course gradually arise; but otherwise the parasite, whether animal or vegetable, operates only as a mechanical presence. Skin and mucous membrane will be irritated in their superficial layers, and in some cases more deeply, by the animal or vegetable parasites which breed on or in them, just as they might be by dead mechanical irritants: solid organs having cystic entozoa in them will in like manner show evidence of irritation by encapsulating the colonists; and the surrounding tissue will of course suffer compression and displacement in proportion as the colonisation (*e.g.* in case of echinococcus) is compact and massive. In the case of trichiniasis—but, in our ordinary experience, in no other—the multiplication of the parasites, the burrowing of their young, and the general diffusion of these in the body, are processes of such extreme activity that, if the quantity of contagium taken into the stomach has been large, the innumerable local irritations suffice to make a very acute fever; but even in this extreme case, the merely irritative type, though exaggerated, is essentially preserved.

As different sorts of animals are notoriously liable to different sorts of parasites, so, even among animals of one sort—as, for instance, in the human kind—the LIABILITY of different bodies to receive particular parasite-infections, does not seem to be quite equal for all. Especially, the vegetable parasites seem to have their affinities determined or modified by the general state of health of the recipient; and there are cases in which it looks as if there ran in particular family-lines (perhaps with some slight chemical idiosyncrasy) a special liability to particular intestinal worms. There, however, is no reason to believe that in regard of the more important animal parasites, as particularly of

trichina and the tæniadæ, the susceptibility of individuals to attacks is other than universal and practically equal.

B.—The true or METABOLIC CONTAGIA (to which the rest of the present article will exclusively refer)—the contagia which, in their respective and specific ways, operate *transformingly* on the live bodily material which they affect, are perhaps the most important of all the incidental physical influences which concern mankind. Whether they may all, at some time hereafter, admit of being named, like the parasitic contagia, in terms of biological classification, is a question which needs not in the first instance be raised; for meanwhile the identity of each separate true contagium is settled in experimental and clinical observation by the uniformity of the operation of each on any given animal body which it affects. Each of the diseases propagates itself in its own form in as exact identity as if it were a species in zoology or botany; and in each such repetition of the disease there is a multiplication—always a large, and sometimes an inconceivably immense multiplication—of material which has the same infective property. Evidences innumerable to that effect are under daily clinical observation in this climate in instances of smallpox, measles, scarlatina, whooping-cough, enteric fever, mumps, typhus, syphilis, cowpox, diphtheria, erysipelas, hospital gangrene, purulent ophthalmia and gonorrhœa, venereal soft-chancere and phagedæna, &c.; for, barring fallacies, no man ever sees any one of those diseases produced by the contagium of any other of them; and any man who has before him a case of any of them can see that, however minute may have been the quantity of contagium by which the disease was started, the patient's diseased body (part or whole) yields for the time an indefinitely large supply of the specific agent. It is more or less habitual to some of the diseases that the infectedness of the patient is first made known to the observer by such *general pyrexia* as tells of change already far advanced in the circulating mass of blood; and it is only after this has shown itself, that other symptoms, adding themselves to the fever, complete the more or less complex type which establishes the identity of the disease. But in many of the diseases it may be the case (either naturally or as the result of experimental infection) that the first, and in some diseases the main or even the only, effects of the contagion are *local changes*, passing where we can from the first observe them; and the broad facts of metabolic infection, as regards waste of bodily material with concurrent increase of contagium, are, in many such cases, among our most familiar experiences. Most instructive, too, are the facts of contagion which are to be learnt in the study of tubercle: the contagium, introducible either by tubercular and

certain septic inoculations through the skin, or internally by the infective action of the milk or diseased organs of tubercular animals if taken as food; and the contagium, when introduced, gradually spreading as it multiplies, and as lymph and blood carry it from the first infection-spot to other organs which now will repeat the process. And similarly in cancer (though the primary disease is at present of unknown origin and cannot be created by experiment) the repetition of the primary disease in secondary and tertiary propagations in the body of the sufferer is one of the most striking of all evidences of contagion; because of the great number of structural types which pass under the name of cancer, and the fidelity with which each of them is reproduced in the organ to which the contagion extends. A further fact of contagion, deserving notice in the present context, is the local spread of certain of these processes by continuity of tissue; as, for instance, in the continuous extension of phagedæna or hospital-gangrene from any centre of first inoculation, or of tubercular softening or cancer at the place where it begins: a mode of extension which indicates successive infective actions of matter on matter in spheres of ever-widening circumference;<sup>1</sup> and the like of which, but in rudimentary degree, may be traced in the areola of any acute inflammation.

In the physiology of the metabolic contagia no facts are more characteristic or more important than those which show the RELATIVENESS of particular contagia to PARTICULAR RECEPTIVITIES of body.—First, and in intimate connexion, as would seem, with a *chemical electiveness* of action which will presently be imputed to contagia, there is the preference which some particular contagia (however introduced into the system) show for particular *organs* of the body; so that by the exercise of this preference, there is given to each of the diseases its own set of clinical and anatomical characters. Compare, as instances in this point of view, the respective local affinities of smallpox, enteric fever, mumps, syphilis, hydrophobia, &c.—Secondly, it may be noted that, in regard to some of the contagia, different *persons*, and particularly persons of different *family-stocks*, show original differences of susceptibility; original, namely, as distinguished from others, hereafter to be mentioned, which are acquired; so that, for instance, the severity with which scarlatina or diphtheria will strike in particular families contrasts with a comparative mildness of the same disease in other families, or perhaps even with cases of apparently complete personal immunity under exposure to the particular danger: and recent researches have seemed to suggest as possible that, in the

very wide differences of degree with which tubercular disease prevails in different families, an essential condition may be, that the families have widely different degrees of original predisposition towards some of the septic contagia.—Thirdly, there is the extremely suggestive fact with regard to many of our best-known febrilising contagia, that they run a course of *definite duration*, and that in this course, provided the patient do not die, all present, perhaps all future, *susceptibility to the particular contagium is utterly exhausted from the patient*; so that re-introduction of the same contagium will no more renew that patient's disease than yeast will excite a new alcoholic fermentation in any previously well-fermented bread or wine. The inference from this fact seems unavoidable, that each such contagium operates with a chemical distinctiveness of elective affinity on some special ingredient or ingredients of the body; and that exhausting this particular material in febrile process, which necessarily ends when the exhaustion is complete, is the bodily change which the contagium 'specifically' performs.—Of not all metabolic contagia, however, can it be said that their operation runs so definite and self-completing a course. For, first, there are particular *acute infections* which, as a rule, kill; either (as appears to be the case in splenic fever when affecting man) because of the extreme magnitude of the transforming process which the contagium sets up, or else (as appears to be the case in hydrophobia) because the elective incidence of the contagium is on an organ indispensable to life; so that in such cases there is in fact hardly such an event as passing alive through the whole process of the disease. And, secondly, there are the *contagious dyscrasies* which are clearly characterised by their tendency to indefinite duration: syphilis, which oftener than not relapses in successive outbreaks, and often as years pass invades the body more and more deeply, and may after all never during life be ended; and tubercle and cancer, which, with almost invariable persistence, will in general steadily advance month by month to infect more and more of the body till the process eventuates in death.

The transmission of various contagious diseases IN COMMUNITIES is of course greatly influenced, both in detail and in aggregate, by such differences of individual receptivity as were mentioned in the last section. Notably, as regards communities through which particular acute infections have had full run, fresh sparks of the contagium may find little or no fuel on which to act; and much new diffusion of the disease may not again be possible, till immigration, or births, or lapse of time operating in other ways, shall have reconstituted a susceptible population. And, given the susceptible population,

<sup>1</sup> Compare Tennyson's

'little pitted speck in garnered fruit,  
Which, rotting inward, slowly moulders all.'

circumstances of time and place are infinitely various (especially as regards quantity and quickness of personal or quasi-personal intercourse) in determining how far this population shall have particular contagia thrown in its way.

Also there are conditions, not primarily of a personal kind, which operate on a very large scale in determining the spread of some of the metabolic infections: giving to them respectively *at certain times*, in ways not hitherto understood, a *special increment of spreading-power*, and in some instances also *special malignity*: and thus enabling them respectively from time to time to come into comparative prominence in national life, and perhaps at once or successively in many different countries, in the form of so-called EPIDEMICS. Thus, it is matter of familiar knowledge that the fevers which are most habitual to this country—scarlatina, measles, smallpox, enteric fever—are of nothing like uniform prevalence; that scarlatina, for instance, will be three times as fatal in one year as in another, and that smallpox is liable to even greater exacerbations: and it is known that temporary differences of this kind are not exclusively local; that, for instance (to quote a late official report), ‘the epidemic of smallpox which began in England towards the close of 1870 and terminated in the second quarter of 1873 was part of a general epidemic outbreak of that disease, of worldwide diffusion, marked wherever it occurred by an intensity and malignity unequalled by any previous epidemic of the disease within living memory.’ The wider the survey which we take of epidemiology, the more certain it becomes to us, that, outside the conditions which are independently personal or local, there are *cosmical conditions* which have to be considered. Doubtless there are great epidemiological facts—such, for instance, as the first spreading of smallpox to America, or in our own times the increasing frequency of Asiatic cholera in Europe, which may be ascribed to novel conditions of international intercourse; but there are others, equally great, to which apparently no such explanation can be applied. For what reason it is that cholera every few years has its definite fit of extension in India,—or why diphtheria, which scarcely had a place in history till it overran Europe in the 16th century, and which since then had been rarely spoken of, began again some twenty-odd years ago to be comparatively important in England,—or why the plague of the Levant has for the last two centuries been so unfamiliar to us,—or why the yellow fever of the Mississippi has in particular years raged furiously in parts of Europe,—or why our black-death of the 14th century, though apparently still surviving in India, has never but that once been in Europe,—or whither has gone our sweating-sickness of three centuries ago,—or whence have

come the modern epidemics of cerebro-spinal meningitis: these, and many like questions, which cannot at present be answered, seem to be evidence enough that, in the making of epidemics, contagion and personal susceptibility may be factors in a partly *conditional* sense. Influences which are called ‘atmospheric’—the various direct and indirect influences which attach to the normal succession and occasional abnormality of seasons, in respect of the insolation of our planet, and of the temperature and humidity of air and earth—are in general far too vaguely regarded as elements of interest in the present question, but are possible factors which no epidemiologist should omit from scientific consideration. For any definite knowledge which exists on the relation of particular conditions of season to the prevalence of particular epidemics, the reader is referred to the article EPIDEMICS, and to articles on the respective diseases.

In the PASSAGE of the metabolic contagia FROM PERSON TO PERSON various agencies may be instrumental,—bedding or clothing or towels which have been used by the sick, dirty hands, dirty instruments or other utensils, the washerwoman’s basket, foul water-supply, stinking house-drains, contaminated milk or other food, the common atmosphere, &c.; but differences of that sort are only differences as to the *means by which such communication is established with a diseased body* as brings its products into relation with healthy persons; and the disengagement of *infectious products* from the bodies of the sick is pathologically the one influential fact. As regards the products which ought to be deemed infectious, the specially-diseased surfaces and organs of the patient, and the discharges and exhalations which they respectively yield, must always be regarded with chief suspicion; but suspicion, however much it may insist on them, must never disregard other sources of danger. Of some of the metabolic contagia we practically know, and of many of the others we may by analogy feel sure, that, when a given body is possessed by one of them, no product of that body can be warranted as safe not to convey the infection. Presumption against every part and product of the diseased body is by everyone readily admitted where there are vehement general symptoms of disease: but it is important to know that not only in such febrile states, but even in states of chronic dyscrasy, and even at times when the dyscrasy may be giving no strong outward sign, the infected body may be variously infective. Thus, in regard to constitutional syphilis, it is certain that the mere utero-catarrhal discharge of the syphilitic woman, or the sperm of the syphilitic man, or the vaccine lymph of the syphilitic infant, may possibly contain the syphilitic contagium in full vigour, even at moments when the patient, who thus

shows himself infective, has not on his own person any prominent outward activity of syphilis. Similarly, in regard to tubercular disease, experiment has proved beyond question that the milk of animals suffering from tubercle will, if taken as food by other animals, infect them through the intestinal mucous membrane; and there are independent reasons for believing that the tubercular contagium (like the syphilitic) will at times during the dyscrasy be contained in the seminal fluid, and that men, tubercular perhaps only in some degree which is not immediately important to themselves, may by that secretion convey fatal infection to women with whom they have conjugal relations.<sup>1</sup> Regarding many of the metabolic contagia, conclusive evidence exists that, when they are in operation in pregnant women, the fetus will in general be infected by them; and this though the diseases (*e.g.* smallpox, cholera, syphilis) be of the most different pathological types: but with regard to pregnant animals affected with splenic fever it is noticeable that Brauell, in his extensive researches, found the blood of the fœtus not to be infective.

In general, each contagium has its own favourite WAY OR WAYS OF ENTERING THE BODY; and these preferences are not only of speculative interest, as attaching to varieties of nature and natural habits among the contagia, but are of obvious practical importance as measures of the widely different degrees in which the different contagia are qualified to spread in communities. Thus, *inoculation at broken surfaces* of skin or mucous membrane has long been known as the ordinary mode by which the infections of syphilis, hydrophobia, splenic fever, cowpox, and farcy or glanders, get admission to the body; and our best knowledge of some other infectious diseases (notably of tubercle) has been derived from inoculations intentionally made with their contagia for purposes of study. While probably all infections which tend to be of general action on the body can be brought into action in that way, and while some infections are not known to pass by any other mode of transmission, there are many infections which spread freely from subject to subject by *atmospheric and dietetic communication*; and the meaning of these preferences is hitherto not fully known. It seems that some contagia are so acted upon by air and water, that they seldom or never reach the body in an effective state by those common means of communication,—some hardly, if at all, by water, and some not by air except with very close intercourse; and further, that, of contagia which reach the body in an effective state, some require, while others do not require, that an abnormal breach of surface shall give them special opportunity for taking hold. In some of the cases where a disease

can be propagated in both ways,—*i.e.* certainly in smallpox, and apparently also in bovine pleuro-pneumonia, the artificially inoculated disease tends to be much milder than the disease otherwise contracted; but pathologically it is difficult to conceive any essential difference between those different modes of contagion. It may be presumed that, in the modes which are not by true inoculation, acts which are comparable to inoculation take place on internal surfaces; that, for instance, when particles of scarlatina-contagium are caught in the tonsils, or inhaled into the bronchi, or swallowed into the stomach, they begin by penetrating the texture of the mucous membrane, and by thus effecting as real an inoculation, with regard to the blood, as that which art or accident provides in other cases through the punctured skin. That previous abnormal breach of surface by artificial puncture or otherwise is not necessary to allow the infection of mucous surfaces is illustrated in ophthalmia and gonorrhœa; where apparently no other condition has to be fulfilled than that a particle of the blenorrhagic contagium shall be deposited on the natural surface of the mucous membrane. It deserves notice that, while a considerable number of the worst diseases of the domestic animals admit of being communicated to man by artificial inoculation, atmospheric communication seems to be very inapt, if not absolutely unable, to infect man with any one of them; and in this connexion it may be of interest to remember that syphilis, one of the most familiar of human infections, but hitherto not traced to any brute ancestry, differs from our other current infections in requiring inoculation to transmit it.

When any metabolic contagium enters the animal body, it requires an INTERVAL OF TIME, and in most cases a considerable interval, before its morbid effects can become manifest even to skilled observation. The period of latency or so-called INCUBATION varies greatly in different cases. In hydrophobia it is very rarely less than of one month, is certainly often of several months, and is said to be sometimes of years. In syphilis the inoculated spot remains generally for at least a fortnight, and may remain even as much as five weeks, without any ostensible change; and the roseola of the general infection will not be seen till some weeks later, when generally at least three months will have elapsed since the first inoculation. In the acute eruptive fevers, when their contagium is transmitted by air, the first changes which ensue on infection are not external, and we cannot be sure what early internal changes may take place; but in smallpox, the fever (which is the first overt sign) does not attract notice till about the twelfth day after infection, nor the eruption till two days later; and in measles the incubation-time, though per-

<sup>1</sup> See Dr. Weber, in *Clin. Soc. Trans.*, 1874.

haps less uniform, seems to be little (if any) shorter than that of smallpox. The septic contagia and the contagium of splenic fever seem to be of particularly quick operation; but even the most virulent septic contagium, when without admixtures which tend to complicate its action, will not begin sensibly to derange the infected animal till at least several hours after it has been inoculated. As regards the contagia last referred to, it is conceivable that the self-multiplication of the contagium in the form in which it proves fatal to life is a process which goes on continuously and uniformly from the moment of inoculation to the moment of death, and that the moment when signs of general derangement become manifest is the moment when this uniformly-advancing process has accumulated in the system a certain quantity of result:—but it does not seem easy to apply this explanation to the diseases of long incubatory period; and we can hardly conjecture what may be the latent processes—for instance of smallpox, during the first ten or more days after contagium has been received.

It is not yet possible to say, in any universal sense, with regard to the metabolic contagia, what is the ESSENTIAL CONSTITUTION of 'contagious matter,' or what the INTIMATE NATURE of the 'transforming power' which the particle of such matter exercises on the particles which it infects.—As regards the question of the FORCE, chemists, when they refer in general terms to the various acts which they designate *acts of fermentation*, allege that certain processes of change in certain sorts of organic matter induce characteristic changes in certain other sorts of organic matter, not by the common chemical way of double decomposition with reciprocally new combinations, but (so to speak) as a mere by-play or collateral vibration-effect of the chemical force which is in movement; and though language can hardly be more vague than this for any scientific purpose, it expresses clearly enough the conviction of experts that a certain great force in nature lies beyond their power even of definite nomenclature, much more of exact identification and measurement. In that most interesting, but most difficult and hitherto almost uninvestigated, branch of chemical dynamics, we are supposed to have our nearest clue to the scientific problems of the present subject-matter. It may be conceded that the 'contact-influences' which are dimly recognised as causing the fermentary changes of dead organic matter have apparent analogues in many of the morbid influences of contagion: for the changes which chemists call 'fermentary' are all catalytic or disintegrative of the organic compounds which they affect; and when living protoplasm is brought by contagion into processes of *characteristic decay*, the analogy seems sufficiently close to justify the word *zymotic* in the naming of the nature of the process. But it must not

be forgotten that, among immediate effects of contagion in the living body, are cases wherein the process (so far as we can yet see) is primarily not *catalytic* or *dis-integrative*, but, on the contrary, *anaplastic* or *con-structive*. Thus, when tubercle gives rise to tubercle, whether by secondary or tertiary infection in a single diseased body, or by infection from the sick to the healthy, each new tubercle which the contagion brings into being is a *growth-product* of the texture which bears it. And similarly, when the innumerable varieties of cancerous tumour propagate themselves by contagion, each after its special type, in the bodies of the respective sufferers, it is growth, not disintegration, which we first see. It would seem that in those cases of anaplastic 'contact-influence' something far beyond the analogy of chemical fermentations must be involved; and, in view of some of them, the physiologist has to bethink himself of the analogy of that 'contact-influence' which becomes the mainspring of all normal growth and development, when the ovum receives spermatric impregnation.—As regards the ultimate ORGANIC CONSTITUTION of the several metabolic contagia—(each of them of course abstracted from accidental admixtures, and seen or conceived in the smallest and simplest units of quantity and quality in which its specific force can be embodied)—modern research seems more and more tending to show that the true unit of each metabolic contagium must either be, or must essentially include, a *specific living organism, able to multiply its kind*. For with regard to those other contagia (as we may properly call them) which spread fermentary processes in common external nature, and of which it is as clear as of the morbid contagia that they multiply themselves in proportion as they act, it seems to be established beyond reasonable doubt that the 'self-multiplication' of each of them as it acts is the self-multiplication of a specific microphyte; and that, directly or indirectly, the microphyte's self-multiplying life is the force which originates the fermentation.<sup>1</sup> This being the case in regard of those fermentations (and the more intimate nature of the process needs not just

<sup>1</sup> In regard of some of these common-world ferments, it seems pretty certain that their characteristic microphytes operate *mediately*—that is to say, by means of special zymotic matters which they respectively produce, and which when produced can be mechanically separated from them: but whether this be true of all the same class of agencies does not hitherto seem fully settled. So far as it may prove to be true for them, that the microphyte is not itself the ferment, but is the producer and evolver of the ferment, the case of these ferments would be in parallelism with that of the chylipoietic and other functional ferments which more highly organised creatures produce for the purposes of their own economy. In the latter case the distinction between the ferment-yielding live bodies (say certain gastric cells) and their not-live product (say pepsin) is already familiar.

here be discussed), it seems probable that the same is in substance true of the specific morbid changes which extrinsic contagia produce in the materials of the living body—probable, namely, that low, self-multiplying organic forms, specific in each case for the particular disease which is in question, are essential to each morbid poison; that the increase of each contagium as it acts is the characteristic self-multiplication of a *living thing*; and that *this* (however obscure may yet remain its mode of operation) is the *essential originator of change* in the affected materials of the diseased body. The fact that low organic forms of the sorts now spoken of have often, or generally, been seen in the morbid products and tissues of persons with zymotic disease, would not by itself be a proof, or nearly a proof, that the forms are causative of the morbid change: for obviously they might be mere attendants on the necrosis and decomposition of bodily material, availing themselves of the process (just as certain insects would) to feed and multiply: and in many of the cases in which micrococci have been seen in morbid material, no direct proof could be given that the meaning of their presence was more than that. There are, however, some cases in which this proof has been completely established; and though such cases are at present but few, the significance of each of them in aid of the interpretation of other cases is of the highest importance. The researches of successive able observers in regard of the splenic fever of farm-stock, and those of Dr. Klein in regard of the 'pneumo-enteritis' (as he names it) of swine, have shown that in each of these cases the microphyte which attends the disease is botanically specific; that it and its progeny can be conducted through a series of artificial cultivations apart from the animal body; and that germs thus remotely descended from a first contagium will, if living animals be inoculated with them, breed in these animals the specific disease. It is equally well known that the organisms (spirilla) which are found multiplying in the blood during the accesses of relapsing fever are botanically specific: but in regard to this disease, experimental proof has not hitherto been given that the spirilla, if separately inoculated, will infect with relapsing fever. Studies as complete as those which have been made in splenic fever and pneumo-enteritis will no doubt sooner or later be made in regard to many other of the diseases, but their progress will necessarily be slow; partly because the objects which have to be scrutinised, and to which specific characters have to be assigned, are so extremely minute, and often so similar among themselves, that none but very skilled and very patient microscopical observers are competent to pronounce on them; and partly again because the conditions of the case are such as to limit very closely the field within

which the essential experimental observations can be made. Meanwhile, however, the two diseases, regarding which the larger knowledge has been obtained, must be regarded as highly suggestive in regard of other diseases of the same pathological group, and particularly as giving importance to fragments of evidence (not by themselves conclusive) which have been gathered of late years in studies of some of these other diseases. Eminently this is true of the large family of the septic infections—including, on the one hand, erysipelas and pyæmia with its congeners, and having, on the other hand, tuberculosis intimately associated with it; and almost equally it is true of enteric fever and cholera and diphtheria, and of the smallpox of man and beast. Thus, though it would be at least premature to say of these diseases that they certainly have as their contagia *microphytes respectively specific* to them, it seems at present not too much to say that probably such will be found the case; and if as much may not yet be said of many other diseases which are due to metabolic contagia, it must be remembered that the right lines of study relating to contagia in this point of view have not till within very recent times been opened. See Postscript, 1.

Of the NATURAL HISTORY of the contagia, considered independently of the part which they play in the living body, there are hitherto only the beginnings of knowledge. The absolutely first origin of contagia may perhaps not be more within reach of scientific research than the absolutely first origin of dog or cat; but their nearer antecedents—the states out of which they come when first about to act on the living body, and generally the variations which they and the common ferments exhibit under natural and artificial changes of circumstance, are within easy reach of investigation; and those humbler studies are likely to give very useful results. For some of our cases we seem to have an instructive analogy in the facts which Professor Mosler has put together in explanation of the blue-milk contagium of dairies—facts showing that the omnipresent penicillium glaucum, if its spores happen to alight in particular (morbid) sorts of milk, will operate distinctively on their casein as an anilin-making ferment, rendering the milk blue and poisonous, and imparting to each drop of it the power to infect with a like zymosis any normal milk to which it may be added.<sup>1</sup> In our own more special field, pathologists have already learnt that certain of the so-called 'morbid poisons'—the contagia of erysipelas, pyæmia, and tuberculosis, are intimately related to the common ferment or ferments of *putrefaction*; and that the most vehement of these contagia can be developed by the artificial culture of successive transmissions in the living body from the comparatively mild

<sup>1</sup> Virch. Arch., vol. xliii

contagium of any *common inflammatory* process.<sup>1</sup>

Two other directions suggest themselves as likely to lead to fields of useful observation and experiment. On the one hand, in *comparative pathology*, and with the tracing of contagion from animal to animal, there is the possibility that at last some lower and relatively worthless order of animals may be found the starting-ground of fatal infections for higher orders; and this, perhaps, by contagia which in their former relations are of mere inflammatory significance. On the other hand, in *geographical pathology*, and with the tracing of contagion from place to place, local centres of contagium-origination may possibly be found, in which the contagium, before it enters the animal body, will show itself an independent microphyte of the earth, first operating on the animal body as the essential force in a local malaria. Some of the worst pestilences known to the human race—yellow fever, cholera, perhaps plague, and also some of the diseases of cattle, have in their history facts which suggest that sort of interpretation: the supposition, namely, that certain microphytes are capable of thriving equally (though perhaps in different forms) either without or within the animal body; now fructifying in soil or water of appropriate quality, and now the self-multiplying contagium of a bodily disease. In regard to our own common ague-poison there seems every reason to suspect that its relation to soil is that of a microphyte; and though we know ague only as practically a non-contagious disease, we do not know that any little transfusion of blood from sick to healthy would not show it to be (in that way) communicable from person to person. See Postscript, 2.

It needs hardly be said that exact scientific knowledge of the contagia, and of their respective modes of operation, is of supreme importance to the PREVENTION OF DISEASE. With even such knowledge of them as already exists, diseases which have in past times been most murderous of mankind and the domestic animals can, if the knowledge be duly applied, be kept comparatively, or absolutely, in subjection; and the fact that at the present time fully a fifth part of the annual mortality of the population of England is due to epidemics of contagious disease is only because of the very imperfect application hitherto made of that knowledge. In the present article it is not necessary to state in detail the practice which ought to be adopted in the various different cases of infectious disease; but

<sup>1</sup> See particularly Professor Sanderson's papers in successive yearly volumes of *Reports of the Medical Officer of the Privy Council* from 1868 to 1877. It concerns the second fact mentioned in the text to remember that apparently every 'common inflammatory process' includes more or less of textural changes which are necrotic and of septic tendency. See *Holmes's System of Surgery*, first edition, article 'Inflammation.'

briefly it may be said that one principle is at the root of all such practice, whatever the disease to which it relates. This principle, which of course becomes more and more important in proportion as the infection is dangerous, and as the persons whom it would endanger are many, is the principle of *thoroughly effective separation* between the sick and the healthy: a separation which, so far as the nature of the disease requires, must regard not only the personal presence of the sick, but equally all the various ways, direct and indirect, by which infective matters from that presence may pass into operation on others. Especially as regards the diseases which make serious epidemics, the principle of isolation is not carried into effect unless due care be taken to thoroughly disinfect in detail all infective discharges from the sick, and all clothing and bedding and towels and like things which such discharges may have imbued, and finally, as regards certain contagia, the rooms in which the cases have been treated; and in order to secure these objects, it is essential in all grave cases to make such nursing-arrangements and such arrangements of the sick-room (whether private or in hospital) that no retention or dissemination of infectious matters will escape notice. It is likewise essential that all who attend on the sick should be careful not to carry contagion to other persons; as they may but too easily do, particularly in scarlatina and in certain traumatic and puerperal infections, if they omit to take special precautions against the danger. See articles QUARANTINE; DISINFECTION; and PUBLIC HEALTH; and those on the special diseases. See also Postscript, 3.

The SOCIAL CONDITIONS through which, in our own country at the present time, the more fatal infectious diseases are enabled to acquire *epidemic diffusion* are chiefly such as the following:—that persons first sick in families and districts, instead of being isolated from the healthy, and treated with special regard to their powers of spreading infection, are often left to take their chance in all such respects; so that, especially in poor neighbourhoods, where houses are often in several holdings, and where always there is much intermingling of population, a first case, if not at once removed to a special establishment, will almost of necessity give occasion to many other cases to follow;—that persons with infectious disease, especially in cases of slight or incipient attack, and of incomplete recovery, mingle freely with others in work-places and amusement-places of common resort, and, if children, especially in day-schools; and that such persons travel freely with other persons from place to place in public conveyances;—that often, on occasions when boarding-schools have infectious disease getting the ascendant in them, the schools are broken up for the time, and scholars,

incubating or perhaps beginning to show infection, are sent away to their respective, perhaps distant, homes;—that keepers of lodging-houses often receive lodgers into rooms and beds which have recently been occupied by persons with infectious disease and have not been disinfected;—that persons in various branches of business relating to dress (male and female) and to furniture, if they happen to have infectious disease, such as scarlatina or smallpox, on their premises, probably often spread infection to their customers by previous carelessness as to the articles which they send home to them; and that laundries further illustrate this sort of danger by carelessness in regard to infected things which they receive to wash;—that purveyors of certain sorts of food, if they happen to have infectious disease on their premises, by carelessness spread infection to their customers;—that streams and wells with sewage and other filth escaping into them are most dangerous means of infection, especially as regards enteric fever and cholera;—and that great purveyors of public water-supplies, so far as they use insufficient precautions to ensure the freedom of their water from such risks of infectious pollution, represent in this respect an enormous public danger;—that ill-conditioned sewers and house-drains, and cesspools receiving infectious matters, greatly contribute to disseminate contagia, often into houses in the same system of drainage, and often by leakage into wells. Of the dangers here enumerated, there is perhaps none against which the law of England does not purport in some degree to provide. At present, however, they all are, to an immense extent, left in uncontrolled operation; partly because the law is inadequate, and partly because local administrators of the law often give little care to the matter; but chiefly because that strong influence of national opinion which controls both law and administration cannot really be effective until the time when right knowledge of the subject shall be generally distributed among the people, and when the masses whom epidemics affect shall appreciate their own great interest in preventing them.

Whenever that time shall come, probably the public good will be seen to require, with regard to every serious infectious disease which is apt to become epidemic, that the PRINCIPLES which ought to be accepted in a really practical sense, and to be embodied in effective LAW, are somewhat as follows:—(1) that each case of disease is a public danger, against which the public, as represented by its local sanitary authorities, is entitled to be warned by proper information; (2) that every man who in his own person, or in that of anyone under his charge, is the subject of such disease, or is in control of circumstances relating to it, is, in common duty towards his neighbours, bound to take every care which

he can against the spreading of the infection; that so far as he would not of his own accord do this duty, his neighbours ought to have ample and ready means of compelling him; and that he should be responsible for giving to the local sanitary authority proper notification of his case, in order that the authority may, as far as needful, satisfy itself as to the sufficiency of his precautions; (3) that so far as he may from ignorance not understand the scope of his precautionary duties, or may from poverty or other circumstances be unable to fulfil them, the common interest is to give him liberally out of the common stock such guidance and such effectual help as may be wanting; (4) that so far as he is voluntarily in default of his duty, he should not only be punishable by penalty as for an act of nuisance, but should be liable to pay pecuniary damages for whatever harm he occasions to others; (5) that all who are in attendance on any case of infectious disease should be similarly bound to use every proper care and precaution not to carry the infection to any other person; (6) that the various commercial undertakings which in certain contingencies may be specially instrumental in the spreading of infection—water-companies, dairies, laundries, boarding-schools and day-schools, lodging-houses, inns, &c., should respectively be subject to special rule and visitation in regard of the special dangers which they may occasion; and that the persons in authority in them should be held to strict account for whatever injury may be caused through neglect of rule; (7) finally, that every local sanitary authority should always have at command, for the use of its district, such hospital accommodation for the sick, such means for their conveyance, such mortuary, such disinfection-establishment, and generally such planned arrangements and skilled service, as may, in case of need, suffice for all probable requirements of the district. *See* Postscript, 4.

Persons who are imperfectly acquainted with the scientific and social facts relating to the present subject-matter, or who have never seriously considered them, may think it would be over-sanguine to expect any general recognition of principles so peremptory as the above may at first appear to them; but, if so thinking, they would perhaps have under-estimated the rapidity with which knowledge is now increasing as to the common interests and mutual duties of mankind in respect of dangerous infectious disease. In the year 1865, when the so-called cattle-plague or steppe-murrain was imported afresh, as a long-forgotten disease, into this country, and was found to affect very large pecuniary interests, primarily of the chief landowners of the United Kingdom, and secondarily of other classes, an immensely valuable stimulus was given to the education of the country, and especially of its Legislature, in regard to

the preventibility of the infectious diseases. And the remarkable zeal and ability which from that time onward have been shown, in providing adequate laws and admirable administrative arrangements against the diffusion of steppe-murrain and other infectious diseases of Farm-Stock, are not likely to be found permanently absent in relation to the interests of Human Life, when once the true bearings of the subject shall have got to be popularly understood. JOHN SIMON.

POSTSCRIPT.—In revising, for the reprint of the Dictionary of Medicine, the above article, written in 1878, the writer has been able to leave the original text almost entirely without change, but has to add the following few remarks referring to steps of later progress.

1. First, with regard to the exact science of Contagion, knowledge of microphyte-life as the power of the metabolic contagia has during the past twelve years advanced considerably both in extent and in minute accuracy, and is rapidly tending to consolidate itself into general law. Work of especial value in that line has been done by Dr. Klein in respect of scarlatina and diphtheria, and also of the foot-and-mouth disease of farm-stock.

2. And with regard to the question of cases in which perhaps the microphytes of given diseases, before infecting the human body, may have multiplied in soils or waters of appropriate quality, an extremely interesting line of research has been opened by Dr. Ballard in his study of the ætiological relations of summer diarrhœa.

3. With regard to the proper action of Local Authorities against the spreading of dangerous contagions in their districts, it has to be noticed that, in 1889, an important step towards supplying the first requisite for such action was represented by the passing of an Act for the compulsory local Notification of cases of Infectious Disease (52 & 53 Vict., cap. 72).

4. Within the last few years, by the admirable studies of Mr. Power and Dr. Klein, it has been made almost certain that scarlatina and diphtheria are diseases common to man with at least some of the animals which are most intimately in his service: both of them appearing to be capable of affecting bovine animals, and to be communicable by means of cows' milk, while diphtheria appears also to be a disease which spreads among cats: and, with this extremely important addition to knowledge which previously existed, the consequence is now strongly suggesting itself that systems of law and administration intended to guard human society against dangerous infections of disease, must extend to the provision of adequate means for observing and controlling the infectious diseases of domestic and farm-yard animals, and for securing that the public health shall not be jeopardised through the negligent or wilful distribution of infected milk. J. S.

CONTINUED FEVERS.—CHARACTERS.—Under the name of Continued Fevers is included a group of diseases which have the following characters in common:—

1. They are attended with *pyrexia*, or a febrile condition sustained for a more or less definite period of considerable duration, without intermission or very decided remission, and not due to any local inflammation. That is, the fever is *essential*, and not merely *symptomatic*. The distinguishing feature of pyrexia is unnatural elevation of the temperature of the body; but there are other symptoms scarcely less constant—increased frequency of the pulse, thirst, loss of appetite, furred tongue, headache, chilliness, and—if the temperature is high—various manifestations of disturbance of the nervous system.

2. They are clearly due to the introduction into the body of a *poison from without*, and this poison is *reproduced in the system*, so that continued fevers are communicable directly or indirectly from the sufferer to others. This statement would not apply to simple continued fever so called; but simple continued fever, when not a mild or abortive attack of one or other of the specific fevers, has scarcely anything in common with them.

3. The continued fevers rarely affect the same individual twice. An attack is *protective against subsequent attacks* of the same fever. This is much less manifest in relapsing than in typhus and enteric fever.

4. The continued fevers have a more or less *definite duration*. A certain time intervenes between the exposure to the poison and the onset of the disease, which is called the period of incubation; and the disease is divisible into the stages of invasion, dominance, and decline.

5. In two out of three continued fevers there is a characteristic *cutaneous eruption*.

ENUMERATION.—The continued fevers are typhus, enteric, and relapsing fever. Common continued fever, or febricula, often associated with them for the sake of convenience, resembles them only as consisting in pyrexia not traceable to any known local inflammation. It does not conform to the characteristics enumerated, and cannot be brought within any definition which applies to the true fevers.

DIAGNOSIS.—The continued fevers have to be distinguished from the intermittent and remittent fevers on the one hand, and from the eruptive fevers and some other diseases on the other.

1. From *intermittent* and *remittent* fevers they are distinguished clinically by the comparatively sustained high temperature; but were this all, the continuous character sometimes assumed by intermittents and the remittent type occasionally seen in enteric fever—especially in children—would bring them close together. The essential distinction is that indicated under the second head, and is

mainly ætiological. Both kinds of fever are due to a poison received from without; but while in continued fevers the source of the poison is for the most part a previous case of fever of the same kind, and the poison is generated anew in the subject of the disease, remittents and intermittents are of malarious origin, and the poison is never reproduced in the system, and therefore never communicated by the sufferer.

The formation of the specific contagium of continued fevers within the system during the disease is of course the cause of their spread by contagion. The mode of this spread is different for the different fevers. Typhus and relapsing fever are directly contagious in an eminent degree; the poison is contained in the emanations from the skin or lungs, and is capable of entering the blood of healthy persons by being breathed or swallowed; it may also be carried by fomites. Enteric fever, if directly contagious at all, is very slightly so; the contagium is apparently not given off in the breath or perspiration, but chiefly or exclusively from the bowels, and the disease is spread mainly by the contamination of drinking-water, or, more rarely, by sewer gases or by the emanations from typhoid excreta, especially after long residence in sewers.

2. The distinctions between the continued and the *eruptive fevers* remain to be pointed out. They are of a very slight character. All the characters given of the continued fevers, including the occurrence of a cutaneous eruption, are common to them and the eruptive fevers. The differences are as follows:—

(a) The liability to the eruptive fevers is almost universal in the absence of protection by a previous attack, and is little affected by the state of health of the individual, while the liability to continued fever is very variable in different persons, and even races, and is greatly influenced by external conditions. There is no parallel in the eruptive fevers to the predisposition to typhus and relapsing fever generated by overcrowding and famine.

(b) The protective influence of a previous attack is more marked in the eruptive fevers, though not to such a degree as would constitute an important distinction. Instances of small-pox after a previous attack, or after vaccination, and second attacks of measles and scarlet fever, are not very uncommon.

(c) While in the eruptive fevers the specific poison is considered to be invariably derived from a previous case, this cannot be said with the same confidence with regard to the continued fevers. It is true that, in by far the largest proportion of attacks of typhus, enteric, and relapsing fevers, the source of the poison can be traced, and that as the experience and trained skill brought to bear on the search increase, the fewer are the examples in which it fails; but it cannot yet be said

definitely that these fevers are not generated anew under certain conditions. The constancy with which typhus and relapsing fever follow in the track of overcrowding and starvation is suggestive of spontaneous origin; but in this country typhus is never so completely extinct that foci of infection are wanting, and epidemics of relapsing fever may be imported. Enteric fever, again, appears from time to time under circumstances which appear to exclude the possibility of the poison having been derived from a previous case, though in most instances of epidemic prevalence of the disease, there is conclusive evidence of specific, and not merely general, contamination of the air or water. It is not, however, necessary to enter upon this controversy here, or to do more than allude to the question whether or not their contagia are of the nature of organic germs. *See* CONTAGION; and ZYME.

The fevers will be fully described under their respective names.

WILLIAM H. BROADEENT.

**CONTRACTION, Muscular** (*contraho*, I draw together).—A term applied to the action or to the shortening of a muscle from any cause, whether in health or in disease. *See* SPASM.

**CONTRA-INDICATION.**—Any circumstance which forbids the employment of therapeutic measures otherwise indicated.

**CONTRE-COUP** (Fr.), Counter-stroke. An injury of a part opposite to but distant from that to which force is applied, as by a fall or direct blow. Contre-coup is chiefly observed in injuries of the skull.

**CONTUSION** (*contundo*, I bruise).—A bruise or injury of the soft parts without breach of surface.

**CONVALESCENCE** (*con*, with; and *valesco*, I grow well).—The *period of convalescence* signifies that period during which a patient is progressing towards recovery, and is returning to a state of health after having suffered from an illness. When the health has been completely restored, convalescence is said to be established, and the patient is regarded as convalescent. The word is used most commonly in association with fevers, inflammatory diseases, and other acute affections. Convalescence may be ushered in by a crisis, and become speedily established; or it may be very slow and protracted in its progress, which is also often interrupted by relapses, complications, or sequelæ. Patients frequently require careful watching and judicious treatment while becoming convalescent, as they are apt to retard or even prevent their recovery, and to lay the foundation for permanent disease, by neglect of due precautions, especially as regards their diet. Much

injury is not uncommonly inflicted by the injudicious administration of medicines, and the employment of other means which are supposed to hasten convalescence; but, on the other hand, it can undoubtedly often be promoted by suitable measures.

FREDERICK T. ROBERTS.

### CONVOLUTIONS OF THE BRAIN and CORTEX CEREBRI, Lesions of.

The pathology of the cortex cerebri is still comparatively in its infancy. The older records and observations, made while the idea was still prevalent that the convulsions of the brain had no definite dispositions and relations, and that the various parts of the hemispheres were functionally equivalent, are not sufficiently exact to be made the basis of trustworthy clinical and physiological conclusions. Recent anatomical investigation into the topography and homologies of the cerebral convolutions, and the experimental researches of Hitzig, the writer, and others in reference to the results of electrical irritation of the brain, have directed greater attention to accurate topographical descriptions of the lesions of the cortex in connexion with observed clinical symptoms. Up to a comparatively recent date physicians and physiologists generally held by the views of Flourens, based on experimental investigation of the brains of the lower classes of animals. According to Flourens the hemispheres were concerned purely with intelligence—a faculty one and indivisible; and each part of the hemisphere possessed the functions of the whole, so that, if part were destroyed, functional compensation might be effected by the parts which remained. These views seemed satisfactorily to explain the cases, not uncommon, in which, notwithstanding the existence of extensive lesions in the hemispheres, no symptoms were observed during life. The frequent association of aphasia with a limited lesion of the cortex cerebri, vaguely indicated by Bouillaud and Dax, and definitely fixed by Broca at the posterior extremity of the third left frontal convolution, was a step towards localisation of function in the brain, which, however, met with much opposition and counter-facts. The clinical and pathological observations of Hughlings Jackson in reference to the causation of limited and unilateral epileptiform convulsions were an important contribution to the physiology and pathology of the cortex. These convulsions he attributed to irritative or discharging lesions of the grey matter in the neighbourhood of the corpus striatum in the opposite hemisphere.

Physiological experiment has demonstrated the correctness of the views advanced by Hughlings Jackson, and shown that, not only can movements be excited by electrical irritation of certain regions of the cortex, but also that definite combinations of muscular move-

ments uniformly result from stimulation of certain specialised areas within this region.

Physiological experiment is, as to precision in its results, considerably in advance of clinical observation, and, from the nature of the two methods, this is what might be expected.

The investigation of diseases of the brain is surrounded by special difficulties. Though, as shown by physiological experiment, the brain is capable of being mapped out into different regions possessing different functions, yet the brain acts as a whole, and it is not always easy to analyse the facts of disease, and to distinguish with certainty between the effects directly dependent on the locality of the brain, and those due to the indirect influence exerted on the functions of neighbouring regions and on the brain as a whole.

That the brain is diseased in insanity, functionally or organically, is a fact now universally admitted; but it is also true that the lesions which cause objective symptoms in the domain of motion and sensation need not cause mental derangement, and also that lesions which cause mental derangement need not manifest themselves in any discoverable disorders of sensation or motion. In fact, for purposes of ideation we have practically two brains; for, though motion and sensation will be paralysed on the opposite side by destruction of one hemisphere, yet intelligence and thought are possible through the hemisphere which remains.

Various forms of lesion have been found in the brains of the insane, including morbid conditions as to vascularity, degeneration of the blood-vessels, and degeneration of the nerve-cells, neuroglia, and membranes; but no constant relation has as yet been established between any one form of degeneration and any one form of mental alienation, or between the latter and any localised lesion. Nor has it been clearly established whether the forms of degeneration found in the brains of the chronically insane are the result or the cause of the mental disorders. An exception, however, is to be made in favour of general paralysis of the insane, where there seems to be a definite connexion between the anatomical lesion and the symptoms manifested. In this disease we find as a constant, if not the only factor, a form of chronic encephalitis, affecting chiefly the cortical regions which physiological experiment has shown to be the motor zone of the hemispheres. This lesion is associated with progressive motor paralysis, varied with intercurrent epileptiform and apoplectiform seizures, and with mental symptoms characterised generally by exalted ideas and delusions as to wealth, power, and grandeur. The motor symptoms are readily accounted for by the locality and character of the cerebral lesion, but the relation between this and the mental symptoms is a

subject which psychological analysis has yet to elucidate.

The objective symptoms of cortical lesions depend on their locality, and on whether they exercise an irritative or destructive influence on the parts they invade. From the localisation point of view alone the intimate nature of the morbid process is unimportant, except

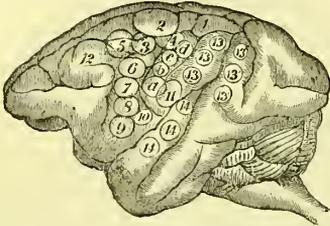


FIG. 30.—Side view of the Left Hemisphere of the Monkey. The areas have the same signification as in the next figure.

in so far as its irritative or destructive character is concerned. Lesions, such as tumours, which from their very nature exercise important indirect effects on the encephalon as a whole, apart from their effects on the regions which they directly invade, can rarely be exactly localised, owing to the difficulty of separating the direct and indirect symptoms from each other and referring each to its exact cause. Also no rigid conclusions as regards localisation can be drawn from morbid affections of the hemispheres which extend over a large area, such as the various forms of meningitis and meningo-encephalitis. In all these cases the nature of the affection must be diagnosed from its own general and special characters; its position and extent in the brain being arrived at approximately from a consideration of the effects of accurately circumscribed lesions, as determined by careful clinical and pathological observation and physiological experiment.

The brain may be considered as divided into a *motor* and a *sensory* zone.

**Motor Zone.**—The motor zone includes the convolutions bounding the fissure of Rolando, viz. the ascending frontal (pre-central), the ascending parietal (post-central), and postero-parietal lobule, the base of the three frontal convolutions, and the internal or mesial aspect of the same convolutions, or marginal gyrus. In this zone are differentiated centres for the movements of the trunk, limbs, head, and eyes, and those of the mouth, tongue, larynx, and correlated muscles. The centres of the lower limb are situated at the upper extremity of the central convolutions and in the paracentral lobule or posterior part of the marginal convolution (numerals 1 and 2, figs. 30 and 31). The centres for the foot and toes are more especially posterior to the upper end of the fissure of Rolando; those of the thigh and leg anterior to the upper extremity of the

same fissure. The centre for the arm is situated below that of the leg, and partly in front of it, and occupies the middle third, or rather two-fourths of the central convolutions (3, 4, 5, 6, 7, *a, b, c, d*, figs. 30 and 31).

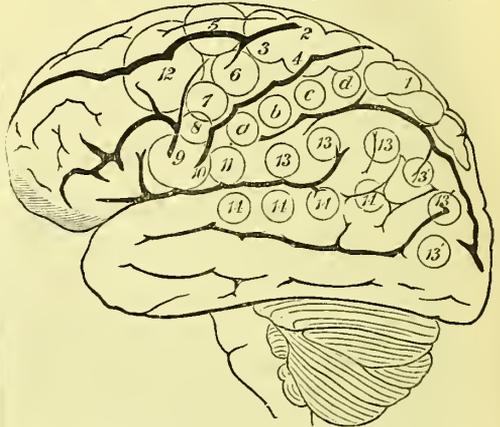


FIG. 31.—Side view of the Left Hemisphere in Man, with the areas of the cerebral convolutions. The effects of stimulation of each area, as ascertained by experiments on the brain of the monkey, are subjoined.

- 1, 2 (at the upper extremity of the fissure of Rolando). Movements of the foot and leg.
- 3, 4. Complex movements of the opposite leg and arm, and of the trunk, as in swimming.
- a, b, c, d*. Ascending parietal convolution. Individual and combined movements of the fingers and wrist of the opposite hand.
5. Posterior extremity of the superior frontal convolution. Extension forward of the opposite arm and hand.
6. Upper part of ascending frontal convolution. Supination and flexion of the opposite forearm.
7. Median portion of ascending frontal convolution. Retraction and elevation of the opposite angle of the mouth.
8. Lower part of the ascending frontal convolution. Elevation of *ala nasi* and upper lip, with depression of lower lip.
- 9 and 10. Inferior extremity of ascending frontal convolution (Broca's convolution). Movements of the mouth, tongue and larynx. Action bilateral.
11. Between the inferior extremities of the ascending frontal and the ascending parietal convolutions. Retraction of the opposite angle of the mouth; and the head turned slightly to one side.
12. Posterior portions of superior and middle frontal convolutions. Eyes opened widely; pupils dilated; head and eyes turned towards opposite side.
- 13 and 13'. Supra-marginal lobule, angular gyrus, and occipital lobe. Eyes moved towards the opposite side, with an upward (13) or a downward (13') deviation. The pupils generally contracted. (Centre of vision.)
14. Infra-marginal (superior temporo-sphenoidal) convolution. Pricking of opposite ear; head and eyes turned to opposite side; pupils largely dilated. (Centre of hearing.)

In this area may be differentiated centres for the upper arm (protraction), (5, figs. 30 and 31), and retraction (4, figs. 30 and 31); movements of the forearm, viz. flexion, supination, &c. (6, figs. 30 and 31); and those of the wrist, fingers, and thumb (*a, b, c, d*, 30 and 31).

The proximal movements of the upper extremity are represented most in the upper part of this area; the distal movements, that is those of the finger and thumb, most at the lower part. Below the arm area, and occupying the lower third of the central convolutions, is the centre for the movements of the face, mouth, and tongue and larynx, &c. (7, 8, 9, 10, 11). The upper facial movements are centralised more particularly in front of 7, 8 (figs. 30 and 31), and those of the platysma behind the fissure of Rolando (11, figs. 30 and 31). The movements of the mouth, tongue, and larynx are represented more particularly at the lower part of this area (9, 10, figs. 30 and 31). The centres for the adduction of the vocal cords are situated more particularly at the lower portion of the ascending frontal convolution, immediately posterior to the precentral sulcus. The centres for the lateral movements of the head and eyes are situated at the base of the frontal convolutions (figs. 30 and 31). The areas or centres for the head and eyes, arm and leg, extend over the margin of the hemisphere into the mesial aspect or marginal convolution. In this convolution, posterior to the centres for the head and eyes, and arm, are centralised the movements of the trunk, and, behind these, those of the leg. The prefrontal area, that is to say the region in advance of the anterior extremity of the precentral sulcus, though anatomically related to the motor tracts of the internal capsule, is a so-called non-excitable area—that is to say, it gives no outward response to electrical stimulation like the centres above described.

*Irritative lesions* of the motor zone proper, such as may be induced by syphilitic lesions, tumours, spicula of bone, depressed fractures, thickening of the membranes, &c., cause convulsions, which may remain limited to one limb, or one group of muscles, without loss of consciousness, or affect the whole of the opposite side with loss of consciousness, or become more or less bilateral with all the symptoms usually observed in so-called idiopathic epilepsies.

If the convulsive phenomena begin always in the same way, and if they frequently remain localised in one limb or one group of muscles, and especially if paralytic symptoms manifest themselves, the exact position of the lesion in the opposite hemisphere may be accurately diagnosed.

*Destructive lesions* of the motor zone cause general or limited paralysis of voluntary motion in the opposite side of the body, according as the lesion affects the whole of the motor zone, or is limited to special centres within this area. The causes of destructive lesions of the cortex may be various—hæmorrhage, laceration by wounds, &c. One of the most common causes is embolism or thrombosis of the arteries supplying the cortical motor area. These are derived from the

Sylvian artery of the middle cerebral. The cortical branches may be occluded without interfering with the circulation in the corpus striatum, which is supplied by special branches, as shown by the researches of Duret and Heubner.

When the motor zone is affected by a general destructive lesion, complete hemiplegia of the opposite side results, in all respects like that resulting from destructive lesion of the corpus striatum and anterior portion of the internal capsule. In this form of paralysis the loss of motion is most marked in those movements which are most independent; hence the arm is more paralysed than the leg or face, and the hand more paralysed than the shoulder movements of the arm. This has been accounted for by the fact, that the centres for bilateral movements are intimately associated in the lower ganglia; hence the stimulus from one hemisphere can to a certain extent call forth the action of the conjoint motor nuclei. The electrical contractility and nutrition of the muscles is not affected by paralysis of cerebral origin. The nutrition of the muscles may, however, suffer from disuse, and frequently paralyses of cortical origin are followed after a time by rigidity and contracture, with descending sclerosis of the pyramidal tracts of the crura, pons, and lateral tracts of the spinal cord. In some cases the descending degeneration invades the anterior cornua of the spinal cord, and atrophy of the paralysed muscles ensues. Occasionally from limited lesions of the cortical motor area complete hemiplegia may occur on the opposite side, for the time at least. This is to be explained by the fact that sudden establishment of a destructive lesion may cause such commotion or perturbation of the centres in general, that their functions are for the time suspended. But in such cases those centres which have been only functionally suspended again resume their functions, and the paralysis disappears except of those movements the centres of which have been permanently damaged.

Limited lesions of the motor zone cause paralysis of those movements the centres of which the lesion invades. The result is not complete hemiplegia, but a *monoplegia* or *dissociated* paralysis. Hence, from a cortical lesion we may get a paralysis of the arm, or of the arm and face, or of the leg and arm, or of the face alone, or of the lateral movements of the head and eyes. If the leg is specially paralysed, the lesion is in the upper third of the central convolution; if the arm, in the middle third of the central convolution; if the face, in the lower third of the central convolution, and we may more precisely localise the lesion in the upper or lower half of these respectively, according as the proximal or distal movements are more particularly affected. The lesion which, while circumscribed at first, causes a monoplegia, may

advance to other centres, and after a time produce general hemiplegia of the opposite side. Hemiplegia so resulting is a succession of monoplegia, and is a sure indication of cortical disease. It is to be noted that destructive lesion of the mouth-centre (Broca's region) on one side does not cause paralysis of articulation or phonation, owing to the fact that each centre has a more or less complete bilateral influence over the movements of the mouth and tongue and larynx. Destructive lesion of this centre in the left hemisphere generally gives rise to aphasia without paralysis of articulation (see APHASIA). In bilateral lesions there is both aphasia and paralysis of articulation (see Dr. Barlow's case, *Brit. Med. Journ.* 1877, vol. ii. p. 103). Limited cortical motor lesions are frequently associated with transitory rigidity of the opposite side of the body; and occasionally also with unilateral convulsions, owing to the morbid process inducing irritation of the neighbouring centres.

Unilateral destruction of the oculo-motor area causes a temporary conjugate deviation of the head and eyes to the side of lesion, a condition which is frequently seen in the early stages of hemiplegia, and it is due to the unantagonised action of the centre in the sound hemisphere. But the effects of unilateral destruction of this centre are not permanent, hence lesion of this region may appear entirely latent. Lesions of the pre-frontal region cannot with certainty be diagnosed. Not infrequently they give rise to an irritable dementia; but it is difficult to distinguish this from the general symptoms of other forms of cerebral disease, such as tumour, abscess, and the like.

Subcortical lesions of the motor zone cause symptoms not readily, if at all, distinguishable from lesions of the cortex itself. They are perhaps less frequently quite limited, owing to the close relation of the various tracts converging towards the internal capsule, though occasionally they have the character of differentiated monoplegia. Theoretically, according to the results of experiment, irritative lesions of the medullary fibres should produce only tonic, and not clonic, epileptiform spasms of the related muscular groups; but, practically, this is not a reliable test, inasmuch as these lesions are apt to cause also cortical irritation and clonic convulsions of the usual type.

In the majority of instances of paralysis from disease of the cortex sensation has been unimpaired; but, on the other hand, a number of cases have been put on record in which, with lesions of various kinds (including tumours) implicating the motor zone, there has been paralysis, not only of motion, but also of sensation in a greater or less degree. But, whereas paralysis of motion is invariably caused by truly destructive lesions of the motor area, anaesthesia is only of occasional occurrence in connexion with apparently

similar lesions. There is no relation between the extent, degree, or duration of the motor paralysis and the impairment of sensation; for there may be the most complete paralysis of motion with perfect sensibility in all its forms, cutaneous as well as muscular; and the motor paralysis may remain when the anaesthesia, if any, has entirely disappeared.

The centres of common and tactile sensibility have been shown by the writer's experiments, and those of Horsley and Schäfer, to be situated in the falciform or limbic lobe, and many of the recorded cases of anaesthesia in connexion with lesions affecting the motor zone can be shown to have directly implicated either this lobe or the sensory tracts of the internal capsule, and we may legitimately assume, even if we cannot demonstrate, a similar direct or indirect implication of these regions in the case of others. The slight degree of anaesthesia affecting the fingers more particularly which has been most frequently noted in connexion with cortical motor paralysis, is, in the writer's opinion, to be regarded as the beginning or the remnant of a general hemianæsthesia, due to direct or indirect implication of the sensory tracts.

**Sensory Zone.**—Experiments on monkeys and other animals, by the complementary methods of excitation and destruction, have shown that in the regions behind and below the motor zone there are differentiated centres for the senses of sight, hearing, common and tactile sensibility, smell, and taste.

The experiments of the writer indicate that the *visual centre* is situated in the angular gyrus and occipital lobe, or occipito-angular region; though those of Munk and Schäfer would limit this centre to the occipital lobe. Unilateral lesion of the angular gyrus, or occipital lobe, may remain latent; but if it is such as to cause extensive destruction of the medullary fibres of this region, homonymous hemianopia towards the opposite side is the result. This is particularly frequent in connexion with lesions invading the mesial aspect (cuneus) of the occipital lobe. Destructive lesions of the angular gyrus of the left hemisphere commonly cause word-blindness, and allied defects in visual ideation. Hemianopia may be caused also by lesion of the optic tract, or of the corpora geniculata. Uncomplicated hemianopia from cerebral lesion is comparatively rare. Usually it is associated with some degree of hemianæsthesia, owing to implication of the adjacent sensory tracts, or, it may be, with a greater or less degree of hemiplegia. In cerebral hemianopia, a pencil of light thrown on the anaesthetic side causes contraction of the pupils; whereas, if the lesion is in the optic tract no reaction occurs (Wilbrand's test). Irritative lesions of the angular gyrus not infrequently give rise to subjective ocular spectra, or visual hallucinations.

The *auditory centre* is situated mainly,

if not exclusively, in the superior temporal gyrus. Owing to the bilateral relations of each centre, unilateral lesion may cause no appreciable symptoms. If, however, the lesion is in the superior temporal gyrus of the left hemisphere, word-deafness is usually met with. This does not occur in connexion with lesion of any other part of the temporal lobe, provided that the superior temporal gyrus is intact. Deafness from cerebral disease is very rare. In order that this should occur, it is necessary that the superior temporal gyrus should be destroyed on both sides. There are only two cases on record of such a bilateral lesion; one reported by Shaw (*Archives of Medicine*, February, 1882), and another by Wernicke and Friedländer (*Fortsch. d. Med.*, March, 1883). In both cases total deafness was observed. In monkeys, however, complete destruction of this region on both sides does not entirely abolish sensitiveness to sonorous vibrations (*Croonian Lectures on Cerebral Localisation*, Ferrier, 1890). There are several cases on record of irritative lesions of this region causing subjective auditory sensations, or epilepsy ushered in by an auditory aura (*ibid.*).

The centre of tactile and common sensibility is situated in the falciform or limbic lobe, as above mentioned (gyrus fornicatus and gyrus hippocampi). This conclusion is founded almost entirely on the results of experiments on monkeys. Pathological evidence in favour of the localisation of the tactile centre in this region is as yet scanty.

Experiments on monkeys indicate that the centres of smell and taste are situated in close relation to each other at the lower extremity of the temporal lobe, or region of the hippocampal lobule (see *Croonian Lectures, sup. cit.*).

The anatomical relations of the olfactory tract, as well as the results of electrical stimulation, indicate that the olfactory centre is chiefly in relation with the nostril on the same side, and cases have been reported in which smell has been affected on the same side as the cerebral lesion. But the facts of cerebral hemianæsthesia, functional and organic, indicate that some at least of the olfactory tracts pass into the opposite hemisphere by way of the internal capsule. It was supposed by Meynert that the olfactory tracts decussate in the anterior commissure, but this is opposed by the anatomical and experimental investigations of von Gudden and Ganser. There are several cases on record of subjective olfactory sensation in connexion with tumours and other irritative lesions implicating the region of the uncinate gyrus in man (Jackson and Beever, *Brain*, October, 1889). The exact position of the centres of taste has not been determined either by experimental research or by clinical observation, but the evidence is in favour of their being situated in close relation to those of smell.

TREATMENT.—The principles of localisation above indicated, combined with those of antiseptic surgery, have in recent years been in several instances successfully applied to the treatment of certain forms of cerebral disease otherwise hopeless ('Cerebral Localisation in its Practical Relations,' Ferrier, *Brain*, 1889; and *ibid.*, Mills). These include traumatic cysts and extravasations, the position of which has been determined by the symptoms, as well as cases of tumour and abscess, the last more particularly in connexion with disease of the middle ear. Excisions of irritable portions of the cortex have also been undertaken for the cure of focal epilepsy; in some instances with benefit, though in others without material improvement.

Up to the month of December, 1889, eighteen cases of cerebral tumour had been operated upon. Of these, seven were successfully removed. Of the remaining eleven cases (including three cases of tumour of the cerebellum, and five of unremovable tumours, two of them operated on *in extremis*) nine have died from various causes, including septic inflammation, cerebral œdema, or shock. This gives a mortality of fifty per cent. Contrasted with the latest statistics of trephining for all causes, or of the mortality from the major operations in surgery, we have reason to regard the surgical treatment of cerebral tumour as having achieved an encouraging measure of success, and probably greater successes may be attained in future when the conditions of successful operation and after-treatment have become better known. In the absence of definite indications as to the character of the tumour, and as to whether it is situated on, in, or beneath the cortex, trephining is, in the writer's opinion, justifiable as a diagnostic measure; for if the tumour should, after all, not be removable, the risks of the operation itself are less than the evil of allowing a case to perish which the autopsy might prove to be one which could have been dealt with successfully.

D. FERRIER.

CONVULSIONS.—SYNON.: Eclampsia; Fr. *Convulsions*; Ger. *Krampf*.

DEFINITION.—This term is commonly given to more or less general, purposeless muscular contractions, occurring simultaneously and successively for a variable time—constituting a 'fit' of this or that kind. It is also, however, applied at times to certain more localised purposeless contractions, though these would be more appropriately (and are in the majority of cases) termed *spasms*. The latter, like convulsions, are of two kinds, *tonic* and *clonic*.

CLASSIFICATIONS.—Convulsions have been variously classified by different authors according to the different points of view from which they have been regarded. From the point of view of their causation, it was the

custom of older writers to recognise both a clinical and a physiological division into classes. From the former standpoint there was the division into (1) primary or essential; (2) sympathetic; and (3) symptomatic: whilst from the physiological point of view convulsions were divided into (1) centric; and (2) excentric. These classifications are altogether arbitrary and superficial, and will not stand the test of a critical examination—though the first of them is to a certain extent useful. Again, looking to the distribution of the convulsions, or to the parts involved, the classification that has been adopted by various writers may be summarised as follows:—(1) *external convulsions*—*a*, general; *b*, unilateral; *c*, partial; (2) *internal convulsions*.

These various terms need little explanation, though before referring to a more recent and more strictly scientific classification something requires to be said in regard to them.

Convulsions are termed *primary* or *essential* when they occur either without assignable cause, from mental or moral perturbations, or as a result of some local irritation. They are called *sympathetic* when the convulsions declare themselves as a prelude to, or in the course of, any of the specific fevers, as a consequence of acute pulmonary or renal affections, or in association with disordered states or structural diseases of any of the organs of the body other than those of the nervous system; while the name *symptomatic* has been applied to the convulsions which occur as a result of injury or structural disease of the nervous system itself. The unilateral convulsions which affect one half of the body only, as well as other more partial convulsions, are almost entirely confined to this latter group, though general convulsions of the symptomatic type are perhaps just as frequently met with. The so-called *internal convulsions* constitute an ill-defined group, the members of which are scarcely worthy of the name of convulsions at all. They are rather tonic or clonic spasms of particular parts. The best-known member of this group is laryngismus stridulus; but another example is a spasmodic and frequently recurring hiccup. Some authors would also include angina pectoris.

Convulsions, either tonic or clonic, or both, enter into or form the semeiological basis of six principal diseases having separate places in our nosology. These are *eclampsia*, *epilepsy*, *tetanus*, *laryngismus stridulus*, *hydrophobia*, and *chorea*. All but the first of these conditions will be considered in their respective places, so that eclampsia alone remains. But eclampsia and convulsions are convertible terms, meaning almost absolutely the same thing. The former term, indeed, is useless except for the mere purpose of literary precision. In epilepsy and in eclampsia we have equally to do with convulsions, which are now admitted

by almost all modern writers to be, so far as the form of the attack is concerned, indistinguishable from one another. The former name, however, is given to convulsions which have a known tendency to recur at variable intervals; whilst the latter has been commonly applied to convulsions which are either solitary or, if not exactly so, which occur as a closely successive cluster or group, more or less distinctly sympathetic with some general or local bodily condition. Seeing that there is, in a very large number of cases, almost nothing in the nature of the attack itself to enable a medical man, called to a patient in convulsions for the first time, to say whether he has to do with an attack which will be repeated or not, it is easy to understand that eclampsia is a word more frequently to be seen in books than to be heard at the bedside. In books we may read of *eclampsia neonatorum*, the eclampsia of parturient women, and *uræmic eclampsia*; though the more common clinical equivalents are *infantile convulsions*, *puerperal convulsions*, and *uræmic convulsions*. The distinction between epilepsy and eclampsia is, therefore, one which is to a very great extent purely artificial, from the point of view of the convulsions themselves.

Convulsions have a frequent though less constant relationship also with many other affections, such as general paralysis of the insane, tubercular meningitis, chronic hydrocephalus, hemiplegia, and hysteria.

The more recent classification of convulsions, previously referred to, is that of Dr. Hughlings Jackson (*Brit. Med. Journ.*, vol. i. 1890). This is an attempt in the right direction—an attempt, that is, to identify the intimate cause of the phenomena, and to range convulsions into different classes according to the different portions of the brain which are the seats of the initial morbid processes in this or that form of the disease. He recognises three kinds of convulsion; each kind including several varieties; and these varieties again comprehending 'fits' of many different degrees of severity. The *first* kind or class corresponds with epilepsy proper, and these attacks are dependent, as he thinks, upon discharges of nerve-elements occurring in some portion of the cortex of the prefrontal lobe; the *second* kind or class comprises what are known as epileptiform attacks (epileptic hemiplegia or Jacksonian epilepsy), due to discharges commencing in this or that portion of the cortex of the so-called 'motor area' (Rolandic region) of the cortex; whilst the *third* kind or class comprises what he terms 'pontobulbar fits,' being due, as he thinks, to initial discharges of nerve-elements occurring in some part of the pons, the bulb, or (much more rarely) the spinal cord.

The aim of this classification is essentially scientific, but even its main divisions are

based to a very large extent not so much upon well-ascertained facts as upon hypothesis and speculation. This is especially the case, as Dr. Hughlings Jackson admits, in regard to the convulsions belonging to the first and third classes; though the pathology of the second class—to a very large extent as the result of his labours—stands upon a much firmer basis of actual knowledge. Some kinds of eclampsia would fall into the first class, whilst many others would belong rather to the third class. It is difficult to say into which category well-developed hystero-epileptic attacks should be placed. It must not be lost sight of, moreover, that the conceptions which have inspired this classification are eminently hypothetical and speculative, however ably and consistently they may have been worked out; so that our notions as to the mode of production of fits, and as to the natural groups into which they are to be ranged, should still be regarded as being in a very rudimentary stage.

**ÆTIOLOGY.**—The causes of convulsions are oftentimes more than usually complex, constituting a web of conditions partly made up (*a*) of predisposing, partly (*b*) of exciting, and partly (*c*) of proximate elements. It is often the fashion to pick out some one of the most prominent or easily recognisable of these factors, and speak of it as 'the cause' of the attack. It must never be forgotten, however, that this so-called cause, in any given case, may be able to act as such only when in conjunction with certain other more obscure, though perhaps not less potent, co-operating conditions.

The question of the causation of convulsions resolves itself into a strictly clinical and a strictly physiological department. It is one thing for the medical man to ascertain what are the particular individual states, conditions of life, and occurrences, physical or moral, which have contributed to induce an attack of convulsions. It is quite a different problem when he endeavours to unravel, by physiologic-anatomical data, the actual mode of production of the convulsions. For the present this is a point of view which concerns us less than the more strictly clinical side of the inquiry.

**Predisposing causes.**—One of the most important of these is the existence of an unduly excitable nervous system—one in which there is an exaltation of the tendency to produce reflex movements. This is a state of things which is often more marked in women than in men, and is also notably prominent in young children of both sexes. It is, moreover, much exaggerated in some children of nervous habit, who, besides being unduly emotional or excitable, are very prone to start or tremble at the least noise, and are subject to muscular twitchings in various parts of the body. With increasing age, and more especially in the male sex, we find the

sensorial and emotional nerve-centres becoming less excitable.

In persons possessing a nervous system of this type there is a lack of proper balance or equilibrium between the functional activities of some of its parts; and there seems to be an undue tendency to errors of nutrition whereby the matter of its nerve-cells becomes from time to time highly unstable and prone to undergo 'discharges' of a morbid character.

The predisposing groundwork being of this nature, how is it caused or to be accounted for? (1) It may be inherited from parents, or from grandparents, who may themselves have possessed a nervous system of this type, and may have been subject to fits or other well-marked disease of the nervous system. But though not inherited in the strict sense of the term, it may (2) be connate: the patient may always (*i.e.* from birth) have possessed a nervous system of this type, as an accompaniment of the mere low vitality which is often seen in children born from parents who are weak or aged and debilitated. (3) At other times the nervous system may have acquired such predisposing characters some time during the life of the individual (especially during childhood or adolescence) owing to the action of various sets of conditions, such as—(*a*) the cachexia which occasionally follows measles, pertussis, &c.; (*b*) insufficient or improper food; (*c*) chronic diarrhoea; (*d*) hæmorrhages or exhausting discharges.

If we turn now to the various *exciting causes*, we find these powerfully influenced by the periods of age, which we may conveniently, though artificially, mark off from one another.

**Infancy** (from birth to end of 2nd year).—In certain cases convulsive attacks are congenital; and here perhaps the most frequent exciting cause is a meningeal effusion of blood which irritates the surface of the brain—the extravasation having been occasioned during parturition. These congenital attacks are most frequently associated either with more or less marked hemiplegia, or with a subsequent partial or distinct condition of amentia or idiocy. Such unfortunate infants may continue quite unable to stand or even sit up; they remain unable to speak, and, as in a case seen by the writer, the child may be quite blind. Meningeal or superficial hæmorrhages may also occur in young infants, under the strain produced by violent fits of coughing; and in some of these cases such effusion may be followed by convulsions. Fits in infancy may also follow falls or blows upon the head.

In infancy, again, convulsions may usher in or supervene in almost any acute disease, this being especially the case in the exanthemata; in pneumonia or bronchitis; and also in tubercular meningitis.

But, still more frequently, convulsions in infancy are excited by mere peripheral irrita-

tions, as during the process of teething, from an overloaded stomach, or from indigestible food. Diarrhœa and worms also take their place as more or less frequent excitants of convulsive paroxysms in infants, though worms only begin to appear towards the end of this period. But though irritations in the field of distribution of the fifth and pneumogastric nerves are especially potent in exciting convulsions, irritations of other parts of the body may also lead to similar results, whether they are occasioned by the injudicious disposition of pins, by tight strings wounding or irritating the skin of the body, or by any other means. The more distinctly that predisposing conditions exist, the more frequently will any or all of these exciting causes give rise to an attack of convulsions.

Lastly, an infant which has taken the breast of a woman who has shortly before been much perturbed by violent anger, grief, or any other strong emotion, may thereafter, if predisposed, be seized with convulsions—probably owing to the milk of the nurse having been so altered in quality as to have led to gastric trouble and irritation in the infant.

*Childhood* (from the 2nd to the 13th year). Most of those exciting causes which are influential during the previous period continue to be occasionally operative in this—especially during the first half of it. Meningeal hæmorrhages are now rarer, though they may still occur during violent paroxysms of coughing, and also from falls or blows upon the head. The latter causes of hæmorrhage may, however, act in producing fits in other ways, *e.g.* by concussion, shock, &c., and they now come to be more frequently operative. The exanthemata are still apt to be preceded or associated with convulsive attacks; and irritations, especially from teething or irritants (undigested food or worms) in the intestinal canal, are also occasionally prone to be followed by such a sequence.

During this period another cause comes into operation with great frequency, and this is fright. The first fit either follows the fright more or less immediately, or it may not take place for days, or perhaps for weeks. During the interval, however, the health and mental condition of the child is generally obviously disturbed. Proper treatment at this stage may prevent the occurrence of fits. Scrofulous or other tumours in the brain sometimes help to determine convulsions in children.

*Adolescence* (from the 13th to the 20th year).—Fright or other sudden emotions, falls upon or blows about the head, still appear as frequent excitors of convulsive attacks which recur (epilepsy) at this period of life, though meningeal hæmorrhages, acute diseases, and peripheral irritations are much less frequently operative than at earlier periods, since the special irritability of the nervous system characteristic of childhood decidedly abates during these years.

Other and new causes, however, come into play at this epoch. The establishment of puberty is a kind of crisis during which, independently of all other causes, convulsions or fits may be initiated in those whose nervous systems are at all predisposed towards such an occurrence. This is more especially so in the case of the female, partly because of the existence of a rather more frequent predisposition in persons of this sex, and partly because of extra excitations in association with the establishment of the catamenia. Ovarian or uterine irritation, or irregularity of the functions of these organs at this period, may occasion fits which may take an hysterical type. Masturbation may also be added as an occasional provocative of epilepsy at this critical period of life, though the writer is inclined to think too much stress is often laid upon this as a cause. This mode of causation occurs more frequently with the male sex.

Excessive study and mental application, as well as worry or anxiety, must also undoubtedly be enumerated among the causes of epilepsy at this period of life.

Neither must we forget the possible existence of aneurysms of the cerebral arteries, or of morbid growths in connexion with some portion of the brain or its meninges. This latter cause also figures in earlier periods of life—more especially in those children who are of a scrofulous type. And in some of such cases the new-growth may lead to the supervention of chronic hydrocephalus, and thus render the occurrence of convulsions even still more likely.

*Early adult age* (20-40 years).—Fits originate much less frequently during this period of life than in adolescence or childhood. They are, however, apt to supervene, more especially when the general health is lowered, under the influence of various exciting causes. Grief and mental worry (particularly when combined with long-continued bad sleep), and the labours or cares of business, are then apt to induce them.

Blows or falls upon the head may still be followed by attacks of this kind, though perhaps with less frequency than in the earlier periods of life.

Syphilitic indurations or growths from the meninges may now occur; and other attacks (often of one-sided convulsion) may be determined by various pathological changes or accidents taking place in regions of the brain where more severe lesions would give rise to hemiplegia. The primary change in these cases may be minute hæmorrhages into the brain-substance, or minute and slight softening produced by blocking of small vessels (embolism or thrombosis). With lesions of this kind hemiplegia and convulsions are often more or less associated. Occasionally the cause may be a non-syphilitic tumour, occupying the side or base of the brain.

Puerperal convulsions in the female, and uræmic convulsions in both sexes, are most frequently met with during this period. During pregnancy the excitability of the nervous system is often greatly increased, and in the production of puerperal convulsions some amount of uræmia also intervenes not infrequently. Intemperate habits, carried to excess, frequently produce fits, and so also may venereal excesses.

*After middle age* (40 years and onwards). The excitability of the nervous system diminishes, so that epileptic attacks commence with much less frequency at this period. At the climacteric this excitability is temporarily increased, and fits again become more frequent, under slight exciting causes.

Although fits are only very rarely liable to be induced by the sequelæ of hæmorrhages or of occlusions of cerebral vessels, yet these events now grow more common as age advances, and are therefore to an almost corresponding extent more liable to figure as causes of convulsive attacks. An attack of hæmorrhage or of softening may be ushered in by epileptiform convulsions (especially when the lesion occurs in certain parts of the brain), and in some cases such attacks may thereafter recur at irregular intervals. Exposure to great heat, or sunstroke, may also at this period, or earlier, act as the exciting cause of convulsions.

Mental overwork, worry, fright, and such-like influences, are much less likely to operate in persons over 40 than in earlier life; and the same is to be said of blows or injuries of the head, short of the most severe, causing actual lesions of the brain. But the malnutrition and degeneration induced by intemperate habits may predispose to symptoms of this kind; and so also may blood-poisoning from renal disease, which is often present.

Various organic diseases of the brain, whether principally characterised by degenerations with a process of more or less general atrophy, or with localised overgrowth of connective tissue, are also not infrequently productive of convulsions, either in persons of middle or of advanced age. A well-marked instance of the former of these associations is to be met with in general paralysis of the insane. Cysticerci on the surface of the brain have also been the cause of most obstinately recurring convulsions.

Lastly, it should always be borne in mind that convulsions are sometimes the result of the action of poisons of various kinds upon persons of any age. Occasionally such poisoning may be brought about by articles of diet, such as mussels or fish in certain states, or poisonous mushrooms; whilst at other times it results from some of the well-known narcotico-irritant poisons, taken either inadvertently or purposely.

**ANATOMICAL CHARACTERS.**—These may be

said, so far as our present knowledge goes, to be absent. It is true that general or partial congestion of the brain may frequently be encountered in those who die during an attack of convulsions. But this congestion is to be regarded as a result rather than as a cause of the fit. The convulsions are due to mere molecular changes in the brain, inappreciable to, or at all events unappreciated as yet by, the most skilled microscopists. When fits occur in association with actual organic growths or other lesions of the cerebral cortex or elsewhere, some of the nerve-cells contiguous to such lesions may have their nutrition so altered as to form occasional starting-points for nervous discharges, which descend so as to upset the equilibrium of certain unstable or highly charged motor centres (ponto-cerebellar) in such ways as to determine the convulsive phenomena. But a similar disturbance of nervous equilibrium with discharge of motor energy, immediately productive of convulsive phenomena, may result, in another case, from abnormal visceral impressions (induced, it may be, by indigestible matters in the intestine, or by an acute disease of the lungs), or from some surface-irritation. In accordance with this point of view, such organic growths or other lesions as may be found in the brains of epileptic patients need receive no further mention here. Those who may be inclined to think otherwise, should bear in mind the fact that convulsive attacks are easily produced in animals from whom the cerebral hemispheres have been removed. It should always be borne in mind, however, that there is one region in particular of the cerebral cortex, viz. the so-called Rolandic area (comprising the convolutions which bound the fissure of Rolando), the irritation of which by new-growths or other organic lesions is specially prone to give rise to unilateral convulsions, whose characteristics are in many respects extremely well-defined ('Jacksonian epilepsy').

**SYMPTOMS.**—The varied nature of the causes makes it impossible to say anything of moderate compass concerning the premonitory signs or symptoms which may precede an outbreak of convulsions. These must necessarily vary immensely in different cases. Some of the characters of the predisposing state have been already alluded to. The onset is, however, often abrupt, and without any distinct premonitory symptoms.

With regard to the actual characters of the attack, it will be sufficient to say here that they also vary extremely in different cases; and as, notwithstanding this great variability, it is impossible in any individual case to tell, from the nature of a first convulsive fit, whether it will form a more or less isolated attack, or whether it will constitute one of a subsequently recurring series, the reader may, for this part of the subject, be referred

to the description of the different forms of the attack given under EPILEPSY. All that is there said concerning the actual phenomena and mechanism of the attacks, holds good for occasional convulsions as well as for those which are habitual. In each well-marked attack we have to do with (1) a more or less distinct stage of tonic spasm, followed (2) by one of clonic spasms, and (3) succeeded by a state of stupor. One or other of such stages is, however, not infrequently more or less abortive. Convulsive attacks may, at times, so rapidly follow one another as to be merged into one long series or *status convulsivus*, differing in no respect from the analogous *status epilepticus*.

COMPLICATIONS AND SEQUELÆ.—The complications of convulsions are most various, seeing that in different cases we may have to do with irritated gums, repletion, diarrhœa, worms, or an acute specific disease; whilst in other cases the causal complicating conditions may be general debility and sleeplessness, mental anxiety, puberty, or the climacteric period. Again, in other cases, either pneumonia, renal disease, pregnancy, ovaritis, or some organic brain-disease may be the accompanying condition.

The nature of the sequelæ will depend principally upon the frequency of the attacks, and the duration of the period during which the patient has been subject to the recurrence of them, so that for this part of the subject the reader may refer to the article EPILEPSY.

DIAGNOSIS.—There is very little difficulty in regard to the diagnosis of convulsions. The differential characters of laryngismus stridulus are given elsewhere, and the absence of any real distinction between a fit of eclampsia and a fit of epilepsy has already been insisted upon. Neither of these affections can be easily confounded with certain forms of chorea, which occasionally present themselves in adults, with movements not unlike those of ordinary convulsions. The more continuous nature of the movements, and the fact that consciousness is not impaired, suffice to distinguish all forms of chorea. The spasms of tetanus and hydrophobia are also easily distinguishable from an ordinary attack of convulsions.

The characteristics of hysterical convulsions are pointed out in the article HYSTERIA.

The real difficulties from the point of view of diagnosis have reference to the cause of the attack. To arrive at a decision in regard to this is often very difficult, and occasionally impossible—at all events when a patient first comes under observation. At other times, however, the indications are so plain that there can be little or no difficulty. It is a question which should always be considered with the utmost care, since on the correctness of our conclusions in regard to this point the efficacy of the particular line of treatment which we adopt must necessarily depend.

Nothing is more to be deprecated than hasty jumping at conclusions, from mere routine and superficial considerations. The condition of the patient must be carefully examined, and the nurse, attendants, or relatives must be closely questioned in order that we may learn as much as possible as to the previous state of health of the patient, and more especially as to the time and events which immediately preceded the first outbreak of an attack of convulsions. Examination and inquiries combined may convince us that the convulsions are (1) of the primary or idiopathic variety, immediately occasioned perhaps by fright, anxiety, overwork, overmuch or indigestible food, &c.; or we may come to the conclusion that the convulsions are (2) of the sympathetic order, dependent upon pregnancy, renal disease, the onset of an acute specific fever or of pneumonia, or due to the existence of whooping-cough, scarlet fever, &c. Or, in the absence of reasons for placing them in either of these categories, we may be forced to conclude that they are (3) symptomatic of some organic brain-disease, the nature of which must then be determined as nearly as possible, judging from the age of the patient, the mode of onset of the attacks, his present state and associated conditions.

In any case we may have to inquire more closely as to the hereditary tendencies, or acquired predisposing causes, which sometimes reduce the nervous system to such a degree of excitability, or disturbed equilibrium of certain parts, as to lead to an attack of convulsions without the aid of any obvious exciting cause. In infants or very young children such a condition of the nervous system may display itself by great restlessness and startings at night, by the child's sleeping with half-open eyes, by drawing in of the thumbs across the palms, by twitchings of the limbs, of the angles of the mouth, or of the facial muscles generally. In older children and in young adults the signs which most easily mark a similar below-par condition of the nervous system are twitching of the muscles about the angle of the mouth, and of the tongue (the former being specially well seen when the latter organ is tremulously protruded for inspection), associated with debility, anorexia, partial insomnia, and general nervousness. In nervous girls fits are induced by very slight causes about the time when the catamenia become first established. The indications for treatment must in fact vary immensely in any five or six consecutive cases of convulsions to which the practitioner may be summoned.

PROGNOSIS.—The possibilities under this head are at least six in number in regard to any case of convulsions:—(1) The patient may recover after having a single fit or a batch of them within a few hours or days, and may never have another attack. (2) The

patient may recover, and though he or she may not have fits habitually thereafter, they may recur at prolonged intervals, whenever predisposing circumstances chance to be strong or are supplemented by an exciting cause of unusual potency. Thus convulsions during teething may cease, and may not recur till the constitution has been lowered by some illness years after, or when the nervous system has been rendered more irritable, as at the time when the catamenia are about to commence, especially if then some slight fright should also come into operation as an exciting cause. (3) The patient may recover, though he subsequently continues to have fits either at irregular or regular intervals; he becomes, in short, a confirmed epileptic. (4) The convulsions may come to be followed by temporary delirium or a more or less marked maniacal condition, recurring after all or some seizures. (5) The patient may recover from the convulsive attack and may or may not have another fit, though he may remain hemiplegic. (6) The patient may die during the attack or almost immediately afterwards—(a) from the effects of it, or (b) by reason of some organic lesion by which the fit itself has been determined.

Recoveries are fortunately the rule, but death, especially in infants, is by no means uncommon. We possess no accurate data to enable us to assign the numerical proportion of these terminations to one another or to the other above-mentioned sequences.

**TREATMENT.**—During the convulsion itself, whether we have to do with an infant or an adult, we must see that all clothes are thoroughly loose about the neck and chest, and the patient should be placed in the supine position with the head slightly raised. Beyond seeing that the patient does not knock or injure himself, owing to the violence of his movements, these should not be much restrained; although efforts should always be made to prevent the tongue being bitten, by slipping the most suitable thing at hand between the molar teeth on one side, when the age of the patient or the character of the fit renders it likely that this event might otherwise occur. Beyond such simple measures as these, the less we do during the actual continuance of the fit the better it will probably be for the patient. We know of no rational or successful means of cutting short an ordinary attack of convulsions, and in the face of such an attack we should be cautious how we interfere lest evil may be done.

Where we have to do with a succession of attacks quickly following one another, and which have already lasted some time, the injection into the rectum of chloral hydrate, with or without bromide of potassium, in suitable doses may be beneficial; or the careful administration of chloroform may be tried, as it is found very serviceable in many cases when a *status convulsivus* occurs in

children or in adults—though it would not be desirable to have recourse to it in infants. Under similar circumstances, for the latter the warm bath may be substituted, and sometimes seems to do good. Antipyrin and antifebrin have also occasionally been given with benefit in cases where fits are succeeding one another rapidly, and such drugs are especially indicated in those cases where the temperature in the rectum rises rapidly to 103° F., or upwards.

On the cessation of the convulsion or convulsions, or during the intervals, the treatment to be adopted to prevent their recurrence must necessarily vary immensely according to the age of the patient, and according to the predisposing and exciting causes which appear to have been operative in inducing the attack.

An overloaded stomach will call for the speedy administration of an emetic; or where indigestible food has already passed into the intestine, an enema or brisk purgative should be given. Diarrhœa must be checked, or anthelmintics administered when worms are suspected. Gums may be lanced if they seem really to need it. In many of these cases an acquired or hereditary predisposition will have to be combated by the careful regulation of the diet, so that nutritious and easily digested food is to be given in place of their opposites, and at the same time the most suitable nervine tonics and antispasmodics.

For general usefulness in such cases no remedies can compare with the bromides of potassium, sodium, and ammonium. The former salt seems to be the most potent of the three; though if the bromide of sodium will check the fits, it is often more desirable to give it than the potassium salt. On the other hand, where the circulation is weak, or mental depression is apt to be induced, it is often best to administer bromide of ammonium. Sometimes the best results may be obtained by giving two of these salts in combination; at other times by administering moderate doses of chloral hydrate in combination with one or other of them. As part of the specific influence which these drugs exercise over nerve-tissue, they fortunately establish a tendency to quieter and sounder sleep, of which such patients often stand much in need. Ten-grain doses three times a day should be given at the commencement to youths or adults, and afterwards slightly increased if necessary. Or a larger dose may be given once a day, either in the morning or at night, or night and morning, according to the indications in each case.

Very large doses of these drugs are rarely beneficial, and may do harm (*see* BROMISM). Their action often seems to be favoured and harmful symptoms warded off by combining with the bromides suitable doses of liquor arsenicalis and tincture of digitalis or of convallaria. Quinine or belladonna are other

remedies which may also often be given simultaneously with great advantage. For young children or infants, the dose of the bromide must, of course, vary with their age.

Valerianate of zinc and oxide of zinc are mostly remedies of altogether less power, though these and other drugs may be tried where bromides appear to fail. Occasionally, too, good results may follow from the administration of borax in doses of fifteen or twenty grains three times a day, freely diluted and combined with liquor arsenicalis, where bromides only seem to aggravate the patient's condition, or, at least, to be powerless for good.

General treatment of this kind is applicable to a large proportion of cases also in which in debilitated or 'nervous' patients fits have been brought on by fright, worry, or anxiety, or by no assignable cause. Wherever the fits have recurred frequently, and a sort of habit has thus been established, such treatment has to be continued over long periods (one to three years or more), with suitable modifications, in order to stamp out the tendency to recurrence and thus effect a cure. In all such cases fatigue of mind and body is always to be avoided; a quiet and regular life, free from excitement, must be led; and in those instances in which over-attention to business or over-study has been in part operative in bringing on the fits, absolute rest must form an essential part of the treatment. In girls or young women in whom fits occur at the time of the establishment of the catamenia, or where they recur in association with an irregular menstrual function, the general health often requires our most careful attention, in addition to such measures as are above indicated.

The convulsions that belong to the class known as *sympathetic* have to be carefully considered in relation to the malady of which they are the forerunners or associates. When convulsions precede an attack of scarlet fever or of small-pox they usually subside of themselves as the disease develops. They are, however, of much more significance when occurring during the course or towards the close of one of these maladies, or during an attack of whooping-cough or of croup. Our indications for treatment must then be derived in the main from the general state of the patient, and this is also eminently the case where we have to do with uræmic convulsions.

The treatment of *symptomatic* convulsions, dependent upon actual organic brain-disease, must also necessarily be subordinated to that appropriate for the affection itself upon which such symptoms depend. No drug will be found more generally useful, however, than bromide of potassium in ten- to fifteen-grain doses for an adult (administered three times a day), in checking or diminishing the repetition of convulsions in these cases. Often here also the action of the bromide seems to be favoured by combining it with liquor

arsenicalis and with moderate doses of digitalis, the latter being indicated especially in those cases in which there is great general nervousness in association with a disordered cardiac rhythm. Where sounder sleep is necessary, chloral hydrate, alone or in combination with bromide of potassium, should be given at bedtime. Sulphonal often has the desired effect. Tinctures of sumbul or of henbane are at times useful adjuvants.

Where we have to do with tumours of the brain, and especially with syphilitic growths in the meninges, much better results are to be hoped for from large and increasing doses of iodide of potassium (that is, x to xxx grains or more, three times a day), either alone or in combination with small doses of bichloride of mercury. With such drugs we should combine the administration of nutritious food, and attention to the improvement of the general health in every way that may be possible. Occasionally, both in these cases and in non-specific cortical growths and thickenings of the dura mater, the operation of trephining has been had recourse to with great benefit to the patient. In some instances a cortical growth, or a portion of cicatricial tissue or of thickened dura mater, has been excised, with the effect not only of curing previous pain and tenderness, but also of curing or greatly diminishing the frequency of epileptiform attacks to which such patients have been subject. The cases for which this treatment is suitable constitute, however, only a comparatively small percentage; and much judgment and knowledge are needed for the selection of those in which such surgical treatment is likely to prove beneficial. See BRAIN, Diseases of; EPILEPSY; and SPINAL CORD, Diseases of.

H. CHARLTON BASTIAN.

**CO-ORDINATION.**—This term is used in reference to muscular movements principally. Certain parts of the nervous system have more especially to do with the calling into activity, and therefore with combining, the contractions of different muscles, both simultaneously and in succession, in the precise order in which they occur in the several motor acts of which they are capable. The nervous arrangements upon which these actions depend have come into being, both in the race and in the individual, by processes of organic growth and development *pari passu* with the possibility of executing these several movements. It would be wrong to expect, therefore, that an isolated organ should exist solely for co-ordinating muscular movements. The execution of the most habitual of these must depend, to a large extent, upon the activity of the ordinary motor (and related sensory) tracts of the spinal cord and brain. The extent or precise mode in which the cerebellum intervenes in certain higher forms of co-ordination is still involved in much

obscurity. That it has some share in such functions may be regarded as certain, though it probably intervenes far less than some would have us believe, who regard the cerebellum as the organ for the co-ordination of muscular movements.

Many nervous affections exist in which the co-ordination of muscular movements is more or less impaired. One of the most familiar of these is locomotor ataxy, a disease dependent upon a morbid process in the posterior columns of the cord. Sclerosis of the antero-lateral columns of the cord also not infrequently disturbs the execution of muscular movements, especially those of the upper extremities. Chorea gives rise to very similar uncertainties in the execution of muscular acts. Spasms of all kinds, in short, tend to interfere with the harmony of the muscular movements in the course of which they intervene. Stammering is an affection of this kind, implicating some of the muscles of articulation; and certain disturbed cardiac actions characterised by disordered rhythm can only be regarded as belonging to the same category.

The above-mentioned are common instances of impaired co-ordination of muscular movements dependent upon structural or functional changes in parts of the nervous system other than the cerebellum. Certain diseases of this organ, however, are known to give rise to a distinct form of inco-ordination. It is characterised by a reeling, unsteady gait in walking, with legs straggling, and mostly wide apart, to which the term 'titubation' is commonly applied. Other kinds of inco-ordination may hereafter be proved to depend upon diseases of the cerebellum. Its morbid conditions are still very imperfectly recognised, and this is especially true in regard to its merely functional perturbations.

Certain inco-ordinations in speech and writing are common. Instances are to be found in that use of wrong words or misapplication of terms which we meet with in aphasic and amnesic persons; also in the substitution of wrong words in the act of writing, or of wrong letters in the writing of words, when such substitution is mechanical and unintentional—and when it is wholly distinct, therefore, from mere inability to spell. These defects are inco-ordinations of a complex kind, dependent upon the perverted action of higher cerebral centres, in the same way that incoherent speech generally is dependent upon incoherent thought. There is reason to believe, indeed, that the same kind of ultimate defective nervous action which leads to inco-ordinations of movements when certain motor regions of the nervous system are affected, may, on the other hand, give rise to perverted perceptions (*illusions*) or to perverted thought (*incoherence*) when the disturbed nervous action occurs in other and in higher parts of the central nervous system.

H. CHARLTON BASTIAN.

**COPHOSIS** (κωφός, deaf).—Deafness. See HEARING, Disorders of.

**COPPER, Poisoning by.**—SYNON.: Fr. *Empoisonnement par le Cuivre*; Ger. *Kupfervergiftung*.—*Metallic copper* may be regarded as innocuous when swallowed, and the recent researches of Hirt show that those who are engaged in the metallurgy and manufacture of copper utensils are not specially liable to any diseases which can be attributed to copper as such. It is, indeed, stated that workers in copper enjoy an immunity from cholera, a conclusion which is based on very insufficient premisses. It is contradicted by the occurrence of cholera among coppersmiths in Breslau in 1866, and by certain other cases of a like nature reported by Hirt. That the disease is seldom found among workers in copper is true, but that the copper has anything to do with this result is not proved.

Though pure copper may be regarded as innocuous, it is otherwise with *alloys of copper*, more particularly those with zinc and tin, known under the names of brass and bronze respectively, and with compounds of copper with lead or arsenic. In these the injurious agent would appear to be the alloy, and not the copper itself. An affection of a febrile character, and known as 'brass-founder's ague,' occasionally occurs on fusing days, and it is attributable to the zinc fumes which are generated by the melting process.

The *salts of copper*, on the other hand, are capable of causing injurious and fatal results. The more important salts, from a medico-legal point of view, are the sulphate, blue vitriol, or bluestone; the acetates (basic and neutral) constituting artificial verdigris; and the carbonate or natural verdigris. The manufacture of verdigris is carried on to a large extent in the South of France. Plates of copper are acted on by the skins of grapes, which are allowed to undergo the acetous fermentation. Those engaged in this industry on the whole enjoy good health, and it is only rarely that symptoms can be directly traced to the work; and then only when through sheer carelessness and uncleanness quantities of the salt have been ingested. It is even said that dogs eat the refuse grape-skins without appearing to suffer from poisonous symptoms. On the other hand, symptoms of poisoning of a family have recently been recorded where they had eaten haricot beans, grown, as is the custom, between the vines, which latter had been watered with a solution of sulphate of copper (Raynaud, *Bull. Gén. de Thérap.*, 1888).

1. **Acute poisoning by copper.**—SYMPTOMS.—The salts of copper, when taken in sufficient quantity, cause symptoms of acute poisoning, frequently terminating fatally.

Twelve to fifteen grains of the acetate have been sufficient to kill a dog within an hour. The fatal dose in man is not quite determined; but doses above the usual emetic dose of the sulphate (ten to fifteen grains) have caused serious symptoms, and death has resulted within four hours after swallowing some pieces of the sulphate. Starr (*Med. Record*, May 1882) records a case of death in four days from one ounce of the sulphate. Half an ounce would probably cause a fatal result. The symptoms are essentially those of irritant poisoning, namely, styptic or coppery taste, constriction of the fauces, epigastric pain, violent vomiting and purging, followed by collapse and death, usually with tetanic or convulsive symptoms. That which characterises copper-poisoning more especially, as compared with other irritants, is the frequent occurrence of jaundice. Hæmoglobinuria with albuminuria has also been observed (Starr's case). In dogs copper usually causes death with symptoms of paralysis of the hinder extremities, proceeding like a form of ascending paralysis, in addition to the usual irritant symptoms. It is said also to have a paralysing action on the heart. Such effects have only followed, at least constantly, introduction into the veins; when taken by the stomach most of it seems to be stopped by the liver, or in the case of some of the salts (*i.e.* acetate) may be reduced by the sugar of the food (Roger, *Revue de Médecine*, 1887, No. 11).

2. **Chronic poisoning by copper.**—It is generally stated that the long-continued introduction of copper into the system in small doses gives rise to a form of chronic poisoning known under the name of 'copper colic.'

**SYMPTOMS.**—The symptoms are essentially those of gastro-intestinal irritation, with nausea or sickness and diarrhœa. They have none of the characters of colic in the sense in which the term is usually employed. The hair and the cutaneous secretions of workers in copper and brass are sometimes found of a green colour, and a line is sometimes found at the margin of the gums and teeth, variously described by authors—Corrigau calling it purple, while Clapton calls it green.

Though symptoms of gastro-intestinal irritation, as above described, have been found among workers in copper, the question is whether they are in reality due to the copper, or merely symptoms of a not uncommon affection showing themselves among copper-workers. That copper does gain access into the system, and may be detected in the urine during life, and found in the bones after death, without the individual showing any manifest symptoms during life, seems pretty well established. But though we may regard it as certain that symptoms of copper-poisoning are more rarely found than those of lead-

poisoning among those who have to deal with these metals, yet it would be a very unwarrantable conclusion, and contrary to all that we know of the action of poisons, to assert that a substance which is undoubtedly poisonous can be taken freely into the system with impunity. This is a point of considerable interest in reference to the accidental or wilful adulteration of articles of food with copper salts. Many cases are on record of severe symptoms resulting from the use of copper utensils in cooking, or more frequently from the storage of water or articles of food in copper vessels, especially if the food contains oil—which, on turning rancid, dissolves the copper—or vegetable acids, or even large quantities of ordinary salt.

Copper salts are also employed intentionally to impart a green, fresh colour to pickles and preserved vegetables, such as peas. It is asserted that the quantity of copper necessary to produce this effect is infinitesimal, and that no poisonous effects can be proved to have resulted even from long-continued employment of these vegetables as articles of food. This is strongly maintained by M. Galippe, who has tried them on himself and family. Assuming the impossibility of proving the injurious effects of copper-tinted vegetables, the question comes to be principally a social and economic one, as to the propriety or legality of adulteration of food at all, and especially with a substance undoubtedly poisonous. Copper is said by Odling and Dupré to be a natural constituent of the human body. They have found it in the blood, tissues, viscera, and in many animal products, such as cheese, eggs, and also in many vegetables. It takes the place of iron in the blood colouring-matter of some invertebrates (cray-fish), and is found as a constituent of the colouring-matter of the feathers of the turaco (plantain-eater).

**DIAGNOSIS.**—The greenish or bluish colour of the vomited matters, which turn bright blue on the addition of ammonia, renders the diagnosis comparatively easy.

**TREATMENT.**—In *acute* cases the stomach should be evacuated by encouraging vomiting, or by the stomach-pump. Albumen in some form, as milk or white-of-egg, should be given, in order to precipitate the copper. Iron filings may be given for a similar purpose.

In *chronic* poisoning the cause should be discovered and removed, or the individual removed from the cause. D. FERRIER.

**CORN.**—**SYNON.**: *Clavus*; Fr. *Clou*; *Cor*; Ger. *Leichdorn*; *die Hühnerauge*.

**DEFINITION.**—A corn is a thickening of the epidermis, caused by undue pressure and friction, as by boots, shoes, or implements of occupation. It is usually situated on a prominence, such as that of a joint, where the skin is subjected to double pressure

and is therefore unable to yield, or between the toes. Corns are most common on the feet.

**DESCRIPTION.**—A corn usually begins as a general and uniform thickening of the epidermis, which is termed a *callosity* (*tyloma*, *tylosis*, *Schwiele*). Callosities may occur on any part of the integument. Thus they may occupy the prominence of a joint, or spread over the heel or the metatarsal cushion of the foot from pressure in walking, or occupy the metacarpal prominences of the hand, as in boatmen.

The callosity is composed of laminated epidermis; is thickest in the centre, becoming thin towards the circumference; and is more or less hard and condensed, smooth and hornlike in appearance, and yellowish in colour. When the irritation which gives rise to a callosity is prolonged, effusion is apt to take place beneath it, and it is raised like a blister, the effused fluid being sometimes serous and sometimes sero-purulent. Whenever this happens, the subsequent separation of the horny layer results in spontaneous cure.

When the pressure giving rise to a callosity, instead of being diffused, is concentrated on a central point, the epidermis corresponding with that point increases in thickness, by its under surface, and forms a conical prominence; further pressure increases the length and breadth of the cone, and in this way a corn is established. Continued irritation enlarges the corn by hyperplasia of epidermic cells, and its pressure produces absorption of the derma, sometimes extending to the bone itself. Not infrequently effusions of serum or blood take place beneath the conical prominence; and, in rare instances, a bursa is found between the corium and the joint.

At a late stage of its growth the corn has the appearance of a central core—technically, *the eye of the corn*—surrounded by a collar of smooth epidermis in the state of callosity. The core is a lamellated ovoid mass, corresponding in external figure with the cup by which it is produced; and consisting in substance of vertical cup-shaped lamellæ closely packed one within the other. In an old corn the shape of the entire core is conical, the point resting on the sensitive skin, and the signification of the term *clou* or nail thus applied by the French is made manifest. The substance of the corn resembles horn both in colour and density, but between the toes, where moisture is generally present, it remains white and soft, like soddened cuticle, and is thence named *soft corn*. Effusion at its base is more common in the soft than in the hard corn.

**TREATMENT.**—The treatment of a corn is to remove its causes, namely, pressure and friction; but when this is impracticable, to equalise pressure, by which the corn will

revert to the state of callosity. The second indication is best effected by some simple unirritating application, such as the soap- or lead-plaster spread on washleather. As a preliminary to this application, as much of the hard epidermis as possible should be removed by soaking and scraping, and the core turned out with a blunt-pointed instrument. The soft corn may be removed as the hard one, or by snipping with scissors. When there is inflammation about the corn, it should be treated by water-dressing. Chronic corns are much benefited by an application composed of Salicylic Acid, 30 parts; Extract of Indian Hemp, 5 parts; Flexile Colloidion, 240 parts.

ERASMUS WILSON.

**CORNEITIS.**—Inflammation of the cornea. See EYE AND ITS APPENDAGES, Diseases of.

**CORNUAL** (*cornu*, a horn).—Relating to the cornua or horns of grey matter of the spinal cord, e.g. *cornual myelitis*. See SPINAL CORD, Diseases of.

**CORONARY ARTERIES, Diseases of.**—SYNON.: Fr. *Maladies des Artères Coronaires*; Ger. *Krankheiten der Kranzarterien*.—The diseases of the coronary arteries may be classified as follows: (a) atheroma and calcification; (b) aneurysm; (c) occlusion of the orifice; (d) thrombosis and embolism; and (e) syphilitic disease.

a. **Atheroma; Calcification.**—Atheroma and calcification of the coronary arteries are frequent, but by no means constant, sequelæ of similar changes in the root of the aorta. The disease may be general, affecting both vessels equally or unequally; or it may be limited to one of them, or even to a primary branch of either; and may lead to fatty degeneration or fibroid change of the corresponding substance of the heart. The experiments of Mr. Erichsen and M. Schiff have proved that the nutrition of the heart in health depends upon a free coronary circulation, and pathology has confirmed this conclusion. Sir Richard Quain found the coronary arteries diseased or obstructed in 13 out of 33 cases of fatty degeneration of the heart, and in one of these cases, the trunks of both vessels being healthy, a calcified coronary branch led to the only portion of the heart exhibiting fatty change. Of 55 cases of fatty degeneration of the heart collated from the *Transactions of the Pathological Society of London*, 21 exhibited atheromatous or calcific change of the coronary arteries. In 10 of these death occurred by rupture of the left ventricle, in 1 by rupture of the right ventricle, and in 2 by rupture of the septum ventriculorum, the coronary branch leading to the seat of rupture having been, in every instance, in a more advanced state of disease than the other portions of

the vessel. From the foregoing statistics it would appear, that whilst fatty degeneration of the heart may exist independently of disease of the coronary arteries, the latter condition may be regarded as an *immediate* cause of fatty change in the heart, in the proportion of about 38 per cent. of all cases. Hüfer points out (Virchow's *Archiv*, vol. lxxxix. 1882) that fibroid disease of the heart is frequently associated with sclerotic changes in the coronary arteries, the fibroid patches corresponding to the site of the changes in the arteries. Fibroid disease may also be produced as a result of embolism of the coronary vessels.

**SYMPTOMS AND SIGNS.**—There are none which are peculiar to this disease; those which exist being due to the consecutive changes in the substance of the heart. The doctrine formerly held, that calcification of the coronary arteries was the cause of angina pectoris, is no longer tenable, though fatal cases of angina are more frequently associated with disease of these vessels than with any other lesion. It is probable, however, that the affection of the coronary arteries produces angina only by disturbance of the functions of the cardiac nerves and ganglia. See ANGINA PECTORIS.

**b. Aneurysm.**—Aneurysm of the coronary arteries is of rare occurrence. It is usually preceded by atheromatous or calcific changes in the coats of the vessels, and may, therefore, be regarded as a disease of middle or advanced age. Dr. Gee has, however, published an example of coronary aneurysm in which the patient was a boy of only seven years.

**SYMPTOMS AND SIGNS.**—There is no positive indication of coronary aneurysm during life. When the termination is fatal, as it usually is, death occurs by hæmorrhage into the pericardium from rupture of the sac.

**c. Occlusion.**—Occlusion of the orifices of the coronary arteries has been met with only in connexion with calcific changes in the root of the aorta, a partially detached calcareous plate overlying the orifice of either vessel (both are rarely affected), and partially or completely shutting off the circulation. There are no *symptoms* distinct from those of the principal disease.

**d. Thrombosis and Embolism.**—*Thrombosis* of the coronary arteries is a frequent result of disease in the coats of these vessels; and owing to their small size, complete blocking and arrest of circulation through them are the ordinary consequences of this accident. *Embolism* of the coronary arteries has been observed in cases of endocarditis especially involving the mitral valve. The heart-substance, depending upon the occluded vessel for its vascular supply, quickly undergoes the atrophic changes of fatty or fibroid degeneration; or appearances like those found in infarction in other viscera

are produced. Special *symptoms* are entirely wanting.

Disease or obstruction of the coronary arteries, with the ordinary consequences—fatty degeneration of the heart, and anæmia with white softening of the brain—are conditions usually found in cases of permanently slow pulse.

**e. Syphilis.**—Syphilitic disease of the coronary arteries has been recorded in a number of instances, but its identification depended entirely upon the history and the concomitant symptoms, the deposit being histologically indistinguishable from ordinary atheroma.

THOMAS HAYDEN. C. J. NIXON.

**CORPORA AMYLACEA** (*corpus*, a body; and *amyllum*, starch).—These minute bodies, which were first described in this country by Sir Richard Quain and Dr. Hughes Bennett in the 2nd and 3rd vols. of the *Transactions of the Pathological Society of London*, were thus named by Virchow. They are generally visible only with the aid of the microscope, but sometimes are large enough to be seen with the naked eye, and now and then attain some size. Usually they are round or oval, and present a concentric, laminated arrangement, which is made more apparent by the action of acetic acid. They often have a yellowish tinge. In appearance corpora amylacea somewhat resemble starch-granules, and they are tinged brown or bluish by the action of iodine upon them. These bodies were formerly supposed to be composed of starch, and hence their name. This is not the case, however, and their actual chemical composition is not clearly known; it probably differs in different structures. Bodies resembling corpora amylacea in appearance and arrangement have been found in various parts, but they have attracted most attention in connexion with the nerve-centres, being particularly observed when these are the seat of atrophy or degeneration; they are especially seen in the choroid plexus.

FREDERICK T. ROBERTS.

**CORPORA QUADRIGEMINA**, Lesions of.—The facts of comparative anatomy and experimental physiology tend to show that the corpora quadrigemina (*corpora bigemina* or *optic lobes* of the lower vertebrates), though related to the optic tracts, do not correspond in their development to the eyes or oculo-motor apparatus, and appear to be largely concerned in those functions, such as equilibration and locomotor co-ordination, which are independent of the cerebral hemispheres.

The facts of human pathology, though not opposed to these data, cannot be made the basis of very precise conclusions as to the diagnostic indications of disease of these ganglia, as it is exceedingly rare to find disease such as local softening limited to

this region anatomically or functionally, and hæmorrhage is unknown.

The corpora quadrigemina are, however, not infrequently involved in lesions which invade neighbouring parts, such as meningitis—simple and tubercular, tumours, especially of the pineal gland and the middle lobe of the cerebellum, &c.

It is stated by experimenters on the lower animals (pigeons, &c.) that when the anterior tubercles, which are more especially connected with the optic tracts, are destroyed, vision is abolished, and that if the lesion is unilateral, the blindness occurs on the side opposite the lesion.

It is questionable whether such results occur in man, apart from indirect implication of the optic tracts (neuritis). Dr. Bastian, however, has recorded a case of total blindness, in which the cause proved to be softening limited to the anterior tubercles of the corpora quadrigemina (*Paralysis from Brain Disease*, p. 115).

It has been found experimentally in animals, and also in man, that atrophy of the opposite tubercle ensues when the eye has been destroyed. Irido-motor action is also paralysed by destruction of the corpora quadrigemina, a result which, however, is stated not to occur unless the injury is more than superficial and implicating the oculo-motor nuclei.

Disturbances of equilibration and co-ordination also result from lesion of the corpora quadrigemina. These are attributed to implication of the subjacent tracts, and, according to Lussana and Lemoigne, more particularly to lesion of the subjacent superior cerebellar peduncle. For his own part, the writer thinks that all attempts at differentiation are pure hypotheses, and from the nature of the question must remain so. But, from whatever cause, there is no doubt that the disturbances alluded to do occur.

Irritation of the corpora quadrigemina on one side causes dilatation of the pupil and a hemiopisthotonos of the opposite side, which becomes general if the irritation is prolonged or bilateral, the head being retracted and the legs extended, trismus also being very marked. According to Lussana and Lemoigne unilateral lesion of the corpora quadrigemina causes an incurvation of the trunk and gyration to the side of the lesion. This would agree with the effects of irritation, being naturally a reversal of the phenomena. Tumours implicating the corpora quadrigemina are generally associated with a reeling gait, which is an early symptom, and also frequently with symptoms of ophthalmoplegia from implication of the oculo-motor nuclei. The affection of the ocular muscles is unequal, according to the degree in which the respective nuclei may be affected by the lesion (Nothnagel). In a case reported by Dr. Duffin (*Trans. Clin. Soc. of London*, vol. ix.), which

the writer had an opportunity of investigating, of tumour of the pineal gland, which, besides passing forward into the third ventricle, pushed underneath the aqueduct of Sylvius, stretching and causing atrophy of the corpora quadrigemina, the symptoms, in addition to those of cerebral tumour—namely, violent occipital headache and double optic neuritis—were double vision followed by loss of sight, vertigo, specially marked when the eyes were open, staggering gait, and tendency to retraction of the head and rigidity of the dorsal muscles. The pupils were large and sluggish. These symptoms, to a certain extent, resemble those caused by tumour in the middle lobe of the cerebellum, and it is a question how far these latter may be due to mechanical irritation of these ganglia. But we may infer that such a combination of symptoms as the above points to lesion of the corpora quadrigemina, or of the middle lobe of the cerebellum, though we cannot be certain of the absolute limitation of the lesion.

Naturally the same secondary affection of the functions of the cerebral hemispheres occurs from tumours situated in this region, as in tumours of the middle cerebellar lobe.

Pressure on the veins of Galen leads to dropsy of the cerebral ventricles, and its consequences on the cerebral circulation and functions.

D. FERRIER.

**CORPULENCE** (*corpus*, a body).—An undue accumulation of fat in the body. See OBESITY.

**CORPUSCLE** (*corpusculum*, a little body).—In physiology and pathology this word is generally used as synonymous with cell. See CELL.

**CORPUS STRIATUM**, Lesions of. The corpus striatum of English anatomy and pathology comprises various structures which have received special names—namely, the *nucleus caudatus*, or intra-ventricular nucleus, which is exposed to view by laying open the lateral ventricle; the *nucleus lenticularis*, or extra-ventricular nucleus, consisting of three divisions, and subjacent to the convolutions of the island of Reil; together with the anterior division of the *internal capsule*, or peduncular expansion, which connects the cortex and crus cerebri.

This differentiation is necessary, as the effects of lesion of the corpus striatum will differ according to whether the grey matter alone, or the internal capsule, is involved.

Physiological experiment has not succeeded in defining the respective functions of the ganglionic masses of the corpus striatum. The experiments of Aronsohn and Sachs, Ott, Hale White, and others seem to show that lesions of these ganglia in animals cause a rise of several degrees in body temperature from increased heat-production.

The corpus striatum is especially liable to lesion from embolism or rupture of its blood-vessels. These are furnished principally by the middle cerebral artery, which in the first part of its course sends off numerous straight twigs, which sink into the anterior perforated space, and supply this ganglion and the adjacent part of the optic thalamus. Owing to their position, and direction as regards the main current, they are easily ruptured or blocked up, and owing to their being of the nature of 'end arteries,' and almost destitute of anastomoses with other cerebral arteries, embolism rapidly leads to softening of the regions which they nourish.

**SYMPTOMS.**—The symptoms of lesion of the corpus striatum may be divided into three groups or stages.

*First stage.*—This includes certain symptoms which are more or less transient, and depend chiefly on the suddenness of the lesion and functional disturbance of other parts.

To the latter belong the symptoms usually accompanying an apoplectic seizure (apoplexy), as also the loss or diminution of sensation on the opposite side of the body, which sometimes occurs in consequence of pressure on, or functional interference with, the sensory tracts of the internal capsule by effusion into the corpus striatum. The symptoms due to the suddenness of the lesion of the corpus striatum, as such, are complete paralysis of every voluntary movement on the opposite side of the body, occasionally varied by convulsive spasms of the paralysed side, and conjugate deviation of the head and eyes towards the sound side. This latter symptom is due to the centres for the head and eyes of the opposite hemisphere suddenly losing their antagonists. The temperature of the paralysed side is, as a rule, higher than that of the sound side. The total paralysis and flaccidity of the opposite side of the body, and conjugate deviation of the head and eyes, are transient symptoms, lasting from a few hours to a day or two.

*Second stage.*—This stage includes those symptoms which continue for a variable period, after those depending on the suddenness and disturbing effect of the lesion have passed off. They constitute the common type of hemiplegia or paralysis of voluntary motion on the side opposite the lesion. The face, arm, and leg, and to a certain extent the thoracic and abdominal muscles on the one side of the body, are affected. The paralysis does not affect all these parts equally. As a general rule it may be stated that those movements are most affected which are most independent of those of the opposite side, and which are most complex and delicate. Hence the movements of the hand and arm are more affected than those of the face or leg, owing to the fact that these are more commonly exercised in associated or

alternating action with those of the other side. The facial paralysis is most marked in the lower facial region. The orbicularis palpebrarum is more or less paretic, but never paralysed to the extent which occurs in Bell's or true facial paralysis, depending on lesion of the portio dura. The angle of the mouth on the paralysed side hangs lower, and the tongue deviates slightly to the paralysed side. The weakness of the facial muscles is best brought out when the patient smiles or tries to whistle. The face then becomes drawn to the sound side. While some volitional control may have been acquired over the leg, the hand and arm remain perfectly motionless.

In the process of recovery, the leg recovers before the arm, and as a rule the recovery proceeds from the proximal to the distal end of the limb, the shoulder and hip movements being regained before those of the hand or foot. The flexors regain their power before the extensors.

The sensibility of the paralysed parts is unimpaired. The superficial reflexes are diminished, the deep (tendon) reflexes increased.

The faradic contractility of the muscles is unimpaired; occasionally it is increased rather than diminished.

The muscles do not undergo atrophy except by disuse.

The temperature of the paralysed limbs, which at first is usually increased, is generally found to be lower than that of the sound side, to the extent of a degree, more or less.

Recovery may take place from all the symptoms of this stage, within a period varying from weeks to months, or the patient may pass into the third stage.

*Third stage.*—The special symptoms of this stage are the occurrence of what is termed 'late rigidity' in the paralysed limbs, a condition of evil import. This rigidity shows itself most frequently in the arm, but it is common enough in both limbs. The rigidity affects the flexors more particularly, and causes the limb to assume a position in which the flexors predominate. It is variable in degree, and at first is capable of being overcome. At first, also, it is remittent, tending to give way when the patient abstains from volitional efforts or from excitement, and seems almost gone on waking from sleep or when the patient yawns or stretches himself. Gradually it assumes a more intense form, and the limb becomes permanently fixed and rigid.

After death, this condition is found to coincide with descending sclerosis of the motor tracts of the brain and spinal cord. The degeneration proceeds from the seat of lesion downwards through the crus, pons, and pyramid of the same side, and then across to the posterior part of the lateral column of the spinal cord on the paralysed side. Frequently, also, a similar track of degeneration is found

on the inner aspect of the anterior column of the spinal cord, and occasionally also in the lateral column on the same side as the brain-lesion (Charcot, Türck, &c.)

Even during the rigid stage, there is, as a rule, no trophic degeneration of the muscles or annihilation of faradic contractility, though the muscles waste from disuse unless artificially stimulated. But in some rare instances, as Charcot has shown, the secondary degeneration invades the anterior cornua of the spinal cord, in which case amyotrophy or trophic degeneration of the muscles ensues.

There is no recovery from this condition.

*Variations and complications.*—Though general hemiplegia of the opposite side, without affection of sensation, is the type of disease of the corpus striatum, certain variations and complications have been observed, some of which still require elucidation.

When sensation is permanently affected along with voluntary motion, we have reason to regard the lesion as not confined to the corpus striatum, but as implicating also the posterior part of the internal capsule and the thalamus, an occurrence by no means rare. When the lesion affects only the grey matter of the nucleus caudatus, the hemiplegia is, as a rule, comparatively slight and transitory.

Similar affection of the nucleus lenticularis is said to produce more marked paralysis than that caused by affection of the nucleus caudatus, but also not of a permanent kind. When, however, the lesion causes rupture of the anterior two-thirds of the internal capsule, the hemiplegia is most marked and most enduring. It is this lesion only which gives rise to secondary degeneration of the motor tracts and permanent rigidity.

Cases are on record in which lesions of the corpus striatum have given rise, not to general hemiplegia of the opposite side, but to monoplegia, such as paralysis of the face or of one or other limb. But these monoplegiæ are without doubt due to limited lesions of the internal capsule, which, as the researches of Franck and Pitres and of Beevor and Horsley have demonstrated, contains the motor tracts corresponding to the differentiated centres of the cortex.

Cases are also on record of paralysis occurring on the same side of the body as the lesion. The real existence and explanation of such exceptional occurrences are still *sub judice*, and though various explanations may be suggested, it is well to wait for further instances, carefully investigated by accurate modern methods, before pronouncing definitely on the question.

D. FERRIER.

**CORRELATION.**—This term is used in medicine almost exclusively in reference to the ætiology of disease, and in this relation principally in regard to the zymotic diseases. The term 'correlation of the physical forces'

may be taken as implying that the several forces are capable of being converted into or of giving place to one another when they are permitted to act under certain conditions, and that they are all related to a common cause. It is very much the same idea that is implied by the term 'Correlation of the Zymotic Diseases,' a subject which has been discussed in a separate work by A. Wolff.

It is contended that the several contagious diseases of a general type (the exanthemata) do not, necessarily and in all cases, reproduce their like; but that occasionally, the contagious particles thrown off from the same sick person suffering from one of the diseases (*e.g.* scarlatina) may suffice to engender one or more different kinds of disease, according to the mode in which this matter operates—that is, according as it may be swallowed, taken into the system through the pulmonary surface, or through some other mucous membrane or skin-abrasion. These diseases are said to be correlated, therefore, on account of this assumed relation to a common cause—a specific contagium. According to this notion the seat of primary action and the constitutional condition of the patient are factors which largely influence the form of disease that ultimately manifests itself as a result of the contact of any given contagium. This view has at present scarcely passed beyond the stage of an ingenious speculation—though it is one which is by no means unworthy of further attention, opposed though it may be to many current doctrines as to the specific and unalterable nature of contagia.

Of late years, moreover, evidence in support of some such doctrine has been strengthening. Thus, to take one example, it has now been shown (*see* Dowdeswell's Report in *Brit. Med. Journ.*, July 19, 1884) that two very distinct forms of septicæmia exist of an extremely contagious character, which may take their origin from common putrefactive material introduced into different parts of the body. The one, known as 'Davaine's septicæmia,' may be set up by introducing two or three drops of putrid blood beneath the skin of an animal; whilst a totally different form of the disease, known as 'Pasteur's septicæmia,' may be induced by introducing a similar quantity of the same putrid blood into the peritoneal cavity of another individual of the same kind of animal. The late Professor Hueter, indeed, held, as a result of experimental observations, that the contagia of many diseases are derivable from common septic organisms and their alkaloids, by virtue of alterations taking place under particular conditions in the process of putrefaction, by which means the common organisms take on 'specific modes of activity.' Instances of such conversion, as well as of the interchangeable morphological characters of bacilli and micrococci associated with different contagious processes, are cited

by him and also by Professor Fokker of Groningen. See *Trans. Intern. Med. Cong.* 1881, vol. i. pp. 329-334.

Diseases may be said to be correlated also when they are severally related to the same cause acting with different degrees of intensity. Thus it is held by some epidemiologists that the plague is only a malignant form of typhus; that yellow fever is due to a more intense form or action of the same poison as suffices at other times to engender intermittent or remittent fever; and that summer diarrhoea, cholera, and cholera are also but different manifestations of one common though variable cause.

Similarly it is held by many surgeons that ordinary surgical fever, pyæmia, and septicæmia are correlated effects, due to the action of the same poison in different degrees of intensity. They maintain that the appearance of one or other of these morbid states after a surgical operation is dependent in part upon differences in the constitutional condition of their patients, and, moreover, that these forms of disease are further linked to one another by numerous intermediate states. This point of view has been both strengthened and extended by some of the experimental researches of Dr. Burdon Sanderson. He found that the subcutaneous injection of different portions of the same inflammatory product, executed at the same time, would often produce quite different effects upon different animals of the same species. In the one a typical septicæmia proved rapidly fatal, in another a slower pyæmic process was established, whilst in a third animal the still more chronic process of so-called tuberculosis was set up. Here again we seem to get out of the region of speculation into that of fact. See *Trans. Path. Soc. of London*, 1872, pp. 303-308.

The term 'correlation' is only applicable to communicable diseases, otherwise its leading signification, namely, convertibility, could not be fulfilled. Hence it is that, though very many diseases may arise from the operation upon different individuals of some common cause (such as exposure to cold), the maladies which may result from such a cause no one would think of speaking of as correlated.

H. CHARLTON BASTIAN.

**CORROSIVE SUBLIMATE, Poisoning by.**—See MERCURY, Diseases arising from.

**CORYZA** (κόρυζα, a running from the head).—A synonym for nasal catarrh. See CATARRH.

**COUGH.**—SYNON.: *Tussis*; Fr. *Toux*; Ger. *Husten*.

**DESCRIPTION.**—The *act of coughing* consists in one or more abrupt forcible expirations, preceded by contraction of the glottis. First a deep inspiration is taken, the glottis is closed for a moment, and then

it is opened by the pressure of the air forced out by the combined action of the thoracic and abdominal expiratory muscles. With the air thus suddenly expelled, any foreign matter that may be in the larynx or bronchi is driven into the pharynx or the mouth.

**ÆTIOLOGY.**—The immediate cause of cough is the presence of an irritant, mechanical or sympathetic, affecting the surface of the air-tubes or the nerves that supply them, and it is the object of the cough to remove this source of irritation.

The sensibility of the respiratory surfaces is greatest at its commencement—the glottis being an ever-watchful janitor. It may be increased by congestion or inflammation, or by the continued act of coughing. Even the mere inhalation of cool or dry air may, in asthma or bronchial congestion, be sufficient to excite cough. The result of the irritation is to increase the natural secretion, and to alter its characters. See EXPECTORATION.

Cough may be due to numerous reflex causes, such as gastric irritation, ear-disorder, aneurysmal, glandular or other pressure on the vagus, recurrent, or sympathetic nerves. The act may also be caused by a long uvula or enlarged tonsil; a granular state of the pharyngeal or laryngeal mucous membrane; polypi or other foreign bodies in the larynx, trachea, or even in the external auditory meatus; various affections of the bronchial tubes—for example, undue dryness, hyperæmia, alteration in the quality or quantity of the bronchial secretion, or inflammatory affections; inflammation of the lung or pleura; or tubercle, cancer, or other growths in or near the lung.

**DIAGNOSIS.**—Cough is not a disease to be treated, but a symptom to be traced to its source. An inspection of the pharynx and larynx, and a physical examination of the chest, will generally suffice to detect the cause.

The character of the cough is often quite pathognomonic—*e.g.* the 'whoop' of whooping cough; the 'bark' of hysteria; the catching, painful cough of pleurisy; the slight 'hack' of early phthisis, and the equally distinctive cough of advanced phthisis with laryngeal ulceration; the tearing cough of empyema threatening bronchial fistula; the loud clanging cough due to pressure on the trachea or laryngeal nerves; the spasmodic, suffocative cough of asthma.

The 'tightness' or 'looseness' of cough, indicating the absence or presence of secretion, is a valuable guide in diagnosis and treatment.

The *absence of cough* is no proof of the absence of serious lesion; thus, while the presence of a few granulations in the lung is often productive of incessant and uncontrollable cough, long-continued destructive disease may exist without it.

**TREATMENT.**—Before prescribing for a

cough it is of course essential to ascertain its cause; and the simplest and most innocuous remedies should be first used. The routine treatment of cough by sedatives is highly injurious. The secretions which ought to be removed are thus locked up, and the irritation, which would have been transient, becomes established.

If the tonsils are found much enlarged, or the uvula pendulous and irritating the epiglottis, caustics or the guillotine will remove the evil. If a granular state of the pharyngeal membrane exists, dependent on torpid or engorged abdominal viscera, gout, or hepatic obstruction, it may be treated by local astringents and general deobstruents.

A lax or congested state of the laryngeal membrane, due to overwork of voice, or the undue direction of attention to the vocal apparatus (clergyman's sore-throat), is best treated, according to the writer's experience, by the local application of a solution of zinc sulphate—ten grains to an ounce of water, or of iodine dissolved in spirit and olive oil. Undue dryness, simple hyperæmia, or hyperæsthesia of the respiratory mucous tract, may often be relieved by the act of sipping and slowly swallowing cold water, or the decoction of Iceland moss, fruit lozenges, gum arabic, liquorice, or linseed tea. Sucking ice or inhaling steam is very often all that is needed. In the early stage of catarrhal sore-throat, chlorate of potassium in crystal, or in the form of lozenge, used in moderation, should not be neglected. The use of glycerine of tannin, or nitrate of silver dissolved in glycerine (half a drachm to one ounce), is of more service in relaxed throat than alum or tannin gargle; indeed, the free use of well-selected lozenges has rendered the employment of gargles well-nigh obsolete. The former can be constantly, the latter but seldom applied.

*Medicinal treatment.*—If it is desired to increase the fluidity of the secretion, squill or ipecacuanha may be used, or, better still, tartar emetic in small doses, which is best given in effervescence with carbonate of ammonium and citric acid. It must not be forgotten that syrups and nauseating expectorants are apt to do harm by enfeebling or disturbing digestion. Tincture of aconite, in three-drop doses, is often of value in allaying irritable cough, especially when fever is present. Gelsemium is a more recent remedy for the same purpose. Of the direct sedatives, morphine is the most valuable; it proves of service in very small doses,  $\frac{1}{12}$  gr. in a lozenge being often adequate. Conium, with or without morphine, suits some persons; hydrocyanic acid still more; and Indian hemp is also of value. The bromides, in combination with chloral, have gained great repute; the latter should be given with caution. The power of the bromide of ammonium in allaying spasmodic cough is re-

markable. An emetic of ipecacuanha, sulphate of zinc, or mustard may be useful in relieving cough, by expelling secretion when this has accumulated in large quantity. If cough causes vomiting, food should be taken in small quantities, fluids should be limited, and a little capsicum or spiced brandy 'stays the stomach.'

*External applications.*—The use of counter-irritants must not be neglected. In the inflammatory stage of bronchitis, for instance, linseed and mustard poultices, and, in the later stages, iodine, are of great use; and in some cases the application of a small blister or vesicating fluid is a remedy not to be forgotten.

*Inhalations.*—Infusion of hop as an inhalation is a useful calmative; iodine is indicated in relaxed conditions in strumous subjects. Chloroform (10 to 15 minims) mixed with eau de Cologne, and inhaled from a handkerchief, is useful in other cases. By means of the spray-inhaler, many non-volatile preparations may be applied to the respiratory passages. A solution of carbonate of sodium is very useful in liquefying tenacious secretion. Tannic acid, alum, perchloride of iron, and nitrate of silver, are all valuable. Of sedatives, henbane, conium, camphor; and of antiseptics, sulphurous and carbolic acids, are serviceable as inhalants. The vapour of tincture of iodine, 15 drops to 2 ounces of water, in the steam draught inhaler, is often useful. In chronic granular disease of the pharyngeal and laryngeal mucous membranes, the sulphurous waters of Aix-la-Chapelle, Aix-les-Bains, and St. Saviour in the Pyrenees, when inhaled in an atomised state, are of distinct service.

Patients may be *taught how to cough* as follows: Try to suppress the inclination, until the secretion that causes the cough is within reach, then take a deep and deliberate inspiration, and the accumulated phlegm is removed at a single effort. By inhaling steam from a hot sponge or a basin of boiling water on first waking from sleep, the inspissated secretion, which is apt to be difficult to move, may be easily loosened and expelled. An ipecacuanha lozenge may serve a similar purpose. E. SYMES THOMPSON.

**COUNTER - INDICATION.** — See CONTRA-INDICATION.

**COUNTER-IRRITANTS.**—SYNON.: Fr. *Contre-stimulants*; Ger. *Gegenreizmittel*.

**DEFINITION.**—The term 'counter-irritation' implies any irritation artificially established with a view to diminish, counteract, or remove certain morbid processes which may be going on in a more or less remote part of the system. The substances employed in establishing this state are called counter-irritants, and may be classified as follows,

according to the degree of their action:—  
 1. *Rubefacients*. 2. *Epispastics, vesicants, or blistering agents*. 3. *Pustulants*.

Although some therapeutists have of late been disposed to question the value of counter-irritants, on the theoretical ground of inability to explain their mode of action, yet there is not wanting evidence, both from clinical observation and physiological experiment, that irritation in one part of the body may affect the functions and nutrition of other parts. That stimulation of the vessels of the surface can influence decidedly the circulation of deeper parts has been demonstrated by Dr. Brown-Séguard; for he found that irritation of the skin of the back, over the kidneys, caused a contraction of the arteries supplying those organs. Zülzer found that when cantharides collodion was painted over the back of a rabbit for fourteen days the vessels underneath the skin were congested, whilst the deeper parts, including the lungs, were pale and anæmic. From these experiments we can understand how a blister may relieve a sudden internal congestion in the lungs or brain, and how it may act in restoring tone to dilated and paralysed capillaries.

*Revulsion* and *derivation* are both examples of counter-irritation. In the first, the induced morbid action is set up in a part remote from the primary disease, as when mustard poultices are applied to the feet in an attack of apoplexy; in the second, derivative action is set up in the neighbourhood of the primary malady, as when a blister is placed on the back of the neck for the relief of cerebral disorder.

**1. Rubefacients.**—**ACTION.**—These remedies, applied to the skin, produce local warmth and redness from increased flow of blood in the cutaneous vessels. The local hyperæmia thus induced subsides gradually on ceasing to employ the rubefacient; but sometimes, when the action of this has been prolonged, the epidermis may peel off, and more or less local soreness remain. Rubefacients are usually quick in action; their local after-effects are trifling; and they may, therefore, be applied without injury over a large extent of surface.

**ENUMERATION AND APPLICATION.**—Examples of rubefacients are found in Ammoniacal liniments or embrocations; Mustard plasters and the Compound Liniment; Volatile Oil of Mustard; Oils of Turpentine and Cajuput; and Iodine. Hot water is at times applied on a sponge or flannel to produce a speedy counter-irritant and derivative effect in relieving sudden internal congestion and spasm, as in the early stage of croup, laryngitis, and laryngismus stridulus. The Cataplasma Sinapis, or mustard poultice, is a useful and rapidly acting rubefacient in inflammation, spasm, and neuralgic pain. Sir Alfred Garrod recommends a very useful

sinapism, made by mixing 10 minims of volatile oil of mustard with 1 oz. of spirit of camphor, and sprinkling this on impermeable piline. Rigollot's mustard leaves, or the Chartæ Sinapis (B.P.), applied to the skin, produce a speedy rubefaction of the surface. Linimentum Sinapis Compositum is also a very active rubefacient. Vinegar should not be added to mustard poultices; but by mixing some oil of turpentine or a little powdered capsicum in a mustard poultice, its rapidity of action as a stimulant and rubefacient can be greatly increased. Where, on the other hand, a gentle stimulation with warmth and moisture on the surface are desired, as in some cases of pneumonia, a linseed-meal poultice may be used with its surface sprinkled lightly over with mustard meal. Generally twenty minutes is as long as an ordinary mustard poultice can be safely borne on the skin. In persons who have a very delicate skin, a layer or two of muslin should be placed between the mustard application and the surface of the body. In applying mustard poultices to those who are unconscious of pain, caution is necessary, for it has happened that the poultice being left on for a long time has produced dangerous ulceration and sloughing of the surface. A mustard foot-bath is at times employed with a view to a revulsive and counter-irritant effect. To prepare a mustard bath, two tablespoonfuls or more of mustard should be tied in a cloth and agitated well with cold water; then hot water may be added to make the bath. It is found by experiment that cold water extracts the active principle or volatile oil of mustard far better than very hot water does.

**USES.**—Rubefacients are used in chronic inflammation and irritation of the mucous surfaces, as in bronchitis, and irritation about the air-passages. Troublesome cough, in cases of phthisis, is often relieved by applying tincture of iodine, or acetic acid and turpentine liniment, to the chest. Rubefacients are of service in removing lingering irritation about a joint, their use also tending to promote the absorption of chronic thickening or effusion in the joint; but friction with a rubefacient liniment over a joint must not be employed till all active inflammatory action has entirely ceased. Various degrees of persistent counter-irritation may be maintained by applying, after the skin has been well cleansed with soap and water, the Emplastrum Picis or Emplastrum Calefaciens of the Pharmacopœia. A mustard plaster applied to the nape of the neck has proved useful in cases of irritable brain with sleeplessness. The same application made to the foot or great toe is a valuable revulsive where gout attacks more important organs. A mustard plaster has the advantage over a blister in rapidity of rubefacient action; and, from the sharp pain caused, the mustard plaster is preferable when it is a matter of

moment to rouse one who is in a state of lethargy or torpor from narcotic poisoning by opium, or alcohol, or from coma in the course of a fever. Where we wish to exercise a prolonged action over chronic inflammation in an organ, we should use a blister rather than a sinapism.

**2. Vesicants, Epispastics, or Blistering Agents.**—**ACTION.**—A blister acts primarily as a rubefacient and powerful stimulant to the cutaneous vessels. The papillæ of the skin become reddened and raised; minute vesicles soon appear on the elevations; and these, gradually coalescing, form a bleb, or large vesicle, between the true skin and epidermis, containing an albumino-fibrinous fluid.

**ENUMERATION AND APPLICATION.**—The agent most commonly employed for blistering purposes is the *Cantharis* or Spanish fly; but there are others that have been used for a similar object. Glacial Acetic Acid applied to the skin produces intense redness and pain, with rapid vesication, but its action may extend too deeply as a caustic, and cause a troublesome sore. *Liquor Ammoniz* dropped on a piece of lint, applied to the skin, and covered with a watch-glass, very soon causes redness and rapid vesication in most persons. This is a good way of raising a blister when it is desired to apply powdered morphine endermically to relieve severe pain. The *Liquor Epispasticus* of the *Pharmacopœia*, applied with a brush, soon raises a blister on the surface. *Blistering Collodion* is used for a similar purpose, but is slower in action.

The application of blisters should not be made directly over an inflamed part. There is some evidence to show that a strong stimulus applied very near an inflamed organ may increase the paralytic dilatation of its capillaries, and so add to the disease. Blisters should not be applied where the skin is loose, nor over any prominence of bone, nor to the breast during pregnancy. It should moreover be borne in mind that the cantharidine of a blister may be absorbed by the skin, and act on the kidneys, producing strangury and bloody urine. This accident may be obviated by sprinkling powdered camphor over the blister before placing it on the skin, or a thin piece of silver paper may be interposed. In persons of feeble vitality, a blister left on for too long a time has been known to induce dangerous sloughing.

When vesication is specially desired, there is no need to leave the blister on for twelve hours or more, for it may be removed at the end of six or eight hours, and a warm linseed poultice applied. If the blister be opened, which is best done by pricking the most dependent part with a disinfected needle, sweet oil and cotton-wool is the best dressing. The practice of maintaining a blister as a running sore or *exutoire*, by applying irritating oint-

ments, is not often resorted to now. The process causes great pain and exhaustion of the system, and is one rather of depletion than of counter-irritation. In the case of children, blisters should be used with caution, being kept on for about one hour or till the skin is well reddened, when they should be replaced by a poultice. It is well not to open the blister, as the effused serum forms the best dressing covered with cotton-wool. By following this plan the child is saved much worry and pain.

**USES.**—In its primary effect a blister acts as a local stimulant, but when it remains on long enough to produce extensive vesication and discharge of serum, it acts as a depletive and depressing agent. This primary and secondary action of blisters has been much insisted on by the late Dr. Graves of Dublin, who found great benefit in cases of fever with apathy and prostration from the application of *flying blisters* to various parts of the surface. Thus a blister over the præcordial region, kept on for about one hour, and then removed, was observed to rouse and stimulate a flagging heart. In other cases the flying blister might be placed at the chest or back, or else behind the head on the neck. Care should be taken not to leave the blister on long enough to cause actual vesication, and the size of the blister should be fairly large.

Vesication by a blister is of service in many brain-affections attended with congestion and tendency to serous effusion, such as in the chronic forms of hydrocephalus, and non-tubercular meningitis. In hysterical paralysis narrow strips of blister placed completely round the affected limb have proved curative. A strip round the throat may cure nervous aphonia. A strip of blister one inch wide may possibly sometimes stay the spread of erysipelas along a surface. In cases of pleuritic or pericarditic effusion the repeated application of blisters to the chest-wall is of manifest advantage. In effusions into joints (*hydrarthrosis*) blisters aid absorption; and it has seemed to the writer that absorbent remedies, such as iodide of potassium, often begin to do good as soon as a blister appears to have once set the absorptive process in action. In the joint-affections of acute rheumatism, 'the blister treatment' has attracted notice. Armbands and wristlets of blister-plaster are applied close to the inflamed joints during the fever, and the serous discharge from the blister is kept up by means of linseed-meal poultices.

In the obstinate acid vomiting of gouty patients, a blister over the epigastrium often gives relief. Some forms of neuralgia, as for example pleurodynia, may yield to a blister over the seat of the pain. At times obstinate pleurodynia, or mastodynia, can be relieved by flying blisters applied in the vertebral groove on the affected side, where a tender

spot can often be detected on pressure. Blisters should be avoided in cases of renal and vesical inflammation, as the absorption of the cantharidine may increase the mischief.

**Counter-irritation by heat.**—The skin can be rapidly blistered by applying a hammer—'Corrigan's hammer'—a small flat iron heated in a spirit-lamp or boiling water. The skin is tapped for a few seconds with the hammer, just to induce redness of the part. In some forms of rheumatism, neuralgia and spinal weakness, this practice has been followed by satisfactory results. Vesication of the skin by the *ferrum candens*, or hot iron, has been used in chronic joint-disease.

**Moxas.**—Moxas were used for the purpose of causing severe counter-irritation. European moxas were made either with cotton-wool soaked in solution of nitrate of potassium, or of the pith of the sunflower, which naturally contains this salt. A wet rag was placed on the skin; in the centre of this was a hole in which the lighted moxa was placed, which gradually burned down to the skin and produced an eschar, which in due time separated by suppuration. In spinal affections, and in some forms of paralysis of the sensory and motor nerves, moxas are said to have done good; but their application is very painful, and now they are seldom employed.

**3. Pustulants.**—ACTION.—The agents belonging to this class of counter-irritants produce a pustular eruption on the part to which they are applied, and their use is not recommended.

ENUMERATION AND APPLICATION.—Among pustulants may be placed Croton Oil, Tartarated Antimony, and strong solution of Nitrate of Silver. When croton oil is applied to the skin, it acts as an intense irritant, producing an eruption which is at first papular, but very soon becomes pustular. The oil is best employed in the form of the Linimentum Crotonis of the Pharmacopœia. Tartarated Antimony in the form of the Ointment, or in hot aqueous solution, is a powerful counter-irritant, producing pustules which resemble those of variola. When applied thus it may, by becoming absorbed, induce symptoms of gastro-enteritis. It should not be applied to parts usually uncovered, as the pustules leave marks behind them; and under all circumstances the remedy, being a painful one, must be used with caution. Strong solution of nitrate of silver will produce pustulation, but it is seldom employed for this purpose.

**Issues.**—Issues have long been used as counter-irritants. An issue is formed by placing on the skin a piece of adhesive plaster, in a hole in the centre of which a fragment of caustic potash is inserted. The caustic causes an eschar, and when this has come away, an issue-pea is placed in the

cavity left by the eschar; this pea acts as a foreign body, and keeps up suppuration. One drachm of pus may be discharged daily by an issue; more than this is too great a drain on the system. An issue requires to be dressed daily, and when it has been long open and running it must not be healed too suddenly. Issues over the spine have been found useful in chronic spinal disease; and in some chronic brain-affections, with hyperæmia and congestive tendency, an issue in the back of the neck or in the arm is of service, but is not now much used.

**Setons.**—A seton is made by passing a narrow-bladed knife under a fold of skin and then carrying a few silk threads through the incision by means of a probe or long needle. The threads remaining in the wound prevent it from healing, and maintain a free purulent discharge. Setons are used for the same purposes as issues, and they have proved useful in certain intractable forms of headache—the seton being inserted in the skin of the neck. Setons have been used in cases of cystic bronchocele with thickened walls; and, as counter-irritants, in chronic inflammation of the bladder and uterus; in chronic skin-diseases of an obstinate character; in inflammations of the eye, with ulceration of the cornea; and in the early stages of pulmonary phthisis.

In acute affections issues and setons are never employed, and they should not be placed over any part where there is much movement, as a troublesome sore may be the result. It is necessary to bear in mind that issues, setons, and pustulants are, like blisters, when kept on long enough to induce serous discharge, of the nature of evacuations. They carry off nutrient material from the blood, and therefore are more or less depressing and exhausting to the system, and their repeated or protracted employment will tend to induce the irritative fever of debility.

JOHN C. THOROWGOOD.

**COUP DE SOLEIL** (Fr.).—A synonym for sunstroke. See SUNSTROKE.

**COW-POX.**—See VACCINIA.

**COXALGIA** (*coxa*, the hip; and *algos*, pain).—Pain in the hip-joint. See JOINTS, Diseases of.

**CRACKED-METAL** or **CRACKED-POT SOUND.**—SYNON.: Fr. *Bruit de pot fêlé*.—A peculiar sound elicited by percussion, and resembling that emitted on striking a broken jar or a metallic vessel. See PHYSICAL EXAMINATION.

**CRADOCK**, in Cape Colony.—See AFRICA, SOUTH.

**CRAMP.**—SYNON.: Fr. *Crampe*; Ger. *Krampf*.—This name is applied to certain painful varieties of tonic spasm. In its most familiar form it affects the calves of the legs,

coming on principally at night, on the occasion of some slight movement of these parts. The affected muscles, mostly on one side, contract with such energy as to give rise to a board-like rigidity, together with sensations of an agonising character. The attack rarely lasts more than a minute or two, though it may more or less speedily recur. It is perhaps best cut short by a vigorous but steady voluntary contraction of the opposing extensor muscles of the foot. Where it is more obstinate than usual, firm pressure around the thigh or upon the great sciatic nerve sometimes gives relief. Cramp is often associated with some irritation of the stomach or of the intestines, especially in children or delicate nervous persons. In this way it is produced not infrequently when arsenic in medicinal doses has been continued for some time, and is beginning to exert some slightly poisonous effect upon the system. In a more general form it often occurs, to a marked extent, in cholera. Localised, painful, cramp-like contractions of muscles may also be due to irritation of anterior root-fibres, either in their intra-medullary or extra-medullary course. Other forms of painful spasm are by no means common, if we except colic. See SPASM; and THOMSEN'S DISEASE.

H. CHARLTON BASTIAN.

**CRANIOTABES** (*cranium*, the skull; and *tabes*, thinning).—A morbid condition of the cranium in children, consisting in spots of local thinning of the occipital, parietal, and (rarely) the frontal bones, as well as in general abnormal flexibility of the osseous tissue. See SKULL, Diseases of.

**CREPITANT** (*crepito*, I make a noise). When applied to a body, this word signifies that it is capable of yielding the sensation or sound of crepitation. It is also associated with a râle, to indicate a peculiar character which it possesses. See PHYSICAL EXAMINATION.

**CREPITATION** (*crepito*, I make a noise).—A sensation or sound of crackling. It may be observed in morbid states of the bones, joints, or subcutaneous tissue; but the term is more frequently applied to a physical sign connected with the lungs. See PHYSICAL EXAMINATION.

**CRETINISM** (*crétin*, Swiss *patois* for *chrétien*, a Christian).—SYNON.: Lat. *Cretinismus*; Fr. *Crétinisme*; Ger. *Cretinismus*.

DEFINITION.—A condition of idiocy arising from endemic causes, associated with imperfect development and deformity of the whole body, varying however in degree.

This condition of physical and mental degeneracy is not limited to any nationality. It obtains in the great mountain-chains of Europe, Asia, and America. In Europe it

is met with in the valleys of Switzerland, Savoy, and Piedmont; and it abounds in the neighbourhood of Salzburg, Styria, and the Tyrol. It is less frequently met with in the Pyrenees and in the valleys of the Auvergne in France. Even in England it has been met with in various parts, among others in the dales between Lancashire and Yorkshire. Although more frequently met with in valleys, it is not unknown on plains which are subject to inundations.

ÆTIOLOGY.—The conditions for the development of cretinism are hereditary predisposition; the action of deteriorating influences on the parents, such as unwholesome dwellings and non-nutritious diet; and accidental causes operating on the infant during the period when its physical and intellectual life are developing. The last-named causes are atmospheric and possibly geological conditions, peculiar to special localities. Humidity of the soil and air in valleys where there is little interchange of the atmosphere, and the existence of magnesian limestone in the soil, are probably the most potent factors. Cretinism is not met with as an endemic disease on elevated plateaux, nor in cold countries where sudden changes of temperature are uncommon. Goitre is a frequent accompaniment of cretinism, and would appear to be developed under the same conditions. The cases of cretinism met with in England present features which are indicative of a scrofulous origin. There is a condition of idiocy associated with arrest of growth and development at the period of first dentition, not infrequently met with in England, which has been termed *sporadic cretinism*. Some of these cases have been traced to alcoholism on the part of the progenitors, and are usually associated with an absence or atrophy of the thyroid body, and with the development of loose fatty tumours in the supra-clavicular spaces. They remain permanent children, both physically and mentally. They have a strong sense of the ridiculous, and are fond of exciting ridicule in others by their grotesque facial expressions. There is reason to believe that children become cretinoid when taken to reside, at the period of their early development, in localities where the disease is markedly endemic. There are numerous well-attested instances of healthy women living during their pregnancy in cretinic districts bringing forth cretinoid children, who, removing from such localities, propagate healthy children. By far, however, the greater number of cretins arrive at their helpless condition by successive steps of degeneracy in their ancestors. It has been thought that cretinism is due to premature ossification of the cranial sutures, especially of the pheno-basilar suture; and that this is caused by drinking water largely charged with lime. It is impossible, however, to regard this premature ossification, when it

does occur, as other than one of the outcomes of the malady, not its cause. Moreover, there are numerous examples where the synostosis is deferred instead of being premature.

**ANATOMICAL CHARACTERS.**—Pathological anatomy shows that the bones of the cranial vault are thickened and without diploë. The basilar groove is generally wanting. The foramina for the passage of arteries and nerves are somewhat smaller than natural. The occipital fossæ are flatter than usual, as if the flattening had resulted from a compression of the cranium from above downwards. Every variety of deformity of the cranium is met with of the brachycephalic type. The brain is usually small, unsymmetrical, pale, and infiltrated with serum. Premature synostosis is occasionally met with at the spheno-basilar suture, and with it a rectangular form of the base of the skull; this, however, cannot be regarded as a constant condition.

**DESCRIPTION.**—The degrees of cretinism are numerous. A residence in one of the valleys where this affection exists, enables one to trace the various steps of degeneracy, commencing with those who are taking part in the industrial life of the valley, down to the helpless individuals who are leading only a vegetative existence.

The typical cretin presents a very marked physical conformation. He is stunted in growth, rarely reaching five feet in height. His skin is of a tawny yellowish hue, thickened and wrinkled; and looks as if too large for the body. There is also a great increase of subcutaneous areolar tissue. His tongue, large and thick, with hypertrophied papillæ, always displays lessened power of co-ordination; and often hangs from the mouth. The mouth is partly open, margined by thick fissured lips, and with the saliva running over the chin. The face is large; the lower jaw is drooping, and its angle obtuse. The eyes are often affected by strabismus, obliquely placed, and small; and the lids are commonly puffy. The belly is pendulous from the laxness of the skin. The lower limbs are generally short and deformed, and the gait is waddling. The head is deformed, the forehead retreating, the top flat, and the occipital region ill-developed. The cranium is brachycephalic. The nose is broad and flattened. Puberty is often delayed to the twentieth year. The mammæ in the female are large and pendulous; the same remark applies to the genitals in the male. The intellectual faculties are imperfectly developed. The cretin is often unable to speak, and his hearing is frequently defective. The affection is usually associated with more or less enlargement of the thyroid gland. His viability is low, few living beyond thirty years of age. The sexual functions are abnormal; masturbation is

frequent; and the subjects of cretinism are often im potent.

**DIAGNOSIS.**—The diagnosis of cretinism may be made in childhood, from the slowness of the development of the body, the stupid expression, the postponement in the evolution of the teeth, and of the ossification of the fontanelles and sutures, the tawny yellow colour of the skin, the thick and goitrous neck, the slaving, and the delay of speech and of walking.

**TREATMENT.**—This consists in removing the child as early as possible from the circumstances which have produced the disease. He should be taken to a locality where the soil is dry and porous, and should have frequent baths with friction to the surface of the body. The diet should be of the most nutritious kind—a diet into which animal food largely enters. The administration of cod-liver oil and of the lacto-phosphates of lime and iron is indicated. Early education should be commenced as to habits of cleanliness, followed by systematic physical exercise of the various muscles. All intellectual advancement must be sought for through the improvement in every way of his physical condition. The lower animal life may thus be supplemented, if earnest efforts are used, by increased capacity for rational enjoyment, and a more or less useful existence.

J. LANGDON-DOWN.

**CRIME, IRRESPONSIBILITY FOR.**—**HISTORICAL SUMMARY.**—A medical opinion as to the condition of an accused person is often necessary in order to determine whether he or she can be held accountable for criminal acts. Such an opinion generally depends on the presence or absence of insanity, or on the connexion which may be traced between this mental condition and the act in question. In the article on legal insanity it is explained that it is only within a comparatively recent period that insanity has been admitted as an excuse for crime, except in those comparatively rare cases in which, as Justice Tracey expressed it in 1723, a person does not know what he is doing, 'no more than an infant, a brute, or a wild beast' (see *INSANITY, LEGAL*). This view fairly represents the state of public and of legal opinion until the later years of the eighteenth century. The subsequent enlightenment of the public mind did not receive juristic expression until the trial of Hadfield in 1800, when Erskine first enunciated the doctrine, that 'delusion where there is no frenzy or raving madness is the true character' of such insanity as implies irresponsibility. The most important case in the history of this question was that of Bellingham, who was executed in 1812 for shooting Mr. Spencer Perceval. In this case Lord Chief Justice Mansfield said, that if a person labouring under mental derangement were capable in other respects of distinguish-

ing right from wrong, 'he could not be excused for any act of atrocity which he might commit.' 'It must be proved beyond all doubt,' he added, 'that at the time he committed the atrocious act he did not consider that murder was a crime against the laws of God and nature.' The trial of MacNaughton in 1843 for the murder of Mr. Drummond led to the most authoritative statement of the law which has ever been obtained in this country. MacNaughton was acquitted on Chief Justice Tindal's direction that the point for the jury to consider was whether 'at the time the act was committed' the accused 'had that competent use of his understanding as that he knew that he was doing by the very act itself a wicked and wrong thing.' The general application of this doctrine would have greatly enlarged the area of irresponsibility, and its enunciation at that time produced considerable surprise and even consternation. The matter was indeed regarded as so urgent that the House of Lords immediately ordered a series of questions to be laid before the fifteen judges with the view of settling the state of the law. In the answers to these questions it was in substance laid down that, to entitle an accused party to acquittal on the ground of insanity, it is necessary that he be either of diseased mind, and at the time he committed the act not conscious of right or wrong; or, that he be under some delusion which made him regard the act as right. But this statement has been far from effecting a final settlement of the question.

Most writers on medical jurisprudence have insisted that the real criterion of responsibility is the freedom of the will, or the power of the individual to control his actions. This has been more or less advocated by Esquirol, Marc, Ray, Pagan, Jamieson, Mittermaier, and von Krafft-Ebing. Esquirol dwells strongly on the importance of the freedom of the will. Ray includes it in the comprehensive statement which has received the approval of so many medical jurists. 'Liberty of will and action,' he says, 'is absolutely essential to criminal responsibility, unless the constraint upon either is the natural and well-known result of immoral or illegal conduct. Culpability supposes not only a clear perception of the consequences of criminal acts, but the liberty, unembarrassed by disease of the active powers which nature has given us, of pursuing that course which is the result of the free choice of the intellectual faculties.' Pagan observes that the 'loss of control over our actions, which insanity implies, is that which renders the acts which are committed during its continuance undeserving of punishment.' Jamieson puts the question: 'Had the lunatic at the time of committing the deed a knowledge that it was criminal, and such a control over his actions as ought, if it existed, to have hindered him from committing it?' Dr. Taylor says: 'The power which

is most manifestly deficient in the insane is generally the controlling power of the will'; and he expresses the opinion that 'we have here a fair criterion on which responsibility or irresponsibility may be tested.' Dr. Bucknill's view is substantially the same. 'Responsibility,' he says, 'depends upon power, not upon knowledge, still less upon feeling. A man is responsible to do that which he can do, not that which he feels or knows it right to do. If a man is reduced under thralldom to passion by disease of the brain he loses moral freedom and responsibility, although his knowledge of right and wrong may remain intact.' The latest German code puts responsibility upon the same basis. 'An act is not punishable,' according to it, 'when the person at the time of doing it was in a state of unconsciousness; or of disease of the mind, whereby free volition was prevented.' Mittermaier and von Krafft-Ebing sanction the attempt to render the meaning of 'free volition' more definite by describing it as made up of *libertas judicii* and *libertas consilii*—freedom of judgment and freedom of choice. Casper somewhat obscurely defines criminal responsibility as 'the psychological possibility of the efficacy of the penal code.' Mr. Balfour Browne, a recent writer, gives as the best definition 'a knowledge that certain acts are permitted by law, and that certain acts are contrary to law, and, combined with this knowledge, the power to appreciate and be moved by the ordinary motives which influence the actions of mankind.' Dr. Guy thinks that every person who is insane must be regarded as wholly irresponsible, and that the law of England ought to be assimilated to that of France in the declaration that: 'Il n'y a ni crime ni délit lorsque le prévenu était en état de démence au temps de l'action.' Mr. Warren, on the other hand, suggests that a person should not be held irresponsible unless he were as 'unconscious of his act as a baby.' Dr. Maudsley and others hold that the determination of responsibility in cases where insanity is alleged depends on whether a connexion can or cannot be traced between existing disease and the act.

Insanity has been pleaded as an excuse for acts of theft; but such cases are rare, and never occur except where the social position of the accused adds importance to the decision. Indeed it may almost be said that the plea is never raised except in order to avoid capital punishment. Hence it is, that in the discussions which have arisen the question has been intimately associated with the law of murder and homicide. A special inquiry into the state of this law by a Committee of the House of Commons<sup>1</sup> has consequently given occasion to the enunciation of important views as to the legal relations of

<sup>1</sup> Report of Select Committee of the House of Commons on the Homicide Law Amendment Bill, July 21, 1874.

insanity and responsibility. Evidence was furnished to the Committee by Lord Chief Justice Cockburn, Baron Bramwell, Mr. Justice Blackburn, and Sir James Fitz-James Stephen. The immediate object of the Committee was to examine a bill drawn by Sir James Stephen for the codification of the law of homicide. In the clause of the bill which deals with the relations of disease and responsibility, homicide is stated to be 'not criminal if the person by whom it is committed is at the time when he commits it prevented by any disease affecting his mind—(a) from knowing the nature of the act done by him, (b) from knowing that it is forbidden by law, (c) from knowing that it is morally wrong, or (d) from controlling his own conduct.' But it is stated to be 'criminal, although the mind of the person committing it is affected by disease, if such disease does not in fact produce one of the effects aforesaid in reference to the act by which death is caused, or if the inability to control his conduct is not produced exclusively by such disease.' It was, however, proposed in the bill that, 'if a person is proved to have been labouring under any insane delusion at the time when he committed the homicide, it shall be presumed, unless the contrary appears or is proved, that he did not possess the degree of knowledge or self-control hereinbefore specified.' That is to say, where delusion exists, the burden of proving moral capacity would be shifted, the prosecutor having to prove its existence, instead of the accused having to prove its absence. The opinions elicited during the inquiry showed that the law is regarded by legal authorities as being at present too uncertain in its operation, and as failing to recognise some of the most important elements in the question. The divergent character of the recommendations which were made showed, however, that legal opinion is much divided, not only as to the proper relations of insanity and crime, but also as to the essential elements of responsibility. In the meantime, therefore, the statements of the fifteen judges after the MacNaughton case remain the chief exposition of the English law where insanity is pleaded in excuse for crime.

PRESENT STATE OF THE QUESTION.—It is necessary, in order to justly appreciate the present aspect of the subject, thus to trace its more recent history, and it might be useful to present an estimate of the comparative value of the several tests or criteria which have been proposed for the determination of cases in which insanity has been alleged. These criteria may be broadly summarised in the following six propositions. According to one view a person should be held irresponsible for an act if at the time of committing it (1) he laboured under insanity of any kind or degree; according to another, if (2) he laboured under delusion;

or (3) if he was ignorant of right and wrong; or (4) had not power to appreciate and be moved by ordinary motives; or (5) had lost the controlling power of the will; or (6) if the act is traceable to, or its nature has been determined by mental disease affecting the agent. The last of these views is the only one to which fatal objection may not be raised, both on the theoretical and practical sides. The others are all too vague to be of much advantage; and they rather tend to introduce new difficulties than to remove those already existing. The proposition therefore which seems to approach nearest to a solution of the difficulty is that irresponsibility must be admitted whenever the act is traceable to, or its nature is determined by mental disease affecting the agent. It will of course be understood that under such a rule the term 'mental disease' must be held to include both congenital and acquired disorders; arrest of development being as much a morbid condition as functional or structural change. This view of the subject may not be ultimately accepted in the precise terms of the proposition here given; but the principle on which it rests seems to afford the only safe basis upon which we can go. As has already been shown, it has not hitherto been regarded in this light by the majority of the judges; but there have been indications of late years that judicial views are tending in that direction. The late Lord Wensleydale and others have given sanction to the principle in their judicial statements. And the late Lord Justice-General of Scotland (Ingis) gave definite expression to it in one case (*Brown*, Sept. 1866). He told the jury that the main question was 'whether the prisoner was in such a state of insanity at the time, as not to be responsible for the act which he had committed;' and in order to constitute such insanity he said that 'it must be clearly made out that at the time of committing the act the prisoner was labouring under mental disease in the proper sense of the term, and that that mental disease was the cause of the act.' In America the doctrine has been frequently acknowledged, but never more fully and tersely than by Judge Doe, of New Hampshire (*State v. Pike*), who comprehensively defined the medical relations of both criminal irresponsibility and civil incapacity, when he stated that a 'product of mental disease is neither a contract, a will, nor a crime.' One important point is to prevent persons from being punished for actions which are the direct outcome of pathological processes. But it is of equal importance to avoid the adoption of a principle which would make the existence of slight mental irregularities incompatible with responsibility. There does not appear to be any danger of this in acting on the principle which is here enunciated. For it lies in the very nature of the cases in which

the doctrine could be applied, that the condition to which it is proposed that irresponsibility should be attached must be one which is known to exhibit itself in acts of serious and even criminal character. If the trained observer of disease is able to recognise in an act—which is ordinarily followed by severe punishment—a direct result or a characteristic feature of a morbid process, of the existence of which there may otherwise be sufficient proof, the question of responsibility cannot present any serious difficulty. It may admit of doubt whether a person is responsible for not controlling his actions, or for not knowing right from wrong, or even in some cases for the harbouring of a delusion. But once let it be proved that an act is the natural result of a disease under which a person is known to labour, and the question must be practically removed from the field of discussion. Before, however, the principle can be accepted as fully satisfactory, it is necessary to inquire whether its application would permit insane persons to be held responsible who ought not to be so considered. In other words, are there states of insanity in which a person is irresponsible for acts to which he has not been predisposed or impelled by the insanity? It is perhaps impossible to give such an answer to this question as would be both definite and complete; but for practical purposes we think it may be answered in the negative. Where the insanity is of such a nature that it does not modify the whole conduct, we believe it will be found in practice necessary to admit the existence of responsibility for acts where there is no demonstrable connexion between them and the mental disease. The insane persons who on this principle might be held responsible, would be found solely among those whose irresponsibility could only be admitted after very searching inquiry, and whose insanity was of that kind and degree which has often been declared by medical writers to be consistent with responsibility. It is not to be supposed that under this rule difficulties would cease. It would often be hard to show in cases of actual disease that there was good reason for believing in its existence, or that it was really contributory to the act committed. This, however, would not result from any defect in the principle, but from that imperfection of our knowledge which renders the perfect application of any principle impossible. Let the task of the medical witness be limited to the demonstration of facts indicative of disease and its consequences, and he will at least be acting quite within his special province, and might expect that reasonable weight would be attached to his opinion. And if juries were instructed that the law does not hold a person responsible for acts committed under the influence of disease, it is scarcely conceivable that any one would be found guilty where good cause

had been shown even for the reasonable supposition of such an influence. Whatever may be the view ultimately adopted, it would seem to be in every way desirable that the attention of the medical expert should be confined to the elucidation of the medical facts, and that he should not be required to deal with questions which are legal and abstract, and in no way specially medical. The condition known as *diminished responsibility* has not been alluded to in this article. It is only indirectly recognised by British law, and therefore, though much is to be said in favour of its recognition, it is unnecessary to deal with the subject here. JOHN SIBBALD.

**CRISIS** (*κρίσις*, a decision, a turn).—SYNON.: Fr. *Crise*; Ger. *Krise*.—Crisis is a term applied to the rapid defervescence of an acute febrile disease. It has wandered somewhat from its original meaning, which was 'judgment'—primarily an operation in the mind of the observer, but reflected upon the phenomena observed. The converse term, employed to designate a gradual subsidence of fever, is *lysis*.

Crisis formed at one time the basis of an important medical doctrine. Certain days from the onset of the disease on which the crisis commonly occurred were considered to be propitious. The seventh day was especially favourable, while the sixth was the most unfavourable; speaking generally, the odd numbers or the multiples of 7 were propitious, and the even numbers and such odd numbers as stood near multiples of 7, such as 19, were unpropitious. The preparation also for a crisis was indicated, and the critical day foretold, by remissions perceptible some days previously. The whole morbid process of fever was represented as a process of elaboration by which a *materies morbi* was prepared for expulsion, and an essential feature of the crisis was a critical evacuation, by means of which this was eliminated.

The doctrine of crisis and even of critical days was not pure imagination or superstition, but was founded originally on careful observation. In times when nothing was known of the organic lesions which give rise to fever, and in countries where a large proportion of the diseases were of a malarious origin, it would afford data for prognosis and conduce to appropriate treatment; and at the present day in hot climates a crisis is anxiously looked for in febrile attacks on a given day, and, as is well known, a critical fall of temperature and improvement in the general symptoms precede in pneumonia improvement in the physical signs.

In order to constitute a true crisis the defervescence should occupy less than forty-eight hours, and it often takes place in a much shorter time. The fall of temperature should be accompanied by a corresponding reduction in the frequency of the pulse, and

should coincide with a feeling of relief and a return of strength; the skin will be warm and soft, the tongue moist, and there will be indications of reviving appetite; there may or may not be a critical evacuation, but the secretions will become more natural in amount and character.

This favourable mode of termination of an acute febrile disease is more common than is usually supposed. The circumstances under which it is most likely to occur are when the attack begins abruptly and the temperature rises rapidly, the natural course of the disease being short and not attended with organic lesions, such as will of themselves keep up fever. After twenty-one days, termination by crisis is not to be expected. The more marked the onset—as, for example, by a definite rigor—the more rapid the rise of temperature, and the greater the height to which it reaches, the greater the probability of an early critical termination.

**OCCURRENCE.**—The diseases in which the conditions favouring a crisis are realised, and in which this mode of termination is observed, belong to various classes.

Among the *specific fevers*, eruptive and continued, it occurs frequently in variola, but in severe cases it is interfered with by the febrile disturbance excited by the eruption. In measles it is very common. In scarlet-fever a true crisis is seen only in mild cases, though the onset of this disease is peculiarly abrupt. The mode of termination of typhus is essentially critical, but as a rule the crisis is not sharp. Relapsing fever affords the best examples of crisis, which is moreover attended by a critical evacuation in the form of profuse perspiration, the temperature sometimes falling 10° F. in as many hours, and the patient passing from a state of extreme suffering and oppression to almost perfect ease and comfort. In enteric fever *lysis* is the mode of termination.

Remittent fevers often present crises, which may be true and curative, or false and illusive; and the sun fever and common continued fever of hot climates, and tropical diseases generally, have a tendency to fever running high very early and breaking abruptly at a critical period.

In this country *feverish colds*, attacking the throat or taking the form of influenza or catarrh, often terminate critically in three or four days. Erysipelas may so end, but at a later and less definite period. The sharp *febrile attacks* which sometimes occur *after childbirth* often exhibit a very decided crisis. In *pneumonia* the natural termination is by a well-marked crisis, which may take place as early as the fifth day, or be deferred to the ninth, after which a critical termination is not to be expected, and the suspicion may be entertained that the case is not one of frank pneumonia, the prognosis becoming grave. As has been already stated, the general im-

provement precedes the indication by physical signs of resolution in the inflamed lung. Pleurisy is said also to terminate critically, but it is not in the same definite way as pneumonia.

The *critical evacuations* which entered into the original notion of a crisis are really a common attendant. The most common is a profuse warm perspiration, which may occur whatever the disease may be. Occasionally the evacuation is a copious flow of urine, or it may take the form of diarrhoea. Epistaxis or hæmorrhoidal flux is a more rare and doubtful critical evacuation. A common critical phenomenon is a prolonged, sound, and refreshing sleep.

The question whether or not a favourable crisis affects the odd rather than the even days has been a frequent subject of dispute, and it still remains undecided. It is not, however, of any importance; but another point handed down with the doctrine is worthy of attention, namely, that indications of an approaching crisis are often given two or three days beforehand in slight remissions of fever. By the presence or absence of such remissions at a certain period of the attack, or by a continuous rise of temperature where a remission might be expected, important prognostic information may be afforded and indications for treatment obtained.

**THERAPEUTIC INDICATIONS.**—The main therapeutic deduction from a study of crisis as a termination of acute disease is, that we should not hastily interfere with the reactions by which the system adjusts itself to altered conditions or meets the incidence of the causes of such disease, but contribute to their completion. We do not assume the existence of a *vis medicatrix* tending invariably to the restoration of health; but we must recognise the power inherent in a living organism to respond by internal changes to external influences, and to regain the balance when this has been disturbed. In this process a certain cycle of changes must be gone through, and the great opportunity for treatment of an active kind, should any be required, arises when the course, direction, and probable duration of these changes are known, and when agencies can be brought to bear at a given moment, which will contribute to bring about the appropriate critical evacuation or a critical sleep, through which a return to a normal condition would naturally be effected. WILLIAM H. BROADBENT.

**CRITICAL.**—Having relation to a crisis. See CRISIS.

**CROUP** (Scotch, *croup* or *roup*, hoarseness).—SYNON.: *Cyananche trachealis*; Fr. *Croup*; Ger. *Croup*.

**DEFINITION.**—An acute febrile affection, occurring in childhood, accompanied by inflammatory swelling of the mucous mem-

brane of the larynx, and in some rare cases by the formation of a fibrinous false membrane.

**ÆTIOLOGY AND PATHOLOGY.**—The symptoms of the most usual form of croup, as described below, are due to an acute catarrh of the laryngeal mucous membrane. The condition is, in fact, a simple catarrhal laryngitis, and is not accompanied by the formation of any false membrane. The mucous membrane swells by reason of the congestion of its blood-vessels, and the pouring out of inflammatory exudation into its tissue. Mucus, too, is prone to accumulate on its surface. These conditions are precisely the same as occur in the ordinary catarrhal laryngitis of adults. But in the child the glottis is much narrower than in adults, and the swelling of the mucous membrane from inflammation is greater, and more rapid in reaching its height. Moreover, the larynx is in children more irritable, and therefore its muscles are more easily excited to spasm. Thus when a simple catarrhal laryngitis makes its appearance in a child, it is very shortly followed by laryngeal stenosis, partly from the swelling of the mucous membrane, partly from accumulation of mucus, and partly too from spasm of the laryngeal intrinsic muscles. The condition described is most often due to exposure to cold and damp, especially at a time when the body is heated. It is caused also by scalds of the larynx, when unduly hot fluids are drunk by accident, by wounds, by the irritation of noxious gases, and by the presence of foreign bodies in the larynx. It is more common in male than in female children, and is usually met with between the ages of two and nine years.

But there also exists an inflammation of the larynx which is accompanied by the formation of a false membrane, and it is in respect to this condition that so much discussion has arisen, and even now doubt exists in the minds of some observers. It is, of course, well known that in diphtheria the characteristic membrane may spread from the pharynx, its usual site, into the larynx, and in doing so may produce all the symptoms of so-called croup, in addition to those due to the diphtheria proper. But much of the confusion which existed as to the pathological entity of croup resulted from the non-recognition of those cases of diphtheria in which the local lesion had its primary seat in the larynx. Pathological observations, however, have now placed it beyond doubt, that such cases really exist. Death may occur entirely from the laryngeal stenosis so produced, as in all other and simple obstructions of the larynx. But the diphtheritic nature of the affection is obvious from the other possible terminations of such cases, and from the general symptoms present at the height of the affection. Thus death may also be due to the heart-failure,

or to the general blood-poisoning, characteristic of diphtheria. Again, in the acute stages of such a disorder, symptoms may appear, such as great asthenia, excessively frequent pulse, hæmorrhages from various parts, albuminuria, and a formation of diphtheritic membrane on sores far removed from the larynx, which symptoms, while inconsistent with a diagnosis of non-specific membranous inflammation of the larynx, are frequent associations of diphtheria when it occurs in its ordinary form. Moreover, when, in fortunate cases, the acute symptoms have passed away, after an interval the characteristic paralysis of diphtheria may show themselves. The facts of contagion are also of importance, for from such cases of membranous laryngitis, other cases may arise, in which the diphtheritic membrane appears first in the pharynx, and the disease runs its ordinary and unmistakable course. The teachings of bacteriology are perhaps, as yet, not sufficiently definite, in reference to this point, for them to be accepted as absolute proof. It may, however, be noticed that Löffler's diphtheria-bacillus (not merely the micrococci which may be found in any decaying membrane) has been demonstrated equally well in those cases of diphtheria which presumably began in the larynx, as in those where the membrane was limited to the pharynx. Thus it must now be admitted that the local lesions of diphtheria may be limited to the larynx; and, in the opinion of the writer, under this category must be placed by much the larger number of cases formerly styled 'membranous croup.' The description of diphtheria of the larynx will be found in the article **DIPHTHERIA**.

There remains still a certain proportion of cases of membranous laryngitis which are not due to the action of the diphtheritic poison. Some of these are produced as secondary complications by the poisons of other specific fevers, and amongst these are to be mentioned scarlet-fever, small-pox, measles, enteric fever, and whooping-cough. Yet this question is before us, whether a membranous laryngitis may occur without the action of any specific poison. The older writers assumed that this occurred in a large number of instances, and the prevalence of this view led to the report by a Committee of the Royal Medical and Chirurgical Society in 1879, upon the Relations of Membranous Croup to Diphtheria. That by far the larger number of cases of membranous laryngitis are, in the opinion of the writer, due to diphtheria has already been stated above. But the evidence is also indisputable that the affection may occasionally arise from other and simple sources. Thus, the late Dr. Hilton Fagge<sup>1</sup> recorded, amongst others, cases where both the fauces and air-passages were covered with false membrane, from a

<sup>1</sup> Guy's Hospital Reports, 1877, p. 345.

scald of the throat by boiling water, and again from burns of the fauces; where a foreign body in the bronchus had caused membranous laryngitis; where a similar appearance followed a cut throat, other diseases of the pharynx or larynx not diphtheritic, and pneumonia.

Mr. R. W. Parker,<sup>1</sup> too, has recorded a case where false membranes formed in the larynx after a scald. The Committee mentioned above, reported that cases had fallen under their notice, where 'membranous affection of the larynx and trachea had shortly followed exposure to cold, but their knowledge of the individual cases was not sufficient to exclude the possible intervention or co-existence of other causes.' General pathology would not negative the view that an inflammation, usually simple, might occasionally become membranous. As is mentioned under DIPHTHERIA in respect to the true diphtheritic membrane, an important factor in its production is the occurrence of coagulative necrosis of the epithelial cells, a condition which may arise from the intense action of an irritant usually productive only of catarrhal inflammation. We have a formation of false membrane, analogous to that of the assumed membranous and non-diphtheritic croup in the affection known as plastic bronchitis, in the intestine in dysentery, in the mouth in syphilitic and sometimes in simple stomatitis, and on various mucous surfaces in septic disorders. Further, membranous inflammation has actually been produced experimentally by the injection of acrid irritants into the larynx of animals. *A priori*, therefore, there seems no reason why a membranous laryngitis should not occur without the intervention of a specific poison. Practically, however, it must be concluded, from the evidence at our disposal, that such cases are exceedingly rare, and that false membranes do not form in the laryngitis which forms the basis of what is met with as the simple non-diphtheritic croup of children. It must here be stated, that in modern times the late Dr. Hilton Fagge held an opposite view, though he with great care and fairness considered the arguments against the position he maintained. He believed not only that, in many cases of croup which recovered, false membrane had formed without being detected, but also that a non-specific membranous croup might spread from the larynx to the pharynx and fauces, causing fresh deposits of false membrane on those parts, which most other observers would assume to be characteristic of diphtheria. He suggested that the term 'Croup' should be applied only to cases of membranous and non-diphtheritic laryngitis, while those where no membrane was formed should be known as 'Spurious Croup.'

In former accounts of the pathology of

<sup>1</sup> Clinical Transactions, 1875, p. 144.

croup, differences are described between the croupal and the diphtheritic false membranes. There are, indeed, differences between the false membranes found on the laryngeal and pharyngeal mucous membranes respectively; but these are due merely to the differences in structure of the mucous membrane attacked. The false membrane is in both cases essentially the same in structure and in its mode of formation. What is said of the diphtheritic membrane will apply to the membrane rarely found in croup, so that no detailed description of the latter need be given here. See DIPHTHERIA.

SYMPTOMS.—In accordance with the view expressed above, little will be said under this heading about cases other than those of non-membranous, or as the late Dr. Hilton Fagge styled it, 'spurious' croup. The child affected by this disorder may suffer from general catarrhal symptoms for a time, varying from a few hours to two or three days. More commonly before the actual attack he appears quite well at bed-time, though very often it may afterwards be ascertained that some time during the day he has been exposed to cold or damp, or has sustained a chill. In a few hours, however, he wakes suddenly, as if frightened, breathing hurriedly with loud, hoarse, inspiratory laryngeal stertor. He coughs frequently, making a harsh, metallic noise, but at first removing no mucus from the larynx. He cries, and perhaps speaks, in a shrill, cracked tone, which soon gives place to a mere whistling sound. The character of the breathing, voice, and cough gave rise to the Scotch vernacular name for the disease, 'croup,' which was adopted by Dr. Home in 1765, when describing the affection, and which is now employed by most nations as a scientific term. The child is very restless, tosses his body and limbs about incessantly, clutches at his throat as if to remove the obstruction there, and seizes any surrounding objects with his hands. The face is at first flushed, the eyes bright, and the expression exceedingly alarmed and anxious. Soon, however, the flush becomes dusky, and gradually pallor supervenes, while the whole body is covered with a cold sweat. The feet, and sometimes the hands, not infrequently assume the position found in tetany, and in extreme cases general convulsions may occur. The pulse is frequent and feeble, the temperature is slightly raised. The difficulty with which air enters the chest is obvious from an observation of the patient's position and respiratory movements. The head is thrown backwards, the chin held out prominently, and this position is accentuated at each inspiration by a contraction of the sterno-mastoid muscles. All the extraordinary muscles concerned in chest-expansion are brought into play, and the nostrils dilate at each breath. Yet, in spite of the extra force employed, the thorax is not filled with

air. During inspiration the supra-clavicular fossæ, the lower ribs, the epigastrium, and even the lower portion of the sternum are depressed. The yielding of the chest-wall is, as might be expected, more pronounced in rickety children, and in such subjects the whole of the ribs may be seen to give way at the sides as each breath is drawn. If the chest and larynx be auscultated, at first nothing is heard but laryngeal sibilus; afterwards rattling noises are perceived in the larynx, due to the presence of mucus, while in the chest rhonchi may be discovered. The cough, which was at first dry, as the attack continues displaces small pellets of thick, tough mucus; sometimes threads of this substance are expectorated, and have been mistaken for false membrane.

Usually, after a time varying from half an hour to two or three hours, the difficulty in respiration subsides, and the child falls asleep again, but not quite free from symptoms indicative of his ailment. His face is somewhat flushed, and there is some degree of pyrexia. The restlessness does not quite disappear even in sleep. The hands and feet frequently retain their tetanoid position. The breathing is still husky and somewhat hurried, and an occasional slight cough has the clanging character noticed during the paroxysm; during the next day, or for the next two or three days, the breathing is hurried and somewhat stertorous, and the attack may return during the next and even succeeding nights. For some time after recovery is apparently complete, any slight cough is prone to assume a 'croupy' character, just as after an attack of whooping-cough the characteristic whoop may at times recur. After one attack, too, or series of attacks, the child may again and again suffer from repetitions of the disorder, even for several years.

As has been stated, this non-diphtheritic membranous laryngitis is not always idiopathic. In such cases the signs of laryngeal stenosis are added to those of the original disease or injury. It is convenient, therefore, to discuss them in the article on DIPHTHERIA, where they will be found in the section upon Diphtheria of the Larynx.

PROGNOSIS.—The prognosis of non-membranous croup is almost invariably favourable in so far as the actual attack is concerned. In spite of the apparent severity of the symptoms, they nearly always subside naturally. The friends of the patient should, however, be warned of the probability that the attack will be repeated at times for some years after its first appearance. Membranous croup, on the other hand, is a much more serious matter. At any moment the false membranes may accumulate or curl up in the larynx, and entirely close the glottis. Nor is the danger merely that of the laryngeal affection. The laryngitis occurs in only severe cases of the specific fevers, and the

blood-poisoning of the original disorder, just as in diphtheria, is usually a most fatal condition.

TREATMENT.—As soon as the attack of non-membranous croup has commenced, the child should be placed in a warm bath, to which preferably a little mustard has been added. Sponges, wrung out of water as hot as can be borne, should be applied over the larynx, and renewed as often as they become cold; or a mustard-leaf may be applied here. An emetic, too, is useful. This may be administered in the form of ipecacuanha wine, in doses of one teaspoonful every ten minutes until vomiting comes on. Sir William Jenner recommends that afterwards a dose of calomel and jalap should be given. It is well, too, to place the child in a tent-bed, under the cover of which the nozzle of a steam-kettle is introduced.

Prophylactic treatment is of importance, when the disease has once shown itself. In cold weather the child should be wrapped up warmly, and protected from chills and draughts. At the same time, the larynx may be, as it were, accustomed to cold, by being sponged once or twice a day with cold water. General tonics, too, are of use in warding off future attacks.

The treatment of membranous croup is practically the same as that of laryngeal diphtheria. See DIPHTHERIA.

ROBERT MAGUIRE.

**CROUP, FALSE.**—A term commonly applied to laryngismus stridulus. See LARYNX, Diseases of.

**CROUPOUS, CROUPY** (Scotch, *croup*, hoarseness).—These terms were originally employed with reference to the peculiar crowing or stridulous character of the respiration, cough, and voice in certain affections of the larynx, and signified 'belonging to croup' in its clinical relations; for example, 'croupy cough,' 'croupy symptoms.' When morbid anatomy demonstrated the occurrence of a fibrinous exudation or false membrane upon the affected surface in a special form of croup, the word 'croupous' was used also to designate this false membrane; thus 'croupous exudation' and 'croupous membranes.' The application of the term was afterwards further extended; and it is now employed to indicate the process that leads to a fibrinous exudation in any situation whatever; such as 'croupous inflammation' and 'croupous pneumonia.' Thus the words 'croupous' and 'croupy,' which were originally associated with peculiar sounds, have come in a remarkable manner to express certain physical, chemical, and microscopical characters in the products of inflammation. See CROUP; DIPHTHERIA; and INFLAMMATION.

**CROWING CONVULSION.** — A popular synonym for laryngismus stridulus. See LARYNX, Diseases of.

**CRURA CEREBRI, Lesions of.**—In the crura cerebri lie the paths of communication between the cerebral hemispheres and the periphery. The motor or pyramidal tracts lie in the *foot* or ventral portion of the crus, which is separated from the dorsal portion, or *tegumentum*, by the *locus niger*. The pyramidal tracts of the internal capsule constitute only the middle third of the foot of the crus; the inner third connects the prefrontal lobe with the grey matter of the pons, and, indirectly perhaps, with the opposite cerebellar hemisphere (Flechsig); the outer third, which has been supposed by Meynert to constitute the sensory path, would appear, from the researches of Flechsig and others, to connect the occipito-temporal regions of the hemisphere with the grey matter of the pons, and probably with the cerebellum. The sensory tracts, in all probability, run in the tegumentum or dorsal division of the crus cerebri. The fibres of the third nerve pass through the internal, or mesial, aspect of the crus on their way to the oculo-motor nuclei in the floor of the aqueduct of Sylvius.

From the anatomical and functional relations of the crus cerebri, lesions of this region, such as may arise from hæmorrhage, pressure, &c., are apt to cause paralysis of motion and sensation on the opposite side of the body, as well as oculo-motor paralysis on the same side as the lesion. The degree of implication of the sensory and motor tracts and fibres of the third nerve varies in individual instances; but an *alternate paralysis*, in which there is some degree of oculo-motor paralysis on the one side and paralysis of the limbs on the other, is diagnostic of lesion of the crus. A typical instance of this form of paralysis has been recorded by Weber (*Med.-Chir. Trans.* 1863). In this case there was oculo-motor paralysis on the side of lesion and complete paralysis of voluntary motion, with partial paralysis of sensation on the opposite side of the body. A similar case has been recorded by Rosenthal (*Med. Jahrb.*, 1870). In this case there was, progressively, ptosis on the left side, right hemiplegia, anæsthesia and analgesia. The right third nerve became implicated later. After death a cyst was found in the left crus cerebri, together with a tumour of the size of a bean occupying the interpeduncular space, and compressing the right third nerve. In paralysis from lesion of the crus cerebri, there is, as a rule, also well-marked vaso-motor paralysis, and the temperature of the paralysed side may be two or three degrees above that of the other.

D. FERRIER.

**CRUSTA LACTEA** (*crusta*, a crust; and *lactea*, milk-like).—Milk-crust: a synonym for *eczema pustulosum* of the face and head, met with in infants at the breast. See ECZEMA.

**CRUVEILHIER'S PARALYSIS.**—

A synonym for progressive muscular atrophy. See PROGRESSIVE MUSCULAR ATROPHY.

**CUPPING.**—SYNON.: Fr. *Action de ventouser*; Ger. *Schröpfen*.

This is a mode of treatment sometimes employed to relieve congestion or inflammation of internal parts by drawing blood to the surface of the body. When the blood thus attracted to the superficial parts is actually abstracted from the body by means of incisions, the operation is called *wet-cupping*; and this has been described in the article BLOOD, Abstraction of. We shall here describe *dry-cupping*, in which no scarifications are made, the blood being simply drawn towards the surface by atmospheric exhaustion, and hyperæmia of the subcutaneous parts or organs thereby relieved.

Formerly cupping was extensively practised, but of late years it has fallen into disuse. In some respects it serves the purpose of counter-irritants, but has a more powerful effect than these; rapid and marked results being sometimes produced upon the circulation of inflamed or congested tissues.

**MODES OF APPLICATION.**—Dry cupping is performed as follows: The flame of a spirit-lamp, being allowed to burn for an instant in the dome of a cupping-glass, is quickly withdrawn, and the cup is then rapidly and evenly applied to the skin over the affected part. The heat expands the air contained in the glass cupola, and, owing to the contraction which ensues on cooling, the skin is forcibly sucked up into the cup. It is well first to sponge the skin of the selected spot with hot water, so as to render it more supple and vascular. Slightly moistening the rim of the cupping-glass helps to increase the degree of exhaustion.

An excellent modification of cupping, which has been demonstrated to the writer by Sir Richard Quain, is practised in the following way: Instead of allowing the cup to remain stationary after its application to the skin, as is usual, the operator dexterously slides it to and fro along the surface. When the operation is to be thus performed the amount of surface drawn into the glass must not be considerable. In this way a large tract of skin may be quickly rendered hyperæmic without effusion of blood into its meshes, as happens when the cups are stationary.

**PRECAUTIONS.**—Cupping-glasses should be applied where the skin is thick and cushiony, as over the loins, nape of the neck, pectoral region of the chest, &c., and not where bony prominences, or other irregularities, are likely to interfere with complete exhaustion. The edges of the glasses must not be so hot as to burn the skin.

**USES.**—Cupping may be advantageously employed in cases of sthenic cerebral congestion, the cups being applied to the nape of the

neck; in hyperæmia of the spinal cord; and in inflammation or congestion of the lungs, kidneys, or other viscera. In renal ischæmia it is eminently serviceable. This may be owing to the fact that the blood-supply of the skin of the loins is in intimate relation with that of the kidneys; the vascular supply to those organs being thus directly and immediately influenced. ALFRED WILTSHIRE.

**CUTIS, Diseases of.**—See SKIN, Diseases of.

**CUTIS ANSERINA** (*cutis*, the skin; and *anser*, a goose).—A state of roughness of the skin, resembling that of a goose when plucked, produced by prominence of the pores or follicles. It is due to contraction of the muscular structure of the corium, and is commonly occasioned by cold.

**CYANIDES, Poisoning by.**—See ANTIDOTE; and PRUSSIC ACID, Poisoning by.

**CYANOSIS** (*κυανός*, blue).—This, which is really not a disease, is the peculiar blue or more or less livid colour of the surface of the body, especially in certain parts, which is observed in several affections that interfere with the circulation and oxygenation of the blood. The condition is most commonly associated with certain forms of congenital malformation of the heart, and reaches its highest development in them, for which consequently cyanosis is not uncommonly used as a synonym. Lesser degrees of similar discoloration are, however, not infrequently noticed in cases of cardiac disease developed after birth, and they may also accompany pulmonary affections which materially obstruct the circulation. A cyanotic appearance is also one of the obvious effects resulting from all modes of suffocation, and it is observed in the collapse-stage of cholera. The upper half of the body may become extremely cyanotic as the result of obstruction of the superior vena cava. For the pathology of cyanosis, see HEART, Malformations of.

**CYNANCHE** (*κύων*, a dog; and *ἄγχω*, I strangle).—SYNON.: Fr. *Angine*; Ger. *Bräune*.

This word is used to express an inflammatory condition of the throat, or contiguous parts, in which difficulty of breathing or of swallowing exists, accompanied by a sense or feeling of choking. The term is used synonymously, more frequently on the Continent than in England, with *angina*; an affix, indicative of the seat or nature of the affection, being employed as a designation for each of the several forms or varieties of disease affecting the throat or adjacent parts. Such, for example, are the terms *cynanche laryngea*, or croup; *cynanche maligna*, or malignant sore-throat; *cynanche parotideæ*, or mumps; *cynanche pharyngea*, or inflammation of the pharynx; and *cynanche tonsillaræ*, or quinsy. See these several diseases.

**CYRTOMETER** (*κυρτός*, curved; and *μέτρον*, a measure).—An instrument for measuring the absolute and relative dimensions and movements of the chest-wall. See PHYSICAL EXAMINATION.

**CYSTICERCUS.**—See ENTOZOA.

**CYSTINE** or **CYSTIC OXIDE** (*κύστις*, the bladder).—A peculiar substance, occurring either in solution or in the form of small crystals in the urine, or as calculi in connexion with the urinary apparatus. See URINE, Morbid Conditions of; and CALCULI.

**CYSTITIS** (*κύστις*, the bladder).—Inflammation of the bladder. See BLADDER, Diseases of.

**CYSTOSCOPE** (*κύστις*, the bladder; and *σκοπέω*, I inspect).

**DEFINITION.**—An instrument designed for inspecting the interior surface of the urinary bladder.

**INTRODUCTION.**—Progress in the improvement of the means for inspecting the interior of hollow organs was slow from the beginnings of Bozzini (1807), Segalas (1826), Avery (1846), and Desormeaux (1853 and 1865), and of others by whose apparatus light outside the body was thrown through an inspecting tube on to the surface of the bladder. Among the many workers in endoscopy, Avery and Grünfeld (1874) were conspicuous for their originality and fertility of resource. Avery devised the means of reflecting light in sufficient amount to illuminate the urethra, and his apparatus was modified by Desormeaux and others, but greatly improved by Grünfeld. That surgeon described many conditions of the interior of the bladder in health and disease when seen during life. Dittel and Nitze, in 1876–1879, fixed an electric glow-lamp to the beak of an instrument, and thus carried the source of light into the bladder itself, and so obtained a great intensity of illumination. Nitze, by suitable optical contrivances, brought into view nearly all the surface of the bladder. Previously, when light reflected from without was employed by Grünfeld and others, only a small part of the wall of the bladder—that in immediate contact with the tube—could be seen.

Nitze has arranged the lamp differently in a second instrument, to obtain a view of that small portion of the bladder which the ordinary cystoscope may miss.

**APPARATUS.**—Leiter's forms of Nitze's instrument are the most useful for internal inspection. The apparatus is contained in two tubes, one of which slides in the other. The outer one has the form of an ordinary *coudé* catheter, the angle of junction of the beak with the stem being 130 degrees. The electric

current for supplying the lamp passes in through the tube itself, and back by an insulated wire carried in a groove inside the outer tube. The stem just above the angle of the bend is cut away at one side where a right-angled prism is cemented into it. This prism has its hypotenuse silvered, so that rays passing through the exposed face of the prism may be reflected into the inner tube. The inner tube is straight, about eight inches long, and slides in the outer tube. It carries four lenses: one, at the end farthest from the observer, is a hemispherical lens, the flat face of which, on pushing the tube in, goes quite close to that face of the prism which is at right angles to the axis of the instrument. Near to this hemispherical lens is another of very short focal length. The two lenses, receiving rays reflected at the hypotenuse of the prism, form a small image of objects situated opposite the prism, within a field of, say, 45 degrees diameter. This image is very near to the inner surface of the second lens. At the middle of the inner tube is a third lens of focal length somewhat less than one fourth of the length of the tube. This lens takes up the little image from the first and second lenses, and forms of it another image of nearly the same size not far from the end of the tube near the eye of the observer. Lastly, there is a short-focussed lens at the eyepiece for viewing the image formed by the third lens. For convenience we may call this inner tube and four lenses "the telescope."

Besides the telescope and the insulated wire, recent instruments contain two small tubes—one for conveying an inflowing stream of some sterilised or antiseptic fluid to distend the bladder, and keep its contents clear during inspection; the other for allowing the injected fluid to escape after circulating in the bladder, carrying with it any blood or muco-pus not evacuated before the cystoscope was introduced. By a Higginson's air-pump, acting on a reservoir, the stream is maintained constantly, and the bladder-walls are firmly distended—a very important condition for satisfactory inspection. This modification for irrigating during inspection was brought into notice by the present writer.

With the cystoscope above described the observer, on looking down the telescope, obtains a view of that portion of the illuminated surface which is situated within about 25 degrees of a perpendicular to the exposed face of the prism, provided the bladder be fairly distended with fluid. The whole surface of the bladder may be examined, in most cases, by pushing the prism farther into the bladder, or by drawing it back, or by turning the instrument on the axis of the stem, or by tilting the stem. But it is obvious that the parts of the surface near where the instrument enters, and in the region opposite to it, are difficult to bring into view in a satisfactory manner.

Nitze has accordingly some different forms of cystoscope adapted specially for those cases, the principal one of which has the window placed in the beak opposite the outer side of the bend, and the light at the farther or posterior end of the beak. By this arrangement the light is thrown directly upon the floor of the bladder, and so passes through the window to the telescope. This form of cystoscope illuminates the area between the uvula and the mouths of the ureters, and often reveals changes there not detected with the ordinary form.

In interpreting the significance of the images obtained, account must be taken of the varying distance and obliquity of different parts of the surface. This will evidently be a matter of difficulty, to be overcome by practice, especially by practice on a model of the bladder, in which the various natural landmarks are represented in bold exaggerated figures.

**APPLICATION.**—In the use of the cystoscope little can be seen if the cavity be not distended or if the contents of the bladder be turbid. When about six or eight ounces of fluid are injected, the surface of the bladder is fairly smooth, reddish or yellow, and racemose blood-vessels collecting into trunks towards the base of the viscus are very distinct. If the walls be tightly expanded the surface may seem glistening white, like mother-of-pearl. On the floor, the trigone is dull red, granular, and somewhat prominent. Near the ends of the ridge at the base of the trigone are placed in two little eminences the openings of the ureters. If the urine coming from a kidney be turbid with pus or other matter, a jet of this turbid fluid can often be seen issuing from the eminence and so discovering the orifice of the ureter. Similarly, if there be ulcers on the mucous membrane, or a papillomatous tumour, a jet of blood can be seen occasionally to dart through the clear fluid, which speedily renders it a luminous red fog, through which nothing definite can be described. In such cases the apparatus for maintaining a constant flow of ice-cold boracic solution through the bladder is of great service, as the opaque fluid is displaced through the outlet tube, and transparency is restored; and an artificially chilled solution represses hæmorrhage during inspection. If the contents of the bladder be allowed to escape during examination, the view becomes dark, or so indistinct as to prevent recognition of any known object, and then there is danger of the lamp growing hot and injuring the wall of the bladder in contact with it. Hence, whenever the view ceases to be luminous, the electric current should be at once cut off and the bladder refilled. The various changes effected in the appearance of the bladder by disease are described in the article **BLADDER, Diseases of**.  
BERKELEY HILL.

**CYSTS.—DEFINITION.**—The word 'cyst' (*κύστις*, the urinary bladder) is used in pathology without strict scientific accuracy for a closed cavity containing fluid or soft matter; which cavity will have, from the nature of its contents, a spherical or spheroidal shape. The objects thus defined differ much among themselves, and are associated together rather from convenience than on account of any real pathological similarity.

**CLASSIFICATION.**—Cysts may be classified according to their structure, as simple or compound; according to their contents, as serous, mucous, fatty, &c.; or according to their mode of origin. The latter, though not free from objection, is the basis of description which will be here adopted. Cysts may originate: (1) from *dilatation* of previously existing closed cavities; (2) from *retention* of the products of secretion of a secreting gland; (3) as a part of *new-growth*; (4) by a vice of *development*. These kinds are distinct structures with a wall lined by epithelium. But the name is often given, less accurately, to spaces containing fluid which result from softening of solid tissue, whether in a new-growth or in an inflammatory product. Cystic parasites have also been confounded with cysts properly so called.

1. **Cysts from Dilatation.**—Spaces, normal or newly formed, in connective tissue may, by irritation and consequent excessive exudation, be converted into cysts; or the same result may happen from the confluence of several such spaces. In proportion as the wall becomes smooth, and the shape uniform, they may be called cysts. Bursæ, whether normal or pathological, are cysts. Ganglion in the sheath of tendon is clearly a pathological cyst. To these and like structures the name *hygroma cysticum* has been given. They all contain clear serous fluid, and are lined by an endothelium. Encysted hydrocele may also be called a cyst. One class of ovarian cysts comes under this head, those, namely, which are due to simple dropsy of the Graafian vesicle. Tubo-ovarian dropsy has the same explanation; and cysts of the broad ligament are enlargements of normal structures which are left as relics of the development of the ovary. The thyroid gland seems, from its structure, containing as it does so many closed follicles, particularly disposed to this kind of cyst-formation, and this is doubtless the explanation of bronchocele. The cause of such an enlargement of closed follicles is very obscure, but appears to be immediately due to increased production of the material normally filling the space.

2. **Cysts from Retention.**—Cystic formations may result from the obstruction of the natural outlet of a secreting organ, and the consequent retention of secretion. It is necessary that the walls of the secreting cavity should admit of enlargement, and that

the tension should not become so great as to check secretion.

All secreting glands present instances of such cysts. The sebaceous glands of the skin are particularly liable to obstruction of their ducts, and in this way are formed sebaceous cysts, the contents of which are sometimes epithelium and the products of normal secretion, sometimes abnormal products. The same term has, with less propriety, been applied to the sebaceous productions called milium and comedo. See FOLLICLES, Diseases of.

The glands or mucous surfaces are liable to similar obstructions, and *mucous cysts* result, such as are sometimes seen in the mouth. Larger cysts in the mouth (*ranula*) result from the obstruction of the ducts of the salivary glands, or are perhaps connected with an abnormal production of gland-substance. The stomach very frequently, other parts of the intestinal canal more rarely, show similar cysts, which, when they project and become complicated in structure, are called polypi. They are occasionally seen on the larynx and trachea. In no part are mucous cysts more frequent than in the uterus, where, indeed, similar formations, the *ovula Nabothi*, must be regarded as normal. The varieties here met with have, as Virchow has pointed out, a close analogy with the various forms of retention-cysts in the skin.

In the mamma, cysts may result from the cutting off of portions of the gland-follicles, but the cysts contained in mammary tumours are not always formed in this way, some being part of new-growths. In the testicle obstruction and cutting off of seminiferous tubes may lead to small cysts, but these are more often connected with new-growths. The curious cysts known as *spermatocetes*, containing spermatozoa, appear to arise from a similar distension of detached portions of testicle-substance, which, by an error of development, have failed to become connected with the excretory ducts. The testicle is also liable to a general cystic degeneration, usually called *cysto-sarcoma*. Cysts of the kidney are of various kinds, but many, no doubt, both large and small, result from the dilatation of uriniferous tubules and capsules of glomeruli when their outlet is obstructed, as occurs in the cirrhotic form of Bright's disease. The origin of the very numerous microscopic cysts has been much disputed. The writer inclines to the belief that they arise from moniliform contraction of the uriniferous tubes, especially such as contain the hyaline cylinders, known as fibrinous casts. Another form of cystic disease of the kidney is developmental. In this the whole of the organ is converted into a mass of cysts, and is usually much enlarged. This condition may be congenital, and the organ may be so large as to obstruct parturition. It is

attributed by Virchow to inflammation of the calyces during intra-uterine life.

To guard against a common error of language, it should be pointed out that the condition of the kidney which results from the obstruction of the ureter, or of the urinary passages lower down, though sometimes called cystic dilatation of the kidney, is not properly a case of cyst-formation, and is better called *hydronephrosis*.

3. **Cysts from New-Growth.**—In many forms of new-growth cysts are produced, but not always in the same way. Occasionally, as in myxoma and enchondroma, softening of portions of new-growth already formed produces spaces containing fluid, which are sometimes, though hardly correctly, called cysts. In many sarcomata, the production of new tissue goes hand in hand with that of cysts, and is sometimes effected, as in glandular organs, by the formation of new follicular structures without an outlet, sometimes by new-growth into the dilated cavities. Polypoid or pedunculated growths on a free surface may sometimes, by the fusion of their extremities, enclose spaces which become converted into cysts. We do not often find cysts forming by themselves a new-growth of so definite a character as to deserve a separate name. When they do so, they may be called *cystoma*.

#### 4. **Cystoma or Cystic Tumour.**—

Cysts are met with in the ovary which come under the definition just given, namely, so-called *compound multilocular cysts*, which constitute the well-known formidable cystic disease of the ovary, and sometimes produce tumours of immense size. In these the originally simple primary cyst appears to become complicated by the formation in its walls of secondary cysts, which may encroach upon or project into the primary. Again there may be papillary growths starting from the inner wall of the primary cyst, which either fill it up, or by fusion enclose spaces, which become secondary cysts. Very complicated structures thus result. The contents may vary in consistence and colour, from clear, pale, albuminous liquid to gelatinous matter, and may be stained through hæmorrhage, or purulent through inflammation. The origin of these structures, which have no precise parallel in other parts of the body, is extremely obscure. It is not even certain whether the primary cysts commence, as might seem *primâ facie* highly probable, in the Graafian follicles; but they are plainly due to an error of development, possibly beginning in early intra-uterine life, and are not set up by any external causes. The presence of a tubular gland-tissue, such as is found in the rudimentary, but not in the perfect ovary, confirms this view, by throwing cyst-formation back into an early stage in the development of the organ.

In another, but rarer, form of cystic

disease of the ovary, equally due to an error of development, and sometimes congenital, the whole organ is found converted into a mass of small cysts, with no striking inequality of size. This variety resembles one form of cystic disease in the testicle and kidney.

*Dermoid cysts* are those of which the wall is composed of skin or mucous membrane, and generally lined by a layer of flat cells resembling epidermis. The wall may be complicated with connective tissue, forming papillæ resembling those of true skin, and may contain hairs, sebaceous glands—either in connexion with them or unattached—and sudoriparous glands. The accumulation of fatty matter within the cysts is doubtless the result of the continuous activity of the sebaceous glands, the products of which cannot escape. Large masses of hair may also be found, from continuous growth, and there are often numerous detached epidermic scales. Distinctly formed mammary glands, with nipples, are sometimes found. Such a cyst has only the characters of a portion of skin, which might be imagined inverted and included by the growth of the surrounding parts in an early stage of development—an explanation which applies only to those situated in certain parts of the integument where a junction has normally taken place—*e.g.* along the lines of the branchial fissures in the neck, in the anterior middle line of the body, &c.

These simple dermoid cysts are sometimes complicated by containing teeth, it may be in very large numbers (*dentigerous cysts*); but since teeth may also be regarded as cutaneous products, the cyst may still have originated in the skin. This explanation no longer holds, however, when masses of bone are found, sometimes serving for the attachment of teeth, sometimes separate; as well as other tissues, *e.g.* nervous tissue and striated muscle. Cysts with this variety of contents have been called *proliferative*. Dermoid, dentigerous, and proliferative cysts appear to be always congenital structures, but may show further growth and development in after-life. At least two-thirds of the known cases have occurred in the ovaries. Next to these organs, the testicles are the most frequent seat, but these cysts have been also found in other parts of the body-cavity, in the mediastinum, lung, and even within the skull. The origin of these growths is extremely obscure; but it is desirable to reject entirely the hypothesis that a mixed tumour of this kind can be the remains of an undeveloped fetus included in the perfect individual; a hypothesis rendered improbable by the extreme irregularity of the tissues produced, the teeth, for instance, sometimes numbering one hundred or more. It would rather appear as if, in some instances, a portion of embryonic epithelial tissue had

become misplaced at an early period of development. In other cases, these cysts originate in obsolete canals, representing rudimentary structures; such as the central canal of the nervous system, the post-anal gut, the branchial clefts, the vitello-intestinal duct, and others.<sup>1</sup>

Several parasitic animals infesting the human body may appear in an encysted form, and may resemble in appearance true pathological cysts. The commonest—the larval form of *Tænia echinococcus*, or *hydatid cyst*—is known by its laminated wall, and by containing a fluid which is not albuminous, but holds in solution sodium chloride. *Cysticercus cellulosa* has a transparent wall and clear contents. The other encysted parasites are either very small, as *Trichina spiralis*, or unimportant.

**CONTENTS OF CYSTS.**—The serous cysts and hygromata contain an albuminous fluid like that of serous cavities, which may hold

enough fibrinogenous material to coagulate spontaneously. Leucocytes may also be present. If inflammation be set up, the proportion of albumin and of leucocytes becomes greatly increased. In the fluid of mucous cysts mucin is contained; in that of colloid cysts, little-known substances which are allied to gelatin. Sebaceous cysts contain neutral fats—sometimes hard, sometimes fluid, and cholesterin. Both mucous and sebaceous products may harden into concretions, and even become calcareous. In renal cysts urea has been found; in biliary cysts, bile-pigment; and in general the products of special secretion may be found in cases of retention, at least in early stages; but if retention last too long, special secretion may cease. Various exceptional contents have been already enumerated.

**TREATMENT.**—The treatment of cysts is referred to under the head of TUMOURS.

J. F. PAYNE.

## D

**DACTYLITIS** (δάκτυλος, a finger).—A term meaning inflammation of the finger. It is applied to syphilis and struma of the part, as in the terms *dactylitis syphilitica* and *dactylitis strumosa*.

**DANDRUFF**, or **Dandruff** (from Celtic *ton*, skin; and *drwg*, foul).—Scurf of the head (see **HAIR**, Diseases of). Dandruff is met with in pityriasis, chronic eczema, and psoriasis of the scalp.

**DANDY FEVER.**—A synonym for Dengue. See **DENGUE**.

**DARTRE** (Fr.).—This term is the French equivalent of the word *tetter*, and is applied to a variety of cutaneous diseases, without strict limitation.

**DAVOS-PLATZ**, in North Engadine, Switzerland, an Alpine valley at an altitude of 5,177 feet. Well sheltered; atmosphere still and cold. A winter health resort, particularly for cases of phthisis. See **CLIMATE**, Treatment of Disease by; and **PHTHISIS**.

**DAY-BLINDNESS.**—A disorder of vision, characterised by the patient being unable to see in the daylight; called also hemeralopia. See **HEMERALOPIA**; and **VISION**, Disorders of.

**DEAFNESS.**—Loss of the sense of hearing. See **EAR**, Diseases of; and **HEARING**, Disorders of.

**DEATH, Modes of.**—The proximate causes of death, whether resulting from natural decay, disease, or violence, may be reduced in ultimate analysis to two, namely, first, cessation of the circulation; and, second, cessation of respiration. On the continuance of these functions, and particularly of the former (if specialisation is possible where all are essential), life of the body as a whole, or of the individual tissues and organs, depends. These functions may cease from causes directly operating on their mechanism, but they may also be brought to a standstill by causes operating indirectly through the nerve-centres which regulate them. Hence it is usual, in accordance with Bichat's classification, to describe this as a third mode of death; so that we speak of death beginning at the *heart*, death beginning at the *lungs*, and death beginning at the *head*. This classification is convenient; for though death beginning at the head is, in reality, death from failure of the respiration or circulation, or of both, through paralysis of the vital nerve-centres, yet the affection of the nervous system is the primary fact, and the phenomena are sufficiently distinct and characteristic to require separate consideration. It must, however, always be borne in mind that, owing to the interdependence of all the vital functions, there is no such sharp line of demarcation, in reality, as we, for convenience sake, make in theory between the various modes of death. The failure of the circulation, however, seems the ultimate factor in the

<sup>1</sup> *Dermoid Tumours*. Bland Sutton, 1889.

extinction of life, as cases are sometimes met with (of cerebral origin—tumour, &c.) in which respiration ceases and the pulse continues for some comparatively prolonged time, and, indeed, if artificial respiration be resorted to, the heart will continue beating for a long period (an hour or more), the reverse condition not being observed. The suddenness of the death is also in direct ratio with the rapidity of interference with the heart's action. In pure asphyxia it is comparatively slow as compared with that from heart disease or stoppage, and, indeed, ends fatally only by producing that result.

**I. Death from failure of the Circulation.**—This may be (1) sudden, as in *syncope* and *shock*; or (2) gradual, as in *asthenia*.

(1) *Sudden failure of the Circulation.*—As the circulation of the blood depends on the difference in the pressure in the arteries and veins, the circulation will be brought to a standstill by any cause which annihilates, or very greatly lowers, this differential pressure. The cause may be in the heart, or in the vessels, or in both.

(a) *In the heart.* As the action of the heart is the chief factor in the maintenance of arterial tension, any organic or structural disease of the heart, rendering it incapable of propelling its contents into the arterial system, will naturally result in cessation of the circulation, and death. Under this general head are to be classed all diseases of the heart and its annexes.

But, apart from structural disease, the heart may suddenly be made to cease through nervous influence. The heart may be inhibited, or be made to cease finally and for ever, either by central causes, such as violent emotion, or a blow on the head, and probably in this way in the sudden death often observed in cases of cerebral abscess and tumour; or by reflex inhibition, as in the case of a violent blow on the epigastrium, or sudden irritation of the sensory nerves of the stomach, as in corrosive poisoning, and even in the ingestion of a large draught of cold water when the system is overheated.

Death from sudden cessation of the heart's action is death from *syncope*. Momentary cessation of the heart's action is transient *syncope* or fainting. There is sudden loss of consciousness, due mainly to the cessation of pressure in, and anæmia of, the cerebral centres.

(b) *In the vessels.* Rapid fall of the blood-pressure, and cessation of the circulation, will naturally be brought about by rupture of the vessels, either from injury or disease, causing death by hæmorrhage; or by occlusion of the pulmonary artery, which seems a commoner cause of sudden death than is usually believed. But, besides actual rupture of the vessels, the vascular area may

in certain conditions become so enlarged or dilated, that we may practically have death from hæmorrhage without any loss of blood externally. This is what we observe in death from *shock* or *collapse*. In certain conditions, such as that resulting from blows on the abdomen, the vascular area of the abdomen and viscera may become so dilated as practically to retain almost the entire volume of blood in the body. Hence, even though the heart may be acting, yet the circulation throughout the body generally, and especially in the extremities and superficially, is practically *nil*. The individual may, however, retain his consciousness, and thus he differs from a patient in a state of *syncope*. But very frequently, in cases of blows on the abdomen, there is not merely reflex dilatation of the abdominal vessels, but also reflex inhibition, for a time at least, of the heart, so that we have *syncope* and *shock* co-existing. The symptoms of *syncope* may pass off, leaving those of *shock* still remaining. *Shock*, like *syncope*, may be either transient or fatal.

(2) *Gradual failure of the Circulation.*—This constitutes death from *asthenia*. This is the natural termination of life, and it is also the mode of death after wasting and exhausting diseases, cold, starvation, &c. The vital powers fade gradually, while consciousness may be retained up to the last moment.

**II. Death from failure of the Respiration.**—The various ways in which the function of respiration may be interrupted, and the phenomena consequent thereon, have been described under the head of *ASPHYXIA*, to which article reference may be made.

**III. Death from paralysis of the vital nerve-centres—Coma.**—As already remarked, death beginning at the head ends by paralyzing respiration and circulation. The nerve-centres situated above the medulla and pons are not essential to life, except in so far as animal life is concerned, and the possibility of adaptation to surroundings. Diseases of the brain, however, are liable to prove fatal by indirect action on the medulla and pons through pressure, extension of inflammation, and the like. Certain poisons also, whether introduced from without—such as opium and narcotics generally—or arising within, owing to the non-elimination of waste products, as in uræmia, affect the nerve-centres, both cerebral and spinal, and not only produce unconsciousness or coma, but also paralyse the respiratory and cardiac centres.

In death arising in this manner, the individual lies unconscious, reflex action becomes abolished, and the breathing becomes stertorous and ultimately ceases, death occurring quietly or in convulsions. In death from coma, in addition to the usual phenomena of asphyxia, there is, as a rule, more or less

marked congestion of the cerebral and spinal centres.

*Management of the Dying.*—In whichever of the three ways death may commence, the management of the dying calls for careful attention on the part of the medical attendant; and requires to be specially discussed. See EUTHANASIA. D. FERRIER.

**DEATH, Signs of.**—It is not always easy to determine when the spark of life has become finally extinguished. From the fear of being buried alive, which prevails more abroad than in this country, some infallible criterion of death, capable of being applied by unskilled persons, has been considered a desideratum, and valuable prizes have been offered for such a discovery. The conditions most resembling actual death are syncope, asphyxia, and trance, particularly the last. We cannot, however, say that any infallible criterion applicable by the vulgar has been discovered, and we do not rely exclusively on any one sign, but combine several.

The most reliable sign of death is proof of *cessation of the heart's action*. This, however, is not to be inferred from mere pulselessness, for the heart may still be beating, and resuscitation may be possible, when no pulse is to be felt in the arteries by ordinary manipulation. The use of the stethoscope is necessary, implying, of course, technical skill. Though, according to Rayer, the heart cannot cease to beat for more than seven seconds without death, yet, considering the very slow and feeble action of the heart (8 to 10 beats per minute) in hibernating animals, which normally have a pulse of 80 to 90 per minute, it is well to regard a similar condition as possible in man, and to spend in doubtful cases some minutes, up to half an hour, in auscultation. The so-called cases of life continuing notwithstanding cessation of the circulation, as that of Colonel Townsend, or of the Indian Fakirs, are to be set down as altogether apocryphal, and not scientifically investigated.

To enable unskilled persons to determine whether the circulation continues or not, Magnus recommends the application of a tight ligature on a finger or toe. If the circulation has quite ceased, no change in colour is produced; but if circulation continues, however feebly, the extremity, in course of a longer or shorter period, assumes a livid tint from strangulation of the venous flow, while a ring of arterial anæmia is observable at the point ligatured.

Cessation of the heart's action, if absolutely established, renders other indications unnecessary. As accessories they are useful, but the following signs are none of them individually conclusive, taken alone. The first is *cessation of respiration*. Respiration may not be very obvious, and yet it may be going on. The popular methods, of

holding a cold mirror before the mouth and nostrils, and looking for indications of moisture; placing a flock of cotton wool on the lips to ascertain whether air-currents exist; and placing a cup of water on the chest, and observing whether the reflection on its surface moves or remains still, are all well adapted for the purpose in view.

With the cessation of the circulation and vital turgor, the skin becomes ashy pale, there is an absence of the pink colour in transparent parts when examined under the influence of a strong light (Richardson), and the tissues lose their elasticity. The eyeball becomes less tense, and the cornea becomes opaque. The pupils cease to react to light; and there is no vital reaction on the application of irritants to the skin. Though the body is dead as a whole, certain parts may continue to retain their independent vitality after somatic death. This is seen in the muscles, which may retain their electrical contractility from two to three hours after death. The existence of electrical contractility of the muscles in a body supposed to be dead indicates life, or death within the previous two or three hours, according to M. Rosenthal.

The subsequent changes which occur in the dead body not only indicate the fact of death, but aid in fixing the probable period at which death occurred. These are the following:—

(1) *The cooling of the body.*—The body after death, except under certain special circumstances, as in fatal cases of cholera and yellow fever, ceases to be a source of heat-production, and therefore is to be looked upon as an inert mass possessed of a higher temperature than the average medium, which parts with its heat according to certain physical laws. The superficial coldness of collapse, which is due to cessation of the peripheral circulation, must not be mistaken for the cadaveric coldness, for there is still an amount of internal heat which has to be parted with; and the body, cold to the touch before death, may after death rise in temperature, as the internal heat radiates. It is impossible to describe here in detail all the circumstances which modify the rate of cooling of the body; but it may be said in general that all circumstances which favour radiation, convection, and conduction of heat in inorganic bodies are equally applicable here, while the opposite conditions retard. Therefore a thick coating of adipose tissue, clothing, &c., retard cooling. The observations of Goodhart (*Guy's Hosp. Rep.* 1870) and Burman (*Edin. Med. Journ.* 1880) show that under ordinary circumstances we may assume the temperature in the axilla at the time of death to be the normal 98·4 F. Cooling occurs at the rate of 1·6° F. per hour. The rate of cooling is greater (2–4°) during the first few hours. Taking the difference

between the axillary temperature in any given case and the normal, and dividing it by 1·6, would give the number of hours that have elapsed since death.

(2) *Hypostasis*.—After death the blood gravitates to the most dependent parts, both externally and internally, giving rise to livid discolorations, termed *hypostases*. These are liable to be confounded with ecchymoses or extravasations externally, and with the results of congestion and inflammation in the internal viscera. They differ from ecchymoses in the fact that the blood is not extravasated into the tissues, but still contained in the vessels, as may be shown by an incision into the skin. So long as the blood remains fluid, these discolorations may be caused to disappear if the position of the body be reversed; they will again form in the parts which are now the most dependent. They usually occur in from eight to ten hours after death.

(3) *Rigor mortis*.—After death the muscles become stiff, giving rise to *rigor mortis* or cadaveric rigidity. It is due to coagulation of the muscle-plasma. This rigidity attacks the muscles usually in a certain definite order, beginning in the muscles of the neck and face, and gradually extending from above downwards. It gives way to putrefaction and the attendant solution of the coagulated plasma, in the same order; so that, while the upper parts of the body may be flaccid, the legs may be found rigid. It can only be overcome by tearing the tissues, and if overcome it does not return. In this it differs from cataleptic rigidity. A certain amount of mobility is still observable at the joints. In this it is unlike the stiffness of freezing, in which all the parts are equally rigid, and crackle if bent.

The period of the occurrence of rigidity, and the length of its endurance, are extremely variable, so that no definite practical rules can be laid down. It may be said generally, however, that the greater the store of muscular energy at the time of death, the longer it is before rigidity sets in, and the longer it lasts. On the contrary, the greater the exhaustion, the sooner rigidity sets in, and the sooner it disappears. Hence rigidity is longer in appearing in subjects dying suddenly in full muscular vigour, than in those dying from exhaustion. As a rule, a period of relaxation intervenes between death and the occurrence of rigidity, but in certain cases the last muscular contraction seems to pass directly into the rigidity of death. This is seen more particularly in death during great nervous excitement, as in soldiers on the field of battle, or in suicides. The same is said to occur also in death from strychnine-poisoning and in death by lightning, in which the tetanic spasm passes into rigidity.

Rigidity may therefore occur immediately on death or within a few hours. It has never

been observed to be delayed beyond a day after death. It may last from so short a time as scarcely to be perceptible, up to a week or more.

(4) *Putrefaction*.—After death the tissues undergo changes in colour, consistence, &c., by which they are ultimately resolved into their simple elements, included under the general term *putrefaction*. Putrefaction, however, may occur locally during life, and general septic changes may occur to some extent before death. The term, however, is not generally applied until the changes are clearly perceptible in alteration of colour, consistence, and smell. The first external sign is a greenish discoloration of the abdomen. Internally the mucous membrane of the larynx and trachea is the first to exhibit change in colour and consistence. The less compact tissues putrefy first, the fibrous tissues resist longer, and—in the female body—the compact tissue of the uterus resists longest of all. In process of time, however, the soft tissues become entirely disintegrated, and the skeleton is exposed and gradually falls to pieces.

The rate of putrefaction is very variable, depending partly on the state of the body itself, but mainly on external conditions as to temperature, moisture, and exposure. A combination of high temperature, moisture, and free exposure supplies the most favourable conditions for rapid putrefaction. A high temperature alone, without moisture, tends to dry the tissues, and thus to produce mummification, instead of colligative putrefaction. Moisture alone, as when a body lies in water or moist earth, tends to produce a saponification of the tissues, more particularly the fatty, with the formation of a substance termed adipocere (*see* ADIPOCERE). The course of putrefaction can be stopped by antiseptics, as in embalming, and in certain cases of poisoning, as with arsenic, as also by freezing. Putrefaction is more rapid in air than in water, and least rapid in earth. Under ordinary circumstances and average temperatures, signs of putrefaction are clearly visible on the third day after death, commencing with the green hue of the abdomen. Many months elapse before the soft tissues become entirely disintegrated. The uterus has been found fit for judicial examination as long as nine months after death, where no antiseptics had been employed. When such has been the case, however, there is practically no limit to the period of preservation—witness the Egyptian mummies, embalmed thousands of years ago.

There is still much to be learnt respecting putrefaction, and it is unsafe to lay down dogmatic rules as to how far putrefactive changes shall have advanced at a given time, for even under apparently similar conditions the most extraordinary divergences have been recorded.

D. FERRIER.

**DEBILITY** (*debilis*, feeble).—SYNON.: Feebleness; Weakness; Asthenia; Fr. *Faiblesse*; Ger. *Schwäche*.

**DEFINITION.**—The body or any of its organs is said to be in a state of debility when its vital functions are discharged with less than the normal vigour, and when the amount of activity which it displays and of work which it can accomplish is diminished. The term 'debility' is also employed in a somewhat different sense in the case of constitutional weakness of an organ, to convey the notion of vulnerability or predisposition to disease. In this acceptation, 'pulmonary debility,' for example, signifies a peculiarly delicate 'build' of the lungs, which renders them more than ordinarily liable to succumb to the causes of disease.

**ÆTIOLOGY.**—Debility is frequently constitutional and inherited; but it is more often developed after birth. It is most commonly due to impaired nutrition, whether this be prolonged and moderate, as in defective hygiene or chronic illness, or, on the other hand, rapid and extreme, as in acute disease. Such failure of nutrition, when local, may arise from the blood being deficient in quality or quantity, or possibly from some interference with the nervous supply. Another frequent cause of debility is the abuse of the affected organ. Over-use of a part leads to fatigue, and if frequently repeated to exhaustion, the chief feature of which is extreme debility, as in cases of sustained and mental exertion, or of repeated strain of the heart. On the contrary, an organ may become feeble from want of exercise. Paralysed muscles furnish the best examples of this condition, but the same may be seen in all organs after unnatural rest. Debility is more liable to occur at certain ages, such as the periods of active growth and development, and in connexion with the menopause; and it is one of the manifestations of advanced life.

**SYMPTOMS.**—The natural ability of the organs to perform their functions varies extremely with sex, age, previous exercise, and many other circumstances. Debility, or the loss of this functional power, is therefore frequently ill-defined; and, when unquestionably present, may vary greatly in different cases, from a condition in which fatigue comes on only somewhat earlier than usual, as in muscular debility, to a state in which the slightest exertion may exhaust the whole of the vital energy, and the functional life of the part may cease—as is seen in the cardiac asthenia of acute fevers, and scarcely less markedly in certain chronic diseases, such as idiopathic anæmia and Addison's disease.

Debility may be *general*, affecting the whole body; or *local*, individual organs only being involved. Speaking broadly, the symptoms of debility of an organ may be said to be chiefly two. These are, first, increased irritability, or an unnatural readiness of the part

to respond to stimulation; and, secondly, a tendency to untimely exhaustion. The phenomena of irritability and exhaustion naturally vary with the organ involved. The symptoms of muscular asthenia are few and simple; those of digestive feebleness are more complex; and in debility of the nervous system the whole of the mental processes, as well as the functions of organic life, may be involved. For a specific account of the phenomena of each of these cases, and of debility of other parts, the reader is referred to the articles upon diseases of the several organs.

**DIAGNOSIS.**—Debility pure and simple is, as a rule, easily distinguished from *disease* by the absence of all evidence of organic alteration, and especially of physical signs of anatomical change. It is more difficult to separate debility from *disorder* or *derangement*, but careful observation will generally determine in the case of pure debility that the functions are normally discharged as long as the demands made upon them are not excessive.

**PROGNOSIS.**—Debility due to acute disease may, in the absence of complications, be expected to disappear during convalescence. If the cause have been more chronic, and be less easily removed, recovery will certainly be more slow and less satisfactory. The prognosis of inherited constitutional debility, as regards its disappearance, is nearly always unfavourable.

**TREATMENT.**—Debility must be treated according to its cause. If nutrition have failed, it must be restored as far as possible; and until this can be done, stimulants and suitable tonics are indicated—especially in the case of acute disease. Rest is of the first importance in most instances; and frequently it is alone sufficient to restore the vital force. In a few cases, however, the opposite line of treatment must be followed, as in muscular debility from want of exercise or in some forms of paralysis. Where the vital activity is low from constitutional defect, age, or sex, the condition may not be remediable; and the treatment of such cases is chiefly prophylactic. The principal indication then is to secure the subject of debility against exposure to damaging influences.

J. MITCHELL BRUCE.

**DECLINE** (*declino*, I decline).—A popular name for any wasting disease; especially associated with pulmonary consumption. The word is also applied to the period in the course of a disease when the symptoms are abating; and likewise to the time of life when the physical and mental powers are failing.

**DECUBITUS** (*de*, down; and *cumbo*, I lie).—The lying posture. *Decubitus* is also the German name for bed-sore. See POSTURE.

**DEFÆCATION, Disorders of.**—On the descent of the fæces, which accumulate in the sigmoid flexure of the colon, into the rectum, the associated movements necessary for their expulsion are excited. These movements are chiefly involuntary, though influenced and controlled by the will.

**ÆTIOLOGY.**—Some of the more important difficulties interfering with the actions of defæcation arise from disorders in the *nervous system*, by which the movements are excited and directed. In injuries and diseases of the brain or cord, the controlling action of volition, especially its influence over the sphincters, is annihilated, whilst the excito-motor movements depending on the spinal cord continue. In these cases the actions of defæcation take place only when the need of expulsion arises, without any power of the patient to induce or restrain them. In injuries destroying the lower part of the spinal cord, the fæces escape involuntarily in varying quantities and at all times.

Serious troubles in defæcation may also arise from excessive as well as from weakened action of the *muscles* concerned in this function. The sphincter may be irritable or subject to spasm, and resist too forcibly the expulsive actions of defæcation (*see ANUS, Diseases of*). Or the muscular fibres of the rectum may lose their tone, and be defective in power and incapable of properly extruding the fæces. Patients thus situated are often obliged, when at stool, to use the finger to dislodge masses retained in the weakened bowel. An atonic condition of the rectum usually arises from over-distension. It may be produced as the result of accumulations, or by too free and frequent use of enemata, the quantity injected being so large as to dilate the bowel and impair the power of its muscular coat. This atonic state of the bowel is itself apt to give rise to fæcal accumulation. The present article will deal with deficient defæcation associated with such accumulation. The opposite condition is described in the next article. *See DEFÆCATION, INVOLUNTARY.*

**Fæcal Accumulation.**—**PATHOLOGY.**—Cases of this kind are not uncommon, yet the nature of the affection is liable to be overlooked. The rectum may become gradually dilated and blocked up by a collection of hard dry fæces, which the patient has not the power to expel, being unable from loss of tone in the distended bowel to overcome the resistance of the sphincter to the passage of so great a body. Some indurated lumps from the sacs of the colon, on reaching the rectum, perhaps coalesce so as to form a large mass; or a quantity accumulated in the sigmoid flexure, on descending into the lower bowel, becomes impacted there. In several instances a foreign body has been found in the centre of the mass. The persons most subject to these troubles are those enfeebled by age or

disease, especially women. They may also occur in infants who have been operated on for imperforate anus, when the artificial aperture contracts, or is left too small for the free passage of the fæces. In these cases the distension of the bowel is sometimes excessive, and its expulsive functions are seriously impaired and weakened.

**SYMPTOMS.**—Such a collection gives rise to considerable distress, producing constipation, a sensation of weight and fulness in the rectum, tenesmus, and forcing pains which women liken to those of labour. In cases of some duration, where the hardened fæces do not quite obstruct the passage, they excite irritation and a mucous discharge, which, mixing with recent fæculent matter passing over the lump, causes the case to be mistaken for diarrhœa.

**TREATMENT.**—Injections have no effect in softening the indurated mass: they act only on the surface, and return immediately, there being no room for their lodgment in the bowel. The practitioner on passing his finger finds the rectum blocked up with a large lump, which feels almost as hard as a stone. In such cases the only mode of giving relief is by mechanical interference. The mass requires to be broken up and scooped out—an operation which may require the use of a cocaine suppository, or the administration of an anæsthetic. After the breaking up and extraction of the larger portions, injections of soap and water will be sufficient for the removal of the remainder.

T. B. CURLING. FREDERICK TREVES.

**DEFÆCATION, Involuntary.**—Under normal circumstances the fæces are retained within the rectum by the closure of the sphincter ani. When defæcation takes place, the sphincter is relaxed; and there is increased peristalsis of the lower bowel and pressure on the intestines, from forcible contraction of the expiratory muscles with the glottis closed. The nerve-governance of these three associated phases of the act is different in each case. The contraction of the abdominal muscles is mainly due to an effort of the will. The activity of the colon and rectum is reflex, from irritation of the accumulated fæces. Lastly, the relaxation of the sphincter depends on the inhibition of the tonic centre in the lumbar enlargement of the spinal cord; such inhibition being brought about either in a reflex manner from the rectum or directly from the cerebral centres. It is obvious that the relaxation of the sphincter is the most important stage in the act, for until that takes place no discharge can occur. Up to a certain point the increased peristalsis and pressure on the bowels may be resisted by a voluntarily increased contraction of the sphincter, but at a certain stage the inhibiting influence is exerted, and relaxation results. Since this influence is only in part under the

control of the will, it would follow that the chief causes of the involuntary discharge of the fæces are to be found in those conditions which diminish the normal tonicity of the sphincter. At the same time, any circumstances which may increase the pressure of the abdominal muscles, or the activity of the bowels, much beyond what ordinarily occurs, may lead to an involuntary discharge.

**ÆTIOLGY.**—The causes of involuntary discharge of fæces may be discussed in the following order:—

1. *Violent contraction of the expiratory muscles.* This cause of involuntary defæcation is such as may be induced by strychnine-poisoning. It is of rare occurrence in tetanus.

2. *Increased peristaltic action of the intestines.* Increased peristalsis is chiefly dependent on causes of irritation situate in the bowels themselves, such as inflammation or ulceration of the walls; irritating contents; or worms. Extreme fluidity of the fæces is frequently sufficient to induce their partially involuntary discharge, and is noticeable in the looseness sometimes associated with accumulation of fæces in the lower bowel, and the diarrhœa of infants and children.

3. *Abnormal relaxation of the sphincter ani.* As already said, the previously mentioned causes are powerless to produce the discharge of the fæces until the sphincter yields; and how far the sphincter may relax as a result of their efforts, or independent of them, is not easy to determine. The nervous influence emanating from the lumbar centre which normally determines the tonic state of the sphincter may be inhibited by various causes:—

a. *Reflexly.* Reflex inhibition of the sphincter ani may arise from such causes as worms, fissure of the anus, or calculus vesicæ. It is quite true that the immediate result of irritating the sphincter is to determine in a reflex manner an increase of its contraction, as may be experienced in introducing instruments into the rectum; but it would also seem that at a certain point the irritation may lead to an arrest of the tonic influence, and so allow the sphincter to yield, and this is particularly the case with affections of the bladder.

b. *From cerebral disease.* That certain emotional conditions may lead to involuntary defæcation is well known, and that a similar result follows some diseases of the cerebral centres is not an uncommon event. The paralysis of the sphincter which occurs under these circumstances is brought about by inhibiting the normal tonic stimulus discharged from the lumbar centres. Involuntary evacuations frequently occur in epileptic fits; in states of profound coma induced by apoplexy, or by opium- and other forms of poisoning; and in death by hanging or suffocation. Its occurrence can scarcely be regarded as in-

dicating lesion of any one part of the cerebral centres, but rather as a result of general brain-states. It constitutes a troublesome complication in such chronic forms of brain-disease as general softening, paralysis of the insane, &c.

γ. *From disease of the spinal cord.* The relaxation of the sphincter may of course be produced by disease or injury of the lumbar enlargement of the spinal cord. Inasmuch as it is from the cells of this region that the normal tonic influence is understood to issue, destruction of the nervous tissues can readily be understood to prevent the origination of such stimulus to contraction. Certain lesions also above the lumbar enlargement may lead to involuntary passing of fæces by interfering with the cerebral control of the sphincter. It is noticeable that diffused changes in the substance of the cord, such as acute or chronic myelitis, intra-spinal hæmorrhage, and compression, are far more frequently accompanied by paralysis of the sphincter than are the scleroses of a 'systemic' or 'columnar' character—e.g. anterior polio-myelitis, lateral sclerosis, progressive muscular atrophy, or inflammatory affections of the spinal meninges.

δ. *From local lesion.* The control over the sphincter may be lost from injury to the muscle itself. This occurs in laceration of the perinæum, involving the lower end of the bowel; sometimes it is the result of surgical operation, such as deep division of the sphincter in connexion with fistula.

**TREATMENT.**—No direct treatment of incontinence of fæces is of avail, beyond removal, when possible, of the cause. This may be done with success when the involuntary discharge depends on the presence of some local irritant, such as worms or fæcal impaction. As a distressing complication of certain diseases of the nerve-centres, which are too frequently incurable, little remains to be done for it beyond adopting such measures as will permit of the escape of the discharge with every regard to cleanliness.

W. H. ALLCHIN.

**DEFERVESCO** (*de*, down; and *fervesco*, I grow hot).—The decline of fever, characterised by a fall of temperature and of pulse, and by other phenomena. See FEVER.

**DEFORMITIES.**—See MALFORMATIONS.

**DEFORMITIES OF THE CHEST.**  
See CHEST, Deformities of.

**DEGENERATION** (*degener.*, unlike one's race; out of kind).—SYNON.: Fr. *Dégénérescence*; Ger. *Entartung*.

**DEFINITION.**—The word 'degeneration,' meaning etymologically change or deterioration of kind, is used in pathology for any process by which a tissue or substance becomes

replaced by some other, regarded as less highly organised, less complex in composition, of inferior physiological rank, or less suited for the performance of its original functions. While some change for the worse is thus the essence of degeneration, there is great variety in the mode by which this change is effected. It may be by direct chemical *metamorphosis*, as of albuminous into fatty material; by *infiltration* of the tissues with some new material, as in albuminoid degeneration; by *deposition* of some foreign substance, such as lime salts, in the tissues; or even by *substitution* of a newly formed tissue, inferior to the original in organisation or in functional efficiency, as in what is called fibroid degeneration. The degenerative process may be in certain circumstances any one of the above processes. Degeneration is very closely connected with atrophy, since on the one hand it is often caused by imperfect nourishment, and on the other hand may be a stage in progressive wasting; so that it may be difficult to draw the line between the two.

**SUMMARY.**—The following kinds of degeneration may be recognised: *albuminoid, fatty, mucoid or colloid, parenchymatous, calcareous, pigmentary, and fibroid*, with possibly one or two minor varieties of less moment. The two first-mentioned are described elsewhere. See ALBUMINOID DISEASE; and FATTY DEGENERATION.

**1. Mucoid: Colloid.**—This kind of degeneration is in one sense a physiological process, since it is probably by a partial transformation of the protoplasm of epithelial cells into mucin that the secretion of mucus is effected.

Mucoid degeneration and colloid degeneration are sometimes distinguished. We are unable to recognise any difference except in situation, and this distinction is better expressed in other words. The process consists in the transformation of portions (usually albuminous) of the tissues into a semi-transparent homogeneous material, varying in consistency from fluid to a gelatinous solid, and consisting of altered albuminates with variable proportions of mucin, a substance allied to albumin, but differing in its entire insolubility in acetic acid and its solubility in alkalis. All masses of gelatinous appearance have not this composition, since the colloid material which fills some ovarian cysts, that of renal cysts, and probably that found in some other cases, is said to contain no mucin. The thyroid gland when enlarged and cystic, as in one form of bronchocele, is a striking instance of colloid degeneration. The enlarged cystic vesicles become filled with colloid material, which plainly results from a transformation of the epithelial elements, and possibly also of some albuminous exudation in the original vesicles. Small masses of colloid material first appear in the cells,

which become confluent into homogeneous masses. In colloid cancer a similar process appears to take place, but it is rather synchronous with, than subsequent to, the growth of the tumour. The alveolar spaces, which in other forms of cancer are filled with cells, here contain colloid material resulting from the metamorphosis of cells; and every transitional stage, from the epithelioid cancer-cell to a homogeneous translucent mass, may often be met with. The stroma is unaffected, and remains fibrous. The gelatinous material of colloid cancer is stated to contain more mucin than that of the enlarged thyroid. It is instructive to notice that this form of cancer usually occurs or commences in parts where epithelium is present, which undergoes the mucous transformation and secretes mucus, as in the stomach and intestines. When colloid or mucoid transformation affects tissues of the connective tissue group, it is the intercellular substance which appears to be chiefly affected. This change is seen in the mucoid softening of cartilage which sometimes occurs in old age, where the chondrin undergoes chemical change, and the intercellular substance softens into a diffuent or liquid substance containing mucin. The so-called mucous tissue, which forms the umbilical cord and the vitreous body of the eye, as well as certain fetal structures, consists essentially of a reticulated connective tissue with mucous intercellular substance, and the same tissue forms the new-growth called myxoma, which may therefore be regarded as formed by mucous transformation of connective tissue. Its cells are quite unaffected by this change, being either fixed stellate connective-tissue cells, or migratory lymphoid corpuscles. This view explains how portions of other tumours, as sarcoma, enchondroma, and lipoma, are often found to have undergone myxomatous degeneration. All these mucoid or colloid substances contain mucin with albuminates.

**2. Parenchymatous.**—**SYNON.**: Granular Degeneration; Cloudy Swelling.—This is a peculiar change met with in some epithelial structures, especially liver and kidney cells, and muscular tissue, occurring only in the course of some infective febrile diseases, especially typhus, enteric fever, scarlatina, diphtheria, and pyæmia. The histological elements are found after death to have lost their transparency, and to be filled with minute granules, so that the general appearance is not unlike that of fatty degeneration. The naked-eye appearance of the organs is also not dissimilar: they are pale, dull, and opaque-looking. This change has been thought to be the precursor of fatty change; but, whether this be so or not, it is at once distinguished by the solubility of the granules in acetic acid, and their insolubility in ether. The cause of this degeneration has been asserted to be simply high temperature; still

it is not found in all febrile diseases. It has also been regarded as a *post-mortem* change, which is possible, but still this implies some abnormality in the tissues during life.

Another change which occurs in febrile diseases is *waxy* or *vitreous* degeneration of the voluntary muscles. They are found after death with little or no striation, and the myosin irregularly coagulated in lumps. That this is a change occurring after death there can be no doubt; while it is equally clear that this abnormal coagulation shows some abnormality of composition to have existed during life.

3. **Calcareous.**—Calcareous degeneration consists in the deposition of calcareous particles in the elements of a tissue, or in some inflammatory products previously formed. It is more appropriately called *calcareous infiltration* or *deposit*. When the normal tissues are thus infiltrated, there is not of necessity any other alteration in the tissues themselves, though the process generally indicates retardation of the circulation or arrest of tissue-metamorphosis. In the case of inflammatory products, new-growths, and parasites, as well as in other cases, the calcareous deposit follows on partial necrosis or local death. It is, therefore, if not a degeneration, the consequence or accompaniment of degeneration. See DEPOSITS.

4. **Pigmentary.**—Pigmentary degeneration is a name which has been given to the changes produced in a tissue or organ by the deposition or formation of pigment. It is very doubtful whether this should always be described as a degeneration, since the change does not necessarily diminish the vital activity of the part, lower its physiological rank, or involve a simpler chemical or anatomical composition. Pigmented tumours do not show less vitality than others, nor is excessive pigmentation of the skin, or any organ where pigment normally occurs, necessarily an accompaniment of degeneration. The colouring matter in this case is the normal pigment called melanin. An entirely different kind of pigmentary change is that which depends upon the extravasation and breaking up of red blood-discs, the hæmoglobin of which becomes converted into a simpler form of pigment, such as hæmatoidin or some other. The pigmentation of the spleen and liver from intermittent fever, and that which is the consequence of chronic venous congestion, are of this kind; and are often the accompaniment of fibroid induration, thus forming part of a degenerative process. Pigmentation in general must not, therefore, be identified with pigmentary degeneration.

5. **Fibroid.**—Fibroid degeneration is the name given to a process frequently met with, in which the original tissue becomes replaced by a form of connective tissue. It is also called *fibroid substitution* or *fibroid change*. In the early stages of this process

we find the tissues penetrated with numerous cells of the lymphoid type, which become slowly organised into connective tissue—at first of the cytogenous form, and rarely very vascular. The infiltrated lymphoid cells are probably chiefly derived from the blood-vessels; but some may be, as theory teaches, the descendants of tissue-cells. The process is an essentially chronic interstitial inflammation (see INFLAMMATION). Since the final result of the process is that tissue of less physiological value is substituted for the original, the process may be described in general terms as a degeneration, though it is not an actual metamorphosis of tissue. It finally leads to induration, contraction, and partial atrophy. J. F. PAYNE.

**DEGLUTITION, Disorders of.**—**SYNON.:** Disturbances of Swallowing; Fr. *Troubles de la Déglutition*; Ger. *Störungen des Schluckens*.

Before describing the disorders of deglutition or swallowing, it is necessary to state briefly in what this physiological act consists, and how the process is performed.

**PHYSIOLOGY OF DEGLUTITION.**—The act of deglutition is commonly divided into three stages. The first is a voluntary effort, accomplished by means of the tongue and the muscles of the cheeks and mouth, as far back as the anterior arch of the fauces. The second stage is an involuntary act, though certain voluntary muscles are engaged in effecting it. It is accomplished by the action of those muscles the duty of which is to retract the tongue, to raise the larynx and close the glottis, to lift the soft palate, to contract the fauces and bring the tonsils in contact with the bolus of food, to close the posterior nares, and to raise and contract the pharynx. Then the food passes into the œsophagus or gullet, when the third stage is entered upon; and as the morsel passes into this tube, a progressive undulatory or peristaltic movement of the gullet is produced, by which the bolus is propelled into the stomach.

**DEFINITION.**—Any condition which interferes with the perfect integrity of this physiological process constitutes a disorder of deglutition. The general term which is applied to this condition is Dysphagia or *deglutitio impedita*, as it is sometimes termed. But, in actual fact, such conditions will be found to be dependent for the most part on some other morbid state of the structures immediately concerned in the act of swallowing, or of those in close proximity to them. Yet, although dysphagia must, as a rule, be regarded merely as a symptom of some more or less serious disorder, still it may be convenient and useful to examine the subject somewhat more in detail than can be done in the consideration of those affections in which it frequently plays so prominent a part.

**ÆTIOLOGY.**—All affections of the throat modify in some way the power of swallowing, and render the act of deglutition painful and difficult. Thus:—1. We meet with it as one of the symptoms in acute catarrh of the pharynx, in tonsillitis, and in ulceration of the throat. 2. Similarly, diseases of the larynx may give rise to disorders of deglutition; such as laryngitis, inflammation of the perichondrium, and laryngeal polypi. 3. Specific diseases—for example, phthisis, syphilis, cancer, scarlatina, measles, and croup—are another fertile cause of difficulty of swallowing, owing to their affecting the throat in various ways. 4. So also are nervous affections—for instance, post-diphtheritic paralysis, hysterical disorders, general paralysis of the insane, progressive muscular atrophy, and glosso-pharyngeal paralysis. 5. Affections of the salivary glands, such as parotitis, may interfere with deglutition. 6. Œsophageal disorders, whether functional, or causing organic obstruction, are important causes of dysphagia. 7. Difficulty of deglutition may result from pressure upon some part of the passage, as by an aneurysm of the thoracic aorta, a solid tumour, whether malignant or benign, or a retro-pharyngeal abscess. 8. It may be set up by the character of substances swallowed—*e.g.* when these are irritant, corrosive, or very hot articles. All these causes, though in different degree, offer some impediment to the act of deglutition.

**SYMPTOMS.**—Although difficulty in the act of swallowing is the essential symptom in many and various affections, yet this differs greatly in degree, as well as in the attendant phenomena, according to the pathological condition of the parts involved. Thus, when irritation of any kind is met with in any part of the track through which the bolus of food has to pass, then the act of deglutition is attended merely with more or less pain, which in such cases constitutes the sole difficulty in the process. When, however, the calibre of this portion of the alimentary canal is reduced by disease affecting its own structure, or when it is encroached upon by morbid growths or other disease in its immediate vicinity, a mechanical impediment is set up, which necessitates a certain amount of voluntary effort to accomplish the act. This supplementary aid is usually sufficient to propel the bolus onwards; and deglutition, though slower than in health, and usually attended with pain, may be successfully performed. It occasionally happens, however, that the obstruction is so great as to prevent the passage of at least the larger portion of the food downwards, and regurgitation takes place through the mouth or nostrils.

A similar result is brought about when paralysis affects any portion of the muscular structures concerned in the act of swallowing, but obviously in a different manner. For example, in post-diphtheritic paralysis, in

consequence of the implication of the soft palate and neighbouring structures in this loss of power, the food, instead of passing into the gullet, returns through the posterior nares.

The dysphagia occasionally observed in hysterical persons, and which appears to partake of the nature of spasm, differs essentially from those forms already described, in the fact that it is not a constant phenomenon, and that it is but one manifestation of the neurotic temperament.

**TREATMENT.**—This will manifestly depend upon the recognition of the cause which gives rise to the impediment in swallowing. Cases of simple catarrh of the mucous membrane of the throat, and those produced by the action of irritant substances generally, yield, after a short interval, to the use of bland articles of diet and demulcents, such as olive oil, milk, linseed tea, &c. It must, however, be borne in mind that permanent stricture of the œsophagus may be the result of causes such as those last mentioned. Of course, when abscess is the cause of the dysphagia, the evacuation of its contents will give immediate relief. In that form of dysphagia dependent upon diminution of the calibre of the œsophagus, the question of its treatment by the use of bougies or stomach-tubes should be considered. *See* ŒSOPHAGUS, Diseases of.

The dysphagia dependent upon specific disorders of the larynx, such as that occasioned by phthisis, syphilis, or cancer, may often be greatly mitigated by the use of warm medicated sprays containing sedative drugs, such as bromide of ammonium, opium, or chlorine water; a 15 or 20 per cent. solution of menthol in liquid vaseline or olive oil, injected into the larynx and pharynx; also a 4 per cent. solution of cocaine, &c. In cases acknowledging a nervous origin, the treatment must bear reference to the general nervous disorder of which the dysphagia is but a symptom. Thus, hysterical dysphagia may be speedily removed by the application of galvanism in the neighbourhood of the œsophagus. Post-diphtheritic dysphagia usually disappears as the health of the patient improves, and is to be treated by the administration of nervine tonics, such as strychnine, iron, and quinine.

The food should be of semi-solid consistence, concentrated, and small in bulk—*e.g.* iced milk, cream-ice, jellies, switched egg, or the unbroken raw egg, custard, and oysters.

C. MUIRHEAD.

**DELHI SORE OR BOIL.**—**SYNON.:** Aleppo Evil; Mycosis Cutis Chronica (V. Carter); Lupus Endemicus (Lewis and Cunningham); Oriental Sore (Fox). *Fr. Bouton d'Alep; Ger. Beule von Alep.*

**DEFINITION.**—An indurated, indolent, and very intractable sore; papular in the early,

encrusted or fungating in the advanced stages; spreading by ulceration of skin; single or multiple; and often occupying extensive surfaces of the exposed parts of the body, such as the face, neck, and extremities. It is capable, if inoculated, of reproducing the disease; and it also affects dogs and horses.

**GEOGRAPHICAL DISTRIBUTION.**—This disease occurs in India, especially the North-west Provinces, Punjab, Cabul and Scinde, Persia, Arabia, Crete, the Sahara of Africa, perhaps China, and doubtless wherever certain peculiar conditions of soil and hot climate coexist; for example, an epidemic lately (1889) prevailed in Greece. Though called Delhi boil, it is neither a furunculus, nor is it peculiar to that city. The Scinde boil, the sores of Roorkee, Multan, Lahore, Meerut, other crowded Indian cities, and Aden, are probably only varieties, if not identical. The same may be said of the Bouton d'Alep of Biskra, Bussorah, Baghdad, and Crete. Slight differences may exist, but essentially they are the same disease. The Yeman and Cochin China sores are probably varieties, as are other indolent indurated and intractable sores occurring in persons of impaired health, residing in hot and malarious climates, who use certain waters, and in whom there is neither syphilitic nor strumous taint.

**ÆTIOLOGY AND PATHOLOGY.**—Drs. Fleming and Smith, V. Carter, and Lewis and Cunningham give the best account of the disease. Though called a local disease, it is probable that the state of the health has much to do with its production, certain conditions of climate, soil, and especially of drinking-water being also concerned. Furunculi of a severe and painful though different character are prevalent in hot climates at certain seasons of the year, in enervating and malarious climates like India, especially after the rains, *i.e.* towards the termination of the most exhausting season, when the vital powers have been depressed during the preceding months, and the functions of the liver and spleen are impaired. The blood, imperfectly elaborated, and not freed from excrementitious matter, is then in a condition in which it not only ministers imperfectly to nutrition, but is prone to fibrinous coagulations, which cause capillary embolism, giving rise to local starvation and death of minute portions of areolar tissue in or under the integument. These result in suppuration, which is set up for the purpose of getting rid of the dead fragment or core. An analogous, though perhaps not precisely similar, pathological condition may be concerned in the causation of the Delhi sore, and is not incompatible with an otherwise fairly good condition of the general health. Water, soil, food, bites or stings of insects, parasitic micro-organisms, insanitary conditions, such as exist in crowded native cities, have all been charged with causing the disease. Improved hygiene, planting of trees, and change of water, food, and locality,

have all been credited with benefit in the treatment of it. The facts that this peculiar form of sore manifests itself under similar climatic conditions in other parts of the world, which, it is to be noted, are generally those of the more arid regions, and that it occurs most frequently at the most exhausting season, seem to point to a constitutional state as a predisposing cause.

The disease is not confined to human beings. In Delhi it has been observed that dogs are very liable to be affected, especially in the nose, and this, from the position of the sore, has been adduced as strong evidence in favour of the theory that it is due to the presence of a parasitic organism which finds its way there from the water. In some districts other animals are affected; and it seems probable that the indolent, indurated, and intractable sore that horses are liable to in India, called *Bursattie* (Rain Sore), is of the same character. Further investigation into the causation and pathology of Delhi boil is needed, especially with reference to the action of drinking-water, and the nature of the structures that form the essential constituents of the disease.

**ANATOMICAL CHARACTERS.**—When the Delhi sore is cut into, yellowish points are seen, consisting of minute cellular growths, which have been described by Dr. Smith as the ova of a parasite (*Distoma*), and by others as of vegetable origin, but are probably the result of cell-growth, connected with the hair- and gland-follicles, perhaps an abnormal development of connective-tissue corpuscles, or an imperfect form of granulation. After ulceration has disintegrated the surface, mycelium or other low forms of organism may be present; but it is a question if these be the essential cause, and not rather an accident of the disease, introduced from without. Dr. V. Carter refers Delhi boil to a parasitic organism, consisting of spheroids and mycelium, which occupies the distended lymphatic vessels in and around the sore, arranged in open and angular meshes, the free ends giving off conidia which multiply and reproduce. Pale, round or stellate granulation-cells are found; and numerous bright orange-tinted particles, arranged as spherical or ovoid groups disseminated throughout the tissues of the tumour. These, it is considered, are the fructification-stage of the fungus. Lewis and Cunningham described lymphoid nucleated cells, the products of a condition which they considered as identical with that of lupus, and which they ascribed to the action of the chemical constituents of certain hard waters; but in a more recent paper, dated 1885, Dr. Cunningham observed, in a specimen of Delhi boil which had not yet ulcerated, certain microscopic bodies, probably zoocysts or sporocysts of some monoclinic origin, which he believes to have been connected with the causation of the disease for more than one

reason, the chief being their presence in greater numbers where the greatest morbid changes had taken place, and in the water in certain localities where the disease occurs.

**SYMPTOMS.**—Delhi sore commences as a small pink and reddish papule, like a mosquito-bite, which gradually extends, generally around a hair-follicle as its centre. This is elevated, and after a time desquamates. There is itching and a stinging sense of pain; on pressure it is somewhat boggy. The progress of the disease is slow, often occupying several weeks, during which time it assumes a semi-transparent appearance, with blood-vessels ramifying near the surface. A vesicle then rises, bursts, and gives exit to an ichor which forms a crust; under this, suppuration and ulceration take place and advance until, by the coalescence of several papules and destruction of skin, an indurated sore is formed, which is either crusted over or fungated. The sore gradually invades the surrounding parts, and, destroying the integument, may give rise, especially on the face, to deformity from cicatricial contraction; and, from the irritation and the pain it causes, may, when the number and extent of the sores are large, seriously compromise the health. An ordinary boil or abrasion may assume these specific characters. The disease is regarded as contagious, and apparently may be produced by inoculation of the specific cell-matter, though not by the pus which forms on the surface.

**TREATMENT.**—*Preventive.*—Cleanliness of person, clothing, and habitation, good food, the use of pure drinking-water, and careful attention to the sanitary condition of the locality—avoiding overcrowding and contact with the disease in men or animals,—are the best means of preventing Delhi sore.

*Curative.*—The most effective measures of a curative kind are—change of locality when practicable; in some cases early destruction of the sore by the potential or actual cautery; the application of metallic astringents, iodine, or carbolic acid lotion; pressure; attention to the state of the health, and any ailment that may be present; tonics and nutritive diet, and especially wholesome drinking-water; and change to another climate.

In the advanced conditions of the disease similar measures are indicated. The sore, if too extensive to be destroyed, should be dressed with stimulating and astringent applications. Soothing measures are indicated if there is pain. Black wash, sulphate and sulpho-carbolate of zinc, copper, Gurjun oil, and lime-water, with change of climate and the use of tonics, will generally prove efficient.

JOSEPH FAYREK.

**DELIRIUM** (*deliro*, I rave).—A derangement of consciousness, characterised by incoherence of thought, and evidenced by various expressions and actions. See CONSCIOUSNESS, Disorders of.

**DELIRIUM TREMENS** (*delirium tremens*, trembling delirium).—A form of acute alcoholism, chiefly characterised by delirium and tremors. See ALCOHOLISM.

**DELUSION** (*deludo*, I deceive).—An unfounded belief in regard to some fact or occurrence which almost invariably concerns the patient, and of the falsity of which he cannot be persuaded, either by the evidence of his senses, by his own knowledge and experience, or by the declarations of others. Such delusions, when distinguished from merely erroneous judgments upon complicated or abstract questions, generally indicate insanity. See CONSCIOUSNESS, Disorders of; and INSANITY.

**DEMENTIA** (*de*, without; and *mens*, a mind).—SYNON.: Fr. *Démence*; Ger. *Blödsinn*.

**DEFINITION.**—A mental weakness, or a deficiency rather than an aberration of intellect, depending for the most part on some antecedent brain-disorder, such as apoplexy or epilepsy; or being the sequel and termination of various forms of insanity. It may also be congenital, deserving rather the name of idiocy; or it may be due to senile decay.

1. **Acute Primary Dementia.**—There is a form of insanity known as primary or acute dementia, which comes on rapidly without any preceding disorder, is accompanied by the most profound vacuity and abeyance of all the mental faculties, yet yields to treatment and disappears, leaving the patients sane.

**ÆTIOLOGY.**—The patients are young persons, boys and girls,—more frequently girls. The complaint is seldom seen in any case beyond the age of 30, and chiefly in those under 20. It seems to be a collapse of all mental power, due to great physical weakness and deficient nerve-force. Owing to imperfect development, to bad food and living, or to the patients having outgrown their strength, the mental condition becomes so weakened that, with or without some moral cause, as a fright or a scolding or something apparently more trivial, or after some illness slight or severe, they suddenly or gradually present that condition which is now to be described. If the immediate exciting cause is some mental shock or fright, the symptoms may come on rapidly. If they are due to ill-health or some protracted exhausting occupation, the access may be gradual; and if they are set down to sulkiness, temper, or idleness, the measures adopted for the correction of the latter may quickly indicate the real state of things.

**SYMPTOMS.**—Nothing can appear more hopeless than the appearance many of these patients present. The face is vacant, with a fatuous grin, and often the saliva dribbles continuously. The sufferer sits motionless and lost, or automatically wags the head,

snaps the jaws, or moves the limbs for hours together, unconscious of fatigue. Or if a limb is placed in any position, it is retained there for a time in a way that no effort of will could accomplish. There may be a repetition of some word or sentence, but all conversation is abolished, and the patient has to be fed, washed, and tended like a baby.

The physical condition of these patients is peculiar, and corresponds closely to the mental. The heart's action and the circulation are so reduced in strength that the blood in the extremities is stagnant. Hands and feet are blue with cold even in the heat of summer. In cold weather they are covered with chilblains, and great care must be taken, otherwise these will give rise to obstinate sores. The tongue is pale and flabby. The pupils are dilated. There is no rapid emaciation, for the waste here is not great; neither is sleep absent, as in mania, but it is irregular and uncertain.

**PATHOLOGY.**—The external physical manifestations sufficiently indicate the condition of the brain in these patients. It is the very opposite of that in acute sthenic delirious mania. In the latter there is an excessive discharge of nervous force, a hyperæmic state of brain, and rapid brain-circulation; the whole leading in a short time to death by exhaustion, if relief does not come. In acute dementia we see the very opposite. The brain-action is reduced to the lowest point, and the circulation is stagnant, as in the extremities, giving rise to passive congestion and œdema.

**COURSE, TERMINATIONS, AND PROGNOSIS.**—In acute dementia there is no sudden exhaustion; but death, if it occur, is caused, not by the brain-disease, but by a general failure of the bodily strength, or by some low form of lung-disease—phthisis, pneumonia, or gangrene. Death, however, in this disorder, is the exception. When taken in time and properly treated, the majority of these seemingly hopeless cases recover, and recover perfectly.

**TREATMENT.**—The treatment of acute dementia may be carried out in a family, or even at home, if means are ample, and if the necessary measures are strictly enforced. But it may be necessary to feed the patient by force, and that for a considerable time, and relations do not always care to enforce this to the extent required. Abundant nutrition is imperatively demanded in order to restore the force that is so greatly in default; and unless abundant nutrition is administered, there will be no recovery, but the patient will die, or sink into permanent dementia. There is not, as a rule, violent resistance to food, but it may be kept in the mouth without being swallowed, and care must be taken in feeding, even if a stomach-tube be not necessary. Food should be given frequently, and so a habit of taking it engendered. Stimulants—

wine and brandy—will be necessary, especially in the early stages. Equally necessary is warmth; an amount of heat is required which to those in health would be oppressive, for the greatest heat of summer fails to warm the hands and feet. Warm clothing must be provided, and the circulation aided by a short sharp shower-bath, cold or tepid, and plenty of friction afterwards. Exercise is useful for the same purpose, but this is to be taken under proper supervision, for it must not be fatiguing, and due regard ought to be had to the debilitated state of the individual. In addition to the stimulus of the shower-bath, that of electricity is of great use in acute dementia. Here, and in certain cases of melancholia, marked benefit follows the application of the continuous current. Of drugs the most useful appear to be steel and quinine. The former, in this as in almost every form of insanity, is a most valuable tonic: the choice of the particular preparation should depend upon the state of the patient at the time.

**2. Chronic Primary Dementia.**—Dementia, however, may be primary, yet may not be that just described. It may come on gradually or suddenly, without previous mental affection, but is in such cases connected almost invariably with disease of the brain. Its first and most prominent symptom is loss of memory. In connexion with apoplectic or epileptic attacks, or after years of drinking, the memory is found defective. This loss may appear quite suddenly, or may be noticed to come on gradually, being at first so slight as to cause little alarm. It may vary at different times. If a patient is kept from alcohol, the memory may gain strength; and if epileptic attacks are reduced in frequency, the same thing may happen. The prognosis in all such cases is unfavourable, for loss of memory points to decided deterioration of brain. Apart from loss of memory, symptoms of dementia or weakness of mind are occasionally found as the first indication of mental unsoundness, following a fright or shock or some severe illness. If they assume the form of acute dementia, we may have hopes that they will pass away; but if with little disturbance of the bodily health the mind becomes weaker and weaker, the hopes of recovery are small.

**3. Secondary Dementia.**—Of dementia which is the sequel of prior mental disorder, such as mania and melancholia, little need be said. It varies in degree, but it is not in our power to remove it. It may be in our power, however, to ameliorate in a great degree the condition of such patients when, as is frequently the case, they are found in a very neglected state. Their friends think that nothing can be done or need be done, and they are allowed to lie in bed, often in a filthy condition, or roam about and get into mischief for want of care and skilled

attendance. Many demented patients have far more mind than is generally imagined by the uninitiated, and can be taught to be cleanly, to take their meals in an orderly fashion, and to keep themselves tolerably neat. They are susceptible of amusement, and open to reward for good behaviour. The vital powers of demented patients are low, and they suffer much from cold. In winter their minds, like their bodies, are enfeebled, and with warm weather they recover somewhat of their energy. There is a tendency in many of these patients, especially women, to become very fat, and in this condition they are subject to bronchitis, and may succumb to acute attacks of this disorder. They require warmth and good diet, for it is difficult to make them take sufficient exercise.

Females, in the writer's experience, are more prone to drift into dementia than males. The latter present various types of chronic mania, with well-marked delusions; but, among private patients at any rate, there are fewer of the hopelessly demented than among the female inmates of private asylums. One cause of this may be that the mortality amongst males is greater, and thus fewer are left to reach the demented stage.

G. F. BLANDFORD.

**DEMODEX** (*δημός*, fat; and *δήξ*, a worm).—See ACARUS.

**DEMULCENTS** (*demulceo*, I stroke softly).

**DEFINITION.**—Substances which soften, protect, and soothe mucous membranes. They are generally of a mucilaginous character. When applied to the skin they are termed *emollients*.

**ENUMERATION.**—The demulcents in ordinary use are: Linseed-tea, Gum, Starch, Bread, Honey, Figs; Linseed, Almond, and Olive Oil; Glycerine, White-of-Egg, Gelatine, and Isinglass.

**ACTION.**—The chief action of demulcents is a mechanical one, in forming a smooth, soft coating for an inflamed mucous membrane, and thus protecting it from external irritation.

T. LAUDER BRUNTON.

**DENGUE.**—**SYNON.**: Dandy Fever (West Indies); Three-day Fever; Break-bone Fever; Fr. and Ger. *Dengue*.

**DEFINITION.**—An eruptive fever, considered by many to be infectious; commencing suddenly; and characterised by severe pain in the head and eyeballs, swelling and pain in the muscles and joints—prone to shift suddenly from joint to joint, catarrhal symptoms, sore-throat, congested conjunctivæ, and affection of the submaxillary glands. The disease may remit, and is liable to relapse.

**NATURAL HISTORY AND GEOGRAPHICAL DISTRIBUTION.**—Dengue occurs epidemically and sporadically in India, Burmah, Persia, Egypt and other parts of Africa, North and South

America, and the West Indies. Epidemic visitations of dengue, extending over wide tracts of country, occur at considerable intervals, and probably depend on certain unknown atmospheric and cosmic conditions that favour its development.

**ÆTIOLOGY.**—Dengue attacks persons of all ages, from infancy to extreme old age. Its infectious character has been deduced from the apparent transmission of the disease from person to person.

**SYMPTOMS.**—The symptoms characteristic of dengue are the presence of severe continuous arthritic and muscular pains; great debility and prostration; the occurrence of an initial and a terminal rubeoloid or scarlet rash; fever, which is subject to remissions and relapses; the possibility that convalescence may be tedious and painful, and complicated by the continuance of general cachexia, pain and swelling of joints, enlargement of glands, orchitis, weakness of eyes, deafness, visceral disease (such as diarrhoea or dysentery of a chronic and intractable character, and hepatic derangement), boils, carbuncles, and perhaps insanity. In the female, uterine hæmorrhage and miscarriage may occur.

The *invasion* of dengue is usually sudden, the patient feeling well up to the period of attack. The earliest symptom is severe pain in some joint, probably of a finger, which rapidly extends to all the other joints and bones; and this pain during the progress of the disease often passes from one joint to the other by a sort of metastasis. Sometimes there is a period of preliminary malaise, of one or more days' duration, marked by anorexia, a sense of weariness and languor, giddiness, nausea, chilliness or rigor, severe pain in the head or in the eyeballs, and pains in the body, limbs, and joints—notably of the fingers and toes. The attack is often, however, strikingly sudden, as was frequently seen in the late Calcutta epidemic, commencing with violent pains and swelling of the joints, or severe pains in the head, eyeballs, neck, and back. In some epidemics certain phenomena are more prominently marked than others.

The eruption commences on the third day. The fever is accompanied by redness of the face, which is puffy and swollen; sore-throat; congested conjunctivæ; and a general redness, like the scarlatinal rash, extending over the whole body. The tongue is red at the tip and edges, and loaded with white fur, through which the red papillæ protrude. The pulse is rapid, ranging from 100 to 120, or even 140; respiration is hurried; and the temperature rises to 103° or even 105° F. These symptoms mark the occurrence of the initial fever and rash, and endure for a period varying from twenty-four to forty-eight hours. After this the rash disappears; the fever subsides; and the remission lasts for a period of two, three, or four days. A recurrence of febrile symptoms then takes place, accompanied by a

second or terminal rash. This differs in character from the first, resembling a rubeoloid or even an urticarial eruption, often showing itself first on the palms of the hands, and in some cases resulting in profuse desquamation of the cuticle, which, however, may sometimes be so slight as to be barely perceptible. These symptoms gradually subside, leaving the patient weak, exhausted, and often still tortured by swelling and pain of the joints, especially the smaller ones, which may continue in this state for weeks, making convalescence tedious and painful. Or there may be repeated relapses, prolonging the suffering and protracting recovery.

**VARIETIES.**—The symptoms vary in different cases, as to the character of the rash, the temperature, and the muscular or osseous pains.

The *rash* not only varies considerably in colour, character, and duration, but it is sometimes almost absent; in other cases it is attended with so much hyperæmia and action of the skin that excessive desquamation results. This hyperæmia also sometimes expresses itself by hæmorrhage from the mouth, nose, bowels, and uterus.

The *fever* is sometimes accompanied by delirium, or in children by convulsions; in the latter, indeed, these occasionally initiate the disease.

Dengue occasionally assumes a *malignant* form, where the amount of poison received has been overwhelmingly large. Dr. Charles says: 'Drowsiness may have passed into coma; the temperature verges on the hyperpyrexia; the heart fails, and the lungs are œdematous; while the whole surface is highly cyanotic. These cases have been popularly termed "black fever," and are justly much dreaded.' Happily they are rare. Again, there are very mild forms of the disease, in which the patients are scarcely ill, and where it is not easy to decide as to their exact nature: 'A trifling sore-throat and slight malaise may be all you can lay hold of till the terminal rash appears to show you what you have had to deal with; and even this may not be seen.'

**COURSE, DURATION, AND COMPLICATIONS.**—The period of *incubation* of dengue is probably from five to six days; it may be a day or two more or less in some cases.

In simple and uncomplicated cases the average period for the *duration* of the disease may be taken as about eight days; but it is frequently prolonged over weeks, and recovery is slow and painful; the constitution often being so much shattered that complete restoration to strength and vigour does not occur for months. It is rarely fatal. *Relapses* are liable to be frequent, and the patient may suffer more than even a second or a third relapse before recovery.

Some of the *sequelæ* already mentioned may remain to torture the patient and retard

his recovery. Albumen is occasionally present in the urine; but it is not, as in scarlatina, especially in the cases of children, a frequent or dangerous result of the disease.

**DIAGNOSIS.**—The distinction betwixt scarlatina and dengue is well marked; though during the outset there is considerable resemblance between the two diseases. There is a high temperature at first in both, but it is more quickly attained, and is transient in dengue; in scarlatina it endures for several days, whilst in dengue the fastigium gives a temperature of 103°, or even up to 105° or 107°, and, this being attained, it rapidly declines. It is exceptional to find a temperature above 102° maintained in dengue. In scarlatina the period of decline extends over several days, and is marked by slight exacerbations in the evening. In dengue it occupies a few hours, and the temperature may even fall below the normal. The severe muscular and arthritic pains of dengue do not occur in scarlatina; and the pulse in the latter is much more rapid during the early stages than in the former.

**PROGNOSIS.**—An attack of dengue does not confer absolute protection from a recurrence of the disease, though it does so to a great extent.

**TREATMENT.**—This is a specific fever, and has to run a certain course. The treatment is simple, and if judiciously directed mitigates the sufferings, and materially aids recovery.

Neither emetics nor active purgatives are necessary. They do no good, but increase the weakness and aggravate the suffering by the muscular movements necessarily induced. Moderate action of the bowels is advisable, a warm carminative aperient, or an occasional dose of calomel, rhubarb, or colocynth being administered, especially if the bowels remain confined, to which there does not appear to be any peculiar tendency, though the evacuations may be dark and often slimy, and confined at the outset. Salines, such as the acetate of ammonium, or citrate of potassium, with nitric ether, combined with aconite, are good during the pyrexia. In cases of very high temperature (105° to 107°) Dr. Charles suggests cold sponging as beneficial; he recommends it when 105° is reached. The danger to life of such a high temperature during the intense heat of the hot months in India is great; and it is then that cold sponging or the cold bath is indicated.

Belladonna seems to confer great relief in this disease. Ten to fifteen drops of the tincture may be given; and two or three such doses, at intervals of an hour, will sometimes produce excellent effects, and afford much relief. The extract may be given if preferred, in doses of one-third of a grain; or the juice in similar doses to those of the tincture. For the pains and nocturnal restlessness, morphine or Dover's powder may be given. Liniments containing

opium, belladonna, and chloroform are serviceable as external applications to the spine, back, and joints.

Tonics, and a carefully-regulated nutritious diet, are also indicated, and depletive measures must be avoided. The tonics should be of the bitter vegetable kind, such as gentian and calumba; with these may be combined a small quantity of quinine, with a mineral acid; or, in some cases, the dilute phosphoric acid, combined with nux vomica or small doses of strychnine.

Quinine is given more for its tonic than for its antiperiodic effects; though, where there is a tendency to relapses, the judicious administration of five- or even ten-grain doses may be beneficial in arresting them and reducing temperature. Bromide of potassium is recommended by some authorities, and especially when convulsions occur in children. Alkalis, colchicum, and other remedies in use in rheumatism have been found to have little, if any, effect in relieving the pains of dengue.

As to wines, claret is probably the best, but others may be given.

For the irritation of the skin, which is sometimes very troublesome, the application of camphorated oil and the use of warm baths have been suggested.

As in so many other diseases, especially those that occur to Europeans in tropical climates, complete restoration to health is likely to be expedited and promoted by change of air; and if the cachexia be severe after a prolonged attack of dengue, return to the patient's native climate for a season is desirable.

JOSEPH FAYRER.

**DENTITION, Disorders of.**—SYNON.: Teething; Fr. *Troubles de la Dentition*; Ger. *Zahnung*.

**GENERAL REMARKS.**—The period of cutting the teeth has been always recognised as a critical time, during which the health of young children is especially liable to become disturbed; and it has been a common practice amongst mothers to attribute every illness occurring in early life, from the irritation of scabies to the distortions of rickets, to the same baneful influence. The evolution of the milk-teeth is no doubt attended with some irritation, especially as at this period the follicular apparatus of the intestines is undergoing considerable development; and we know that, on account of the impressibility of the nervous system in young children, any irritation is apt to be followed by general disturbance. It is no doubt also the case that local functional derangements are frequent at this period, but it is often unfair to attribute these *directly* to the irritation of an advancing tooth. One of the most common direct results of teething is pyrexia, which may be intense; and a feverish child is particularly susceptible to impressions of cold,

and to the irritation of unsuitable food. Catarrhal attacks coming on at this time need not be therefore the immediate result of the condition of the gum. It is at least admissible to attribute them to the ordinary causes of such derangements, acting upon a body rendered for the time particularly susceptible to injurious influences. This view is supported by the fact that diarrhœa, which is a very common complication of dentition, is especially frequent during the warmer months, when the temperature is apt to undergo rapid variations, while the dress of the child remains unchanged; and is far less common during the winter, when the temperature is more uniform, and the child is more carefully guarded against the cold.

As a rule, the first milk-tooth appears in the seventh month after birth; but dentition may begin at an earlier period. It is not rare for an infant to cut a tooth at the age of four months; and occasionally at the time of birth one tooth is found to be already through the gum. In cases where the ordinary time of teething is anticipated, a pause generally ensues after the appearance of one or two teeth, and further dentition is delayed until the usual age. Constitutional conditions influence the time of teething. Thus tuberculous and syphilitic children cut their teeth early, while in rickety children the teeth are very slow to appear.

In the majority of cases the teeth pierce the gum in the following order: lower central incisors, upper central incisors, upper lateral incisors, lower lateral incisors, first molars, canines, back molars. A child of twelve months old should have eight teeth and be cutting his first molars; and the whole number (twenty) should be through the gums soon after the end of the second year. The order given above is frequently departed from. The incisors, in particular, are often cut irregularly, and it is not rare for the molars also to appear out of their proper order. A double tooth will often be seen to precede the lateral incisors, and may indeed in exceptional cases be the first tooth to appear through the gum. The canines seldom or never precede the first molars.

Some infants suffer more than others from the cutting of a tooth, and it is not always in cases where the eruption of the teeth has been delayed that dentition, when it occurs, is attended with special inconvenience. On the contrary, in severe rickets, where the delay is great, the teeth are often cut with remarkable ease.

The second dentition begins at about the sixth year. The permanent teeth are cut with more regularity than is the case with the earlier crop. The first to be seen are usually four molars, which appear behind the last of the temporary teeth. Next come the central incisors at about the eighth year; the lateral incisors at about the ninth; the

first and second bicuspids, in the place of the temporary molars, at the tenth and eleventh; the canines between the twelfth and thirteenth; and the second molars at about the time of puberty. The last four permanent molars are cut later.

**SYMPTOMS.**—The phenomena which may be looked upon as natural to the first dentition are salivation; swelling of the gum, which becomes more and more tense, hot, and painful; slight general pyrexia, with flushing of one or both cheeks; irritability of temper; and some degree of restlessness at night. These all subside when the point of the tooth appears through the gum. The complications not necessarily attendant upon the process are high fever; inflammation of the mouth and aphthæ; vomiting (gastric catarrh); diarrhœa (intestinal catarrh); cough (pulmonary catarrh); otitis, with purulent discharge from the auditory meatus; various eruptions of the skin, with, sometimes, swelling, and even suppuration of lymphatic glands; and certain troubles of the nervous system, such as convulsions, squinting, &c.

The peculiarity of the pyrexia of dentition is its irregular character. It is often higher in the morning than at night, and varies in intensity in a remarkable manner from day to day. A temperature of 104° F. at 8 A.M. is not at all uncommon in a teething infant; indeed such an amount of fever in the morning should alone lead us to inspect the mouth, as few diseases are marked by so much pyrexia at that hour of the day. It is important in practice to bear in mind this simple cause of elevation of temperature, for any disease in a child is apt to be complicated by teething, and much needless anxiety may be occasioned by overlooking the condition of the gum. We must not, however, in every case where the gums are swollen and tense, rush to the conclusion that they are the sole cause of the symptoms, for the most serious cerebral disease may co-exist with the eruption of a tooth. Thus, to take an example: if we find cerebral symptoms to supervene in the course of dentition, we must most narrowly scrutinise their character, before passing them over as merely harmless indications of the general disturbance. Headache, delirium, vertigo, startings, twitchings, and convulsive attacks, may merely indicate functional disturbance of the brain, such as is common to many disorders, and these phenomena are not necessarily symptomatic of cerebral disease; but if the bowels become obstinately confined, the pulse slow and irregular, and the respiration unequal and sighing; and if in addition there be photophobia, with sullenness and tendency to drowsiness, we may conclude that something more than mere functional derangement is present, and that there is every reason to suspect the existence of tubercular meningitis

In the case of the second dentition there may also be digestive derangement. During this period the child is subject to gastro-intestinal catarrhs, which give rise to looseness of the bowels. He is often irritable in the day and restless at night. Night terrors are common, and he may wake up from his sleep screaming with fright at some horrible dream. Besides the discomfort due to fermentation of food and acidity, considerable loss of flesh may be the consequence of digestive derangement if long continued.

**TREATMENT.**—The treatment of the complications which occur during dentition must be conducted upon ordinary principles. Aphthæ of the mouth are readily cured by the administration of rhubarb and soda, and the application to the mouth of a solution of chlorate of potassium or borax (ten grains to the ounce) in water, and sweetened with glycerine. Perfect cleanliness is necessary, and the child's mouth should be washed out each time after taking food with a piece of soft rag dipped in warm water.

Vomiting is best checked by clearing out the stomach with an emetic of ipecacuanha wine, giving a tea-spoonful every ten minutes until sickness is produced. Afterwards, a few grains of bicarbonate of sodium may be given with one drop of liquor arsenicalis in a tea-spoonful of water three times a day. Diarrhœa should be treated on the same principle: first a dose of castor oil to remove irritating products from the bowels; then a mixture containing chalk and catechu, or oxide of zinc (one grain to the dose). If afterwards the motions continue large, pasty-looking, and offensive, and are passed too frequently in the day, one drop of tincture of opium may be added to the mixture, as there is usually in such cases too rapid peristaltic action of the intestines. In the case of either of these derangements (vomiting or diarrhœa), it is of great importance to keep the body warm, and this is most effectually done by applying a broad flannel bandage to the abdomen. The diet also should be temporarily modified, reducing the quantity of farinaceous matter that is being taken, on account of the tendency to acid fermentation of food which is set up by such a condition of the alimentary canal.

Looseness of the bowels during dentition has been looked upon by some writers as a natural method of relief to the system, and fears have been entertained of grave troubles which might ensue if the looseness were too suddenly arrested. Such fears are, however, quite groundless. A catarrhal condition of the bowels should be cured as quickly as possible, especially during dentition, for at this time the susceptibility to chills is heightened, and the danger of severe diarrhœa being set up correspondingly great. In some cases of teething, where the lungs as well as the bowels are the seat of catarrh, and there is

a risk of bronchitis, Trousseau recommends that the intestinal derangement should not be suddenly put an end to; but even in these cases a dose of castor-oil may be safely given, to remove irritating matters from the canal, for, according to the writer's experience, any irritation of the bowels is apt rather to increase than to diminish pulmonary mischief.

The favourite remedy for all disorders occurring at the time of dentition is lancing the gums, but the practice is one which ought not to be pursued indiscriminately. Unless the gum be actually swollen and tense, incising it has no object whatever, for to cut the top of the gum can have no influence in promoting the development of the tooth below. If, however, there be very much inflammation and swelling, and the child seem to be in pain, relief may be obtained by lancing, but in this case the object is merely to relieve tension. Excitement of the nervous system dependent upon the condition of the mouth can perhaps be allayed by the same means; but in the case of convulsions more benefit is to be gained by the use of warm baths, cold applications to the head, and the gentle action of a mild alterative aperient.

In the second dentition, care should be taken to keep the child's feet warm and his body properly protected from cold. Attacks of indigestion and diarrhoea must be treated by suitable remedies, and attention should be paid to the diet, limiting the quantity of starches and sweets allowed, on account of the tendency to fermentation and acidity. Night terrors, which are merely attacks of nightmare, can be cured at once in children by a mild aperient to clear away undigested food from the bowels, and by a careful diet. Sweets, starches, and fruit should be forbidden; and the child must be fed on meat, fish, poultry, milk, butter, cooked green vegetables, eggs, stale bread, and any kind of non-fermentable food suited to his age.

EUSTACE SMITH.

**DENVER**, in Colorado.—Situated 5,200 feet above the sea, on a plain backed by the Rocky Mountains. A dry, cold, clear, and bracing climate. Diathermancy considerable. Temperature: mean daily range, 24° F., showing great variation; mean temperature, 49°. Rainfall, 15 inches. A valuable high-altitude station for phthisical patients. Railway communication and accommodation excellent. For invalids the suburbs of Denver are preferable to the city itself.

**DEOBSTRUENT** (*de*, from; and *obstruo*, I obstruct).—A measure which removes obstructions, such as an aperient; or which opens the natural passages or pores of the body. See PURGATIVES; and DIAPHORETICS.

**DEODORANTS** (*de*, from; and *odoro*, I cause to smell).

**DEFINITION.**—The term 'deodorant,' although it has a more extended signification, is generally used to signify a substance that destroys offensive odours.

**GENERAL PRINCIPLES.**—Odorous bodies are essentially volatile, and those which are offensive frequently contain sulphur in some state of combination. Deodorants usually produce the effect for which they are used by causing a chemical change in bodies to which they are applied; but sometimes their action consists in absorbing and condensing odorous substances, and thus destroying or counteracting their volatility. Substances which, like charcoal, possess this latter property may, however, indirectly produce chemical changes by bringing the odorous substances into contact with oxygen in a condensed and active condition.

Deodorants may be classed as *volatile* and *non-volatile*.

**1. Volatile Deodorants.**—These all consist of substances the action of which is immediately and exclusively chemical. Being intended to act on bodies which are themselves volatile, they admit of more generally useful application than those which are not volatile.

**ENUMERATION.**—Chlorine and its lower oxides, Sulphurous Acid, Nitrous Acid and other oxides of Nitrogen, Ozone, and Peroxide of Hydrogen, are the most important members of this class.

**APPLICATION.**—In the selection and use of volatile deodorants, it is necessary to distinguish between bodies which possess the power of destroying or removing a noxious smell, and those which merely cover one smell by another. Carbolic acid, for instance, which is a valuable disinfectant, is of little use as a deodorant, although its powerful odour may render other weaker but more objectionable odours imperceptible or indistinguishable. On the other hand, the so-called chloride of lime (chlorinated lime of the Pharmacopœia), while it possesses a strong and characteristic smell itself, is capable of destroying other noxious odours, and is an excellent deodorant.

The chemical action by which noxious odours are destroyed is principally one of oxidation, and therefore the agents forming this class of deodorants are generally oxidising agents. Ozone, or active oxygen, is the natural deodorant contained in the atmosphere, which no doubt largely contributes to the destruction of noxious vapours in the air. Volatile oils, which emanate from the flowers and other parts of plants, in contact with atmospheric oxygen produce peroxide of hydrogen, and this as an oxidising agent possesses deodorising as well as disinfecting properties. The moderate and judicious use of perfumes may thus produce a beneficial

effect, although their undue employment, by hiding rather than removing what is objectionable, may be injurious to health, and cannot be recommended. The preparation called 'Sanitas' is principally a solution of peroxide of hydrogen.

**2. Non-volatile Deodorants.**—**ENUMERATION.**—Among this class of deodorants are included Charcoal, Earth, Lime, Oxide of Iron, Sulphate of Iron, Chloride of Zinc, Nitrate of Lead, and Permanganate of Potassium.

**APPLICATION.**—Although very efficient when brought into contact with the class of odorous substances referred to as noxious gases, these deodorants are less generally useful than they would otherwise be on account of their non-volatile character. Charcoal owes much of its efficacy as a deodorant to the surface-attraction and power of condensation which it possesses, by virtue of which it brings noxious gases, such as sulphuretted hydrogen, into contact with oxygen in a condensed and active state, so that they are burnt up and resolved into innocuous compounds, or compounds less noxious than those from which they are produced. Earth and oxide of iron, which, like charcoal, are used in the solid and dry, or nearly dry, state, absorb and combine with, or promote the combination of, noxious gases, producing innocuous products. Lime may be used either dry or in the state of milk of lime. The other substances named are used in the form of solution in water. Where large quantities of decomposing animal or vegetable matter are required to be deodorised, dry lime or solution of sulphate of iron (green vitriol) may be economically and advantageously used.

T. REDWOOD.

**DEPILATORIES** (*depilis*, without hair).—**SYNON.**: *Psilothron*; *Rusma*.

**DEFINITION.**—Depilatories are measures used for the destruction of superfluous hairs.

**Hypertrichosis.**—The patients who present themselves for treatment by depilation are, of course, almost always women. In the immense majority of cases the disfiguring hairs are on the upper lip, chin, neck, or in front of the ears, the annoyance and mental depression caused by them being often so extreme as to justify any procedure likely to give permanent relief, provided it is rationally employed and its limitations are properly understood. It must be carefully borne in mind, however, that in a certain number of cases hypertrichosis is only temporary, dependent perhaps on pregnancy or menstrual disorders, or associated with acne and other sebaceous affections; and that in these circumstances it may disappear spontaneously, or as the result of treatment directed to these associated conditions. As a rule, the disfigurement is first noticed at about the age of eighteen, and is

progressive till twenty-five or thirty, after which it remains permanent till the menopause, when it frequently becomes aggravated.

**ENUMERATION.**—Various caustic applications are occasionally used as depilatories. Sir Erasmus Wilson recommended the following:—Quicklime (three parts), sulphide of sodium or of barium (one part), diluted with starch (four parts). The powder, mixed with water to the consistence of a thin paste, is laid on the affected part by means of an ivory paper-knife, and allowed to remain for from five to fifteen minutes. It must then be carefully scraped off, when the hair, shrivelled and burnt, comes away with it. The skin must then be washed with water, dried, and anointed with cold-cream.

Although the immediate result of this method is sometimes satisfactory, a troublesome degree of dermatitis is generally occasioned by it. The hairs, too, always return, their roots not being destroyed, but only stimulated by the treatment; and the subsequent growth is invariably stronger, darker, and more disfiguring than the original, while considerable scarring too frequently results.

Many women keep the disfigurement due to hypertrichosis in abeyance by habitually extracting the hairs with forceps. There are many obvious objections to the procedure, but in some cases, where no other treatment is applicable, it answers fairly well; as its result, however, the growth becomes darker, stronger, and more bristly. In a certain number of cases good cosmetic results are obtained by bleaching the hair with a saturated solution of peroxide of hydrogen.

**Depilation by Electrolysis.**—The only efficacious and permanent method of destroying superfluous hair is by electrolysis. First practised by Michel, of St. Louis, U.S., in 1879, subsequently by Hardaway, G. H. Fox, and other American dermatologists, it has of late years been introduced to the continent of Europe and to this country, where, however, owing to its indiscriminate employment, too often by unscientific persons, the treatment appears likely to fall into unmerited discredit.

**INDICATIONS.**—Generally speaking, the cases in which electrolysis is most applicable are those where—(1) the growth is limited in extent, and stationary, not progressive; (2) the hairs are strong and dark; (3) the amount of downy undergrowth is slight; (4) the patient stands pain well, is sufficiently intelligent to understand the bearings and restrictions of the process, and is in a position to submit to its necessarily protracted duration. There is little doubt that downy hairs in the regions operated upon sometimes become dark and strong with undue rapidity as the result of electrolysis; but it is also more than probable that, independently of it, these hairs would ultimately, although more

slowly, become sources of disfigurement. The writer is of opinion that cases in which the growth is most abundant upon or confined to the upper lip—and, unfortunately, they form a considerable proportion of those who present themselves for treatment—are, as a rule, unsatisfactory, owing to the large number of hair-follicles there present, the extreme sensitiveness of the part, the small number of hairs which can be extracted at a sitting (seldom exceeding twelve to fifteen), and the amount of inflammatory œdema set up in the lax tissue.

**APPARATUS.**—The necessary apparatus for depilation by electrolysis comprises—(1) a reliable constant-current battery of from ten to twenty cells, the nature of the elements being a matter of indifference; (2) an absolute dead-beat galvanometer, graduated in milliampères, and preferably incorporated with the battery; (3) long, light, pliable rheophores; (4) a suitable needle-holder, connected with the negative pole of the battery, many forms of which are now made (those which have the current-interrupting apparatus in the handle are clumsy and inconvenient); (5) a fine needle made of steel or platinum, or of gold or platinum tipped with iridium—(the writer, after giving all these a fair trial, has reverted to the finest steel sewing-needles—Abel Morrall's 12's, the only disadvantage of which is their brittleness); (6) a carbon or brass cylinder covered with chamois-leather, constituting the positive electrode, and connected with the positive pole of the battery. A steady hand and good vision are indispensable on the part of the operator, all suggested arrangements of magnifying-glasses being unsatisfactory. The best of such is a watchmaker's glass mounted on a spectacle-frame.

**METHOD.**—The patient being placed in a good light, and preferably in the recumbent posture, the needle is introduced into the hair-follicle for a distance of  $\frac{1}{16}$  to  $\frac{1}{8}$  of an inch or more, the follicle being, as it were, catheterised. The experienced touch is even able to distinguish when the bottom of the follicle is reached, by a slight increase in the resistance encountered. The current is closed by the patient firmly grasping the positive electrode, previously well soaked in warm water, and a sharp pain is experienced at the moment of closure, which persists, but in a modified form, throughout the destruction of the hair. The hair may then be seized with forceps, held in the left hand; and, when destroyed, it comes away without appreciable traction. The current is then broken by the patient relaxing the grasp of the positive electrode, and the needle is withdrawn. Whilst the current passes, a little froth, evidence of the electrolytic action, wells up by the side of the hair, and an urticarial wheal forms. This soon subsides, leaving a pink, hardish papule, which lasts a

few days; whilst occasionally a minute scab forms, which may take about a week to separate. In either case a tiny white depressed scar is left, but it is usually practically imperceptible after a short time. Occasionally, however,—especially in persons of dark complexion—the scars are rather deeply pigmented, and the resulting disfigurement disappears but slowly. A current of five milliampères employed for half a minute is sufficient to destroy the strongest hairs, if the follicle is accurately struck; and from six to ten cells are usually sufficient to generate this current strength. Weaker currents are effectual in destroying finer hairs, but it is strongly inadvisable to attempt to destroy lanugo. The amount of pain experienced varies within very wide limits; it is seldom so intolerable as to contra-indicate the operation. In some cases ointments containing 10–20 per cent. of cocaine, rubbed in for ten minutes beforehand, diminish it sensibly. The number of hairs extracted at a sitting ought never to exceed fifty, and an interval of several days ought always to be observed between the destruction of hairs close together. A few recurrences necessarily take place, their number being in inverse ratio to the expertness with which the operation has been performed. They are easily recognisable, and are generally most abundant beneath the chin and on the neck, where it is difficult to determine the precise direction of the hair-follicles.

It is impossible here to dwell upon all the details of the procedure, the success of which mainly depends upon skill, patience, and experience.

J. J. PRINGLE.

**DEPLETION** (*depleo*, I empty out).  
SYNON.: Fr. *Déplétion*; L'Action de vider; Ger. *Entleerung*.

**DEFINITION.**—By the term 'depletion' is understood: (a) the unloading, or rendering less full, of that which is over-burdened or over-full, for example, portions of the turgid vascular system—as the portal vessels; or (b) excessive evacuation, causing exhaustion—as in choleraic or other severe diarrhœa.

**USES.**—Depletion, local or general, as a therapeutic agent, may be practised in a variety of affections, such as cerebral congestion, venous turgescence, engorgement of the portal system, pulmonary congestion, renal ischæmia, aneurysm, or general plethora.

**METHODS.**—The agencies whereby depletion may be produced are blood-letting, general or local; purging; vomiting; sweating; and abstinence from food and drink.

1. **General Blood-letting.**—There is no more powerful or prompt depleting agent than general blood-letting by venesection or arteriotomy. Indeed, the chief indications for bleeding are to be found where it serves a depleting purpose—e.g. in engorgement of

the right heart and venous system as a result of disease of the heart itself, or from disease of the lungs or from impaired respiratory movement; also in visceral congestion and arterial turgescence (*see* BLOOD, Abstraction of). It is seldom, however, that abstraction of blood needs to be carried to such an extent as to exert a marked depleting effect on the whole system; it is required, rather, for the relief of limited vaso-motor disturbances.

2. **Local Bleeding.**—Bleeding by means of leeches, scarification, or cupping, may serve a very valuable depleting purpose in limited congestions, as in those of the pericardium, pleura, peritoneum, lungs, eyes, kidneys, tongue, uterus, and hæmorrhoidal vessels.

3. **Purgation.**—For general depleting purposes free purging by means of hydragogue cathartics is a most efficacious agent. When it is required to give relief to turgid vessels without abstracting blood in cerebral, cardiac, and hepatic congestions, nothing depletes so readily, efficiently, and safely as watery purges. The best purgatives are elaterin, jalap, senna, scammony, sulphate of magnesium or sodium, tartarated soda, acid tartrate of potassium, croton oil, and gamboge; to which list may be added mercury, antimony, and resin of podophyllin. The great advantage of hydragogue purges is that they deplete by removing serum from the blood-vessels without the loss of red corpuscles. Depletion by means of such purges is of cardinal service in cerebral congestion. In congestion of the intestinal tract arising from hepatic, cardiac, or pulmonary disease, a mercurial followed in a few hours by a saline purge is of great service. Antimony has been mentioned in the above list because of its usefulness in combination with sulphate of magnesium and other saline purges. As it also acts as a powerful circulatory depressant, we should remember that it can be only employed when such action is permissible.

4. **Vomiting.**—Antimony, when used as an emetic, has a powerfully depressant, as well as evacuant or depletory effect; but depression and depletion are not synonymous. The lowering effects of free purging are pronounced, and, ordinarily, sufficient. Emetics may act as depletants by evacuating the contents of, or producing a flux from, the stomach and intestine; but when used with this object their depressant action is always to be borne in mind.

5. **Sweating.**—Sweating is a less effectual method of depletion, but if freely induced may, as in cases of renal disease, serve a good purpose in lowering arterial tension and assisting the elimination of retained materials. The usual modes of exciting diaphoresis are active exercise, the hot air (or Turkish) bath, the vapour bath, wet packing, nitrate of pilocarpine (*Jaborandi*), antimony, Dover's powder, and spiritus ætheris nitrosi,

or a judicious combination of these measures. Copious draughts of hot liquids often aid the action of diaphoretic remedies. The evacuant action of sweating and purging may be advantageously combined when speedy depletion is desired. The combination is valuable in certain dropsies. *See* DROPSY.

6. **Abstinence.**—General depletion may be produced by abstinence from food and drink, and this method is sometimes employed in the treatment of aneurysm.

ALFRED WILTSHIRE. JOHN HAROLD.

**DEPOSITS.**—The term 'deposit' had at one time a much wider extension in pathology than it has now. In accordance with the doctrine of morbid *crases* or *dyscrasia*, it was customary to regard cancers and all new-growths, the products of tubercular and scrofulous diseases, as well as those of the specific fevers and ordinary inflammation—in fact, almost every kind of morbid product in the body—as 'deposited' from the blood in consequence of some alteration in its composition (*see* BLOOD-DISEASE). In many of these cases the term *deposit* is still often used, even though an entirely different view may be taken of the processes leading to these morbid changes. The name remains, though the idea has departed; and this is also true of other morbid products called 'deposits.' The term *secondary deposits* was formerly used for what are now called pyæmic or secondary abscesses, from a belief that pus was removed from the original seat of disease, and deposited in distant parts. The term *atheromatous deposit* is due to Rokitansky, who described this change as due to the deposition upon the arterial wall of material precipitated from the blood. In both these cases the term seems now misleading. Again, several processes which we now call degenerations or infiltrations were formerly spoken of as deposits; for example, lardaceous, fatty, and pigmentary deposits; and although these processes may now be better described by another name, it cannot be doubted that in some of them an extraneous substance is actually deposited in the tissues. The term *fibrinous deposits* was also used for the masses now known as infarctions or blocks (as for example in the spleen or kidneys), where the appearance of a mass of extraneous material is produced by the degeneration as a mass of tissue, mixed with products of hæmorrhage, inflammation, and exudation. In quite another sense the various substances precipitated from urine are spoken of as *urinary deposits*, with which we are not here concerned.

When these exceptions are made, the present use of the word *deposit* is a limited one, but is appropriate in those cases where something different from the elements of the tissue, and especially if it be an inorganic material, is found in their substance, and

when it may reasonably be supposed that this material has been brought to the tissues by the circulating fluid and there deposited. The term is clearly inappropriate when the foreign matter is formed by some chemical change in the substance of the tissue-elements; but between such cases and those just mentioned it is very difficult to draw the line. We shall speak, in the sense just defined, of *calcareous*, *metallic*, and *pigmentary* deposits.

**1. Calcareous Deposits.**—All calcification of the tissues of the body, whether normal or degenerated, of inflammatory products, of new-growths, or of any other morbid products, appears to depend upon the deposition of lime-salts in the form of granules, this deposition taking place either within the substance of the elements, or more rarely in the intercellular substance, or, possibly, sometimes in the interstices of the tissue. Calcareous deposit takes place towards the close of life in several parts of the body, as in the walls of arteries, in cartilages and tendons, in the valves of the heart, and the crystalline lens of the eye; though in most of these cases, if not in all, some other pathological change precedes the deposition of lime. Still more frequently this change occurs in masses of dead or degenerated materials, as, for instance, in organs which have partially or wholly lost their vitality, in new-growths which have reached the stage of retrogressive change, in old blood-clots, or in products of bygone inflammation. A wasted eyeball always becomes partly calcified (sometimes ossified); tumours, such as myoma and fibroma, are especially subject to calcification; venous clots which remain long undisturbed, as in the deep veins of the pelvis, undergo the same change, and become phlebolites. Old inflammatory products, as pus and lymph, seem to become necessarily calcified if they are not absorbed; so do especially the products of chronic degenerative inflammations which have become necrotic, as seen in scrofulous lymphatic glands, and in tubercular masses in the lungs. A similar explanation applies to calcareous deposit in the walls of arteries, where the lime is generally deposited in new products which result from chronic arteritis or the atheromatous process, though lime may also be deposited in the muscular walls independently of atheroma. Necrotic masses resulting from embolic infarction are frequently calcified. Parasites of all kinds, occurring in solid organs, are liable to become surrounded by a calcified wall; a change frequently seen in hydatid cysts.

From all this it appears that calcareous deposit rarely occurs in normal healthy tissues, but is common in such parts as are dead or of deficient vitality. Its deposition must be attributed to some chemical reaction between the tissues thus altered and the lime-

salts in the blood; and it is possible that the presence of an excessive quantity of lime-salts in the blood sometimes favours the change, since deposition of lime in one part sometimes coincides with removal of it from another part. In senile decay the wasting of bones goes on simultaneously with the calcifying processes just mentioned, and in some rare cases rapid absorption of bone from special disease has appeared to be the determining cause of its deposit elsewhere by a sort of *calcareous metastasis*.

**2. Metallic Deposits.**—Other minerals besides lime-salts are rarely found deposited in the tissues, although in cases of chronic metallic poisoning, compounds of lead, silver, and copper may be found thus deposited. Zinc and mercury are less clearly traced, but probably follow the same law. The state or chemical combination in which the metals occur is not positively known, but appears to be some combination with albumen.

**3. Pigmentary Deposits.**—Pigmentation as a process is discussed in the article **DEGENERATION**, and has been shown to depend very frequently upon the occurrence of hæmorrhage and transformation of the extravasated blood. But physiological pigment, or melanin, is deposited in many parts of the body, both normally and pathologically, quite independently of hæmorrhage. Normally this is seen in the skin, the choroid coat of the eye, &c.: pathologically in the same situations, but in excessive quantity; and also in abnormal situations, as on the mucous membrane of the mouth. The arrangement of the pigment is in every case the same, forming minute black granules in the protoplasm of the cells around the nucleus. Its deposition and removal are regulated by causes as yet very imperfectly known, but are probably in some way dependent upon the nervous system.

The deposit of such substances as fat, lardaceous material, colloid, &c., is not a simple process, but depends either upon chemical metamorphosis of the cell, or on general pathological changes, which are dealt with in other parts of this work.

J. F. PAYNE.

**DERBYSHIRE NECK.**—A synonym for goitre, which is thus called from the prevalence of the disease in that county. *See* **GOÏTRE**.

### DERIVATIVES (*derivo*, I drain).

**DEFINITION.**—Medical appliances or remedies which lessen a morbid process, such as inflammation, in one part of the body, by producing a flow of blood or lymph to another part.

**ENUMERATION.**—Derivatives include Local Bleeding, Cupping, Leeches, Blisters, Sinapisms, and Setons.

**ACTION.**—The name 'derivative' was applied in ancient times under the belief that

diseases were caused by morbid humours, which might be drawn away from the part which they were affecting. It is now used chiefly to signify the diminution of blood in an inflamed part, by increased circulation in some other vascular district, either adjoining or remote from it.

T. LAUDER BRUNTON.

**DERMATALGIA** (*δέρμα*, the skin; and *άλγος*, pain).—Pain and aching in the skin. See NEURALGIA.

**DERMATITIS** (*δέρμα*, the skin).—Inflammation of the skin: a term applicable to every variety of inflammation of the integument, but especially to an acute inflammation attended with exfoliation of the cuticle and copious desquamation—for example, *dermatitis exfoliativa*, the  *pityriasis rubra*  of Devergie.

**DERMATOLYSIS** (*δέρμα*, the skin; and *λύσις*, a loosening).—Looseness or relaxation of the skin. See MOLLUSCUM.

**DESQUAMATION** (*de*, signifying separation; and *squama*, a scale).—The process of separation or shedding of the epithelium of any surface. It is of most importance in connexion with the skin in scarlatina, where the epidermis usually desquamates extensively.

**DESQUAMATIVE NEPHRITIS**.—A synonym for certain forms of Bright's disease, applied on account of their being characterised by shedding of the epithelium lining the tubules. See BRIGHT'S DISEASE.

**DETERGENTS** (*detergo*, I cleanse).

**DEFINITION**.—Substances which cleanse the skin.

**ENUMERATION**.—The principal detergents are—Water, Soap, Alkalis, Ox-gall, Milk, Vinegar, Alcohol, Charcoal, Sand, Oatmeal, Sawdust, Pumice-stone, Oil, and Borax.

**USES**.—Detergents are used to remove either extraneous dirt adherent to the skin, or epidermal scales which have accumulated upon it and interfere with its function. The chief detergent is warm water, but its action is greatly aided by such substances as soap, alkalis, borax, or vinegar, which act chemically in the removal of dirt or epidermis; or by such substances as oatmeal, sawdust, charcoal, pumice-stone, and sand, which act mechanically. Oil, or alcohol in the form of *eau de Cologne*, removes the resinous deposit left on the skin by plasters. Where the skin is tender, as in the case of the scalp, and where at the same time the detergent employed cannot very readily be removed, borax with elderflower water may be found preferable to the more irritating soaps as a means of removing scurf.

T. LAUDER BRUNTON.

### DETERMINATION OF BLOOD.—

Increased flow of blood to a part or organ: synonymous with *active hyperæmia* or *active congestion*. See CIRCULATION, Disorders of.

**DEVELOPMENT, Arrest of**.—The causes of arrest of development are in most cases still very doubtful, and for the most important theories and observations on the subject the reader is referred to the article on MALFORMATIONS. Such arrests may take place at any stage in the development of the embryo and of its organs; but only the most important of them, and the mode in which some typical examples are brought about, will be mentioned here.

**VARIETIES**.—Those which occur very early in fetal life are complex and, for the most part, incompatible with viability; whilst those which occur later often affect only one organ or a set of organs, and in some cases form no barrier to a prolonged existence. Not only must the fetus be considered, but also the placenta and membranes in which it is enclosed *in utero*; for diseases of these lead to many forms of monstrosity, either by interference with the nutrition and respiration of the embryo, as in the case of many so-called 'true moles'; or the normal changes may be checked by adhesions between them and the fetus. Persistence of the umbilical vesicle is excessively rare, but a patent vitelline duct is not uncommon, and explains many of the diverticula in connexion with the small intestines. A want of closure of the visceral laminae is the source of many deformities, from a simple fissure in the sternum, or a ventral hernia, to a complete anterior cleft, with the thoracic and abdominal viscera lying bare out of the body-cavity. In other cases the skin and muscles only may cover the viscera, or the muscles may not be developed. The thorax is closed before the abdomen, so that ectopia of the abdominal is more common than of the thoracic viscera. The abdomen, however, is sometimes closed in whilst the thorax remains open, and varying degrees of ectopia cordis result; but this is rarely complete. Epispadias and hypospadias to varying extents are further examples of imperfect fusion of the ventral laminae. These may or may not be attended with displacement or deficiency of the urinary and genital organs. Similarly, from an incomplete fusion of the vertebral laminae, the various forms of spina bifida occur; and these are generally accompanied with an excess of fluid in the spinal canal, or hydrorachis.

The arrests in the development of the cerebro-spinal centres and of the organs of special sense are very numerous. The whole brain may be wanting, or the medulla oblongata may be developed and the remaining portions missing, or any part of it may be absent or quite rudimentary. From

incomplete fusion of the bones of the skull hernia cerebri or encephalocele occurs, and this is generally complicated with hydrocephalus, though the latter is frequently found as an independent and solitary affection. Coloboma and deficiency of the olfactory, optic, or auditory nerves are well-known examples of arrested development of the organs of special sense. The special malformations of the heart and vascular system, the digestive, respiratory, and urino-genital systems, will be found described under the various organs; but reference must here be made to congenital fissures leading into the pharynx (persistent branchial clefts), to the various fissures of the face and palate, to persistent cloacæ, to the numerous forms of hermaphroditism, and to imperforate anus, as all coming under this head. Varying degrees of ill-developed extremities are common, from a diminution of number in the fingers or toes, or their coalescence, to a complete absence of one or all the limbs. Finally, ducts, vessels, and openings—such as the urachus, ductus arteriosus, umbilical vessels, and foramen ovale—which normally close soon after birth, may remain patent throughout life.

JOHN CURNOW.

**DEVONSHIRE COLIC.**—A synonym for lead colic, which has arisen from the frequency of lead-poisoning in that county, supposed to be due to the contamination of cider with lead. See LEAD, Poisoning by.

**DIABETES INSIPIDUS** (*διαβαίνω*, I walk with the legs apart; *διαβήτης*, a thing with outstretched legs, a pair of compasses, a siphon—whence the idea of free flow is derived; *insipidus*, tasteless).—SYNON.: Polyuria; Fr. *Diabète insipide*; *Diabète non sucré*; Ger. *Polyurie*. A synonym for polyuria. See POLYURIA.

**DIABETES MELLITUS** (*διαβήτης*; and *μέλι*, honey).—SYNON.: Glycosuria; Fr. *Diabète*; Ger. *Harnzuckerruhr*.

**DEFINITION.**—The term Diabetes, meaning an excessive flow of something, has been applied to the pathological condition indicated by an excessive flow of urine. It has, moreover, been almost wholly limited to the kind of malady characterised by the presence of a notable quantity of sugar in the urine—a condition more strictly described by the term Diabetes Mellitus. Another condition is sometimes seen where no sugar is to be found in the urine, which is, however, excessive in quantity. This is designated Diabetes Insipidus, or (better) Polyuria.

Diabetes is a malady more or less chronic, characterised by the persistent presence of a notable quantity of sugar in the urine, which is in most cases markedly abundant. It is accompanied by thirst, hunger, and bodily wasting. If unrelieved, it invariably tends to death. The ordinary form of diabetes is thus

to be distinguished from certain other conditions, where, for instance, a small, or hardly perceptible trace of sugar may be detected in the urine; or where, yet again, considerable quantities of this substance may be detected occasionally, and for a short time only. To this abnormal condition the term *glycosuria*, which is often also employed to describe artificial diabetes, best applies.

**GENERAL CONSIDERATIONS.**—Three important facts lie at the bottom of our knowledge of the pathology of diabetes. These are:—

I. That grape sugar is found in the healthy human body.

II. That glycogen, a substance closely allied in chemical composition to grape sugar, is also found in the healthy human body.

III. That both of these may be formed in the healthy human body.

Beyond these, certain other fairly definite propositions may be made:—

1. Glycogen is found most abundantly in the liver, inasmuch that, with due precautions, it can always be detected there: after a certain time sugar takes the place of glycogen, but the exact mode and time of this conversion are not known.

2. Nevertheless, it is fairly certain that the sugar called glucose can always be detected in the liver; still more certain that it is to be found in the blood; but

3. This sugar never appears in any notable quantity during a state of health in the urine.

4. As sugar is not to be found in any appreciable quantity in any other of the excretions, it follows:

5. That this sugar must disappear in the body.

6. It is commonly asserted, and, upon the whole, believed, that sugar is less plentiful in venous than in arterial blood.

7. From this, if true, it follows that sugar must be used up in the course of the circulation.

8. Where the combustion, or oxidation, occurs is not quite clear.

9. But it is plain that, from a fault in either direction, sugar may become over-abundant in the blood, namely:—

(a) *By over-production, or*

(b) *By diminished consumption.*

10. The over-production and the diminished consumption of sugar in the body may depend on various causes. The most notable of these are (a) an increased ingestion of saccharine material into the stomach and bowels, without a corresponding destruction; and (b) such an alteration of nerve-influence as will completely modify the relative proportions of the sugar produced and the sugar destroyed.

11. With an excess of sugar in the blood, only one easy road of egress from the body is available, that is, by way of the kidneys; but this is not a sufficient outlet when there is great superabundance in the blood. Sugar

may then be found in almost every one of the secretions or excretions.

12. With this unnatural discharge of sugar there is usually a corresponding discharge of urine, but not always.

13. Thus there may be no greatly increased flow of urine, yet the urine may be rich in sugar.

14. And yet, again, there may be a copious flow of urine without any sugar, as in *polyuria*.

15. Hence the over-production or the diminished consumption of sugar in the system has no necessary connexion with increased flow of urine.

16. Both the abnormal action of the liver and that of the kidneys seem in the main to depend on similar but not identical causes.

17. Both seem to be under the control of the sympathetic, but the special fibres are not the same as regards the two organs.

(a) In the case of the liver, the fibres seem to originate in the medulla oblongata, to descend in the spinal cord to the lower cervical or upper dorsal vertebræ, thence to leave the cord to join the gangliated sympathetic, and so ultimately to reach the liver.

(b) In the case of the kidneys, the active fibres proceed further down the spinal cord, but are ultimately connected with the great abdominal plexus, for such it may well be called, whence the fibres proceed to the kidneys.

**ÆTIOLOGY.**—The classification of cases of diabetes according to causation is in very many cases practically impossible.

As to the circumstances that call the morbid processes into play, we know very little. It is certain, however, that the disease is much more frequent among men than among women, and among the middle-aged than among the very young or the very old. The disease is very much more fatal in young adults than in those over, say, forty-five. That the disease is more rife in certain districts than in others may probably be best explained by its undoubted tendency to heredity. This heredity, as in many other maladies, is peculiar; the diabetic tendency in one branch of a family being represented in another branch by various nervous disorders, especially epilepsy and imbecility.

Of the so-called *exciting* causes there are two of the first rank, namely, injury or disease of the brain; and mental excitement, or, perhaps still more, worry. Tumours and other local brain-mischiefs sometimes give rise to a fatal diabetes. Certain mental emotions, at once powerful and prolonged, which may be epitomised in the single word 'strain,' apparently act as exciting causes of diabetes, such as continuous anxiety, long-lasting grief, or excitement followed by reaction. Certain errors of diet—such as excessive use of hydrocarbons, especially sugar, or other interference with the laws of health, may originate a fatal diabetes, especially in those who have

any hereditary tendency to the disease. It is possible that the frequency of the malady among the Jews (as noted by Seegen) may be accounted for on dietetic grounds.

**ANATOMICAL CHARACTERS.**—Many pathological conditions have been recorded as occurring in those who had been the subjects of diabetes, but we know little of its real pathology. In many cases nothing which can be directly connected with the diabetes has been found *post mortem*. Latterly attention has been mainly directed to the investigation of certain parts of the nervous system and of the liver itself. Perhaps the most reliable data for the ordinary anatomical appearances are to be found in Seegen's analysis of Rokitansky's experience, embracing 30 necropsies.

Connected with the *brain and spinal cord* various lesions have been found, such as tumours of different kinds pressing on the medulla, and softening, with or without the marks of extravasated blood. In some cases extravasation has been the only morbid change discovered. In two cases under the writer's care there were extravasations of blood in the spinal canal in the cervical and upper dorsal regions, and the same was observed in another case under the care of a colleague. In one of these there was very marked softening of the cord in the regions named. In these three cases death took place suddenly. Dr. Dickinson's theory of the origin of diabetes in lesions of the nervous system, represented by enlarged perivascular spaces, the sites of existent or pre-existent extravasations of blood with destruction of the surrounding nerve-tissue, is hardly tenable. The exploration of the sympathetic system has not been more satisfactory.

Rokitansky found (in 15 out of 30 cases) that the *liver* was enlarged, hyperæmic, and hard, of a dark-brown colour, with its acini imperfectly defined. The same conditions were present in the cases examined by the writer. When the disease has lasted a long time, the liver may be smaller than natural. The same would appear to hold good with regard to the cells themselves. Early in the disease, the cells, especially in the outer portions of the acini, are large, plump, and rounded, instead of angular, with large and distinct nuclei. They tend to assume a wine-red colour with solution of iodine, from the presence of unchanged glycogen. There are also sometimes found signs of active cell-growth at this early stage. Later the cells seem smaller, and as if undergoing pigmentary degeneration.

The condition of the *pancreas* in diabetes is highly interesting. In 13 out of Rokitansky's 30 cases, it was strikingly small, hard, and bloodless; and in many cases it has been found so shrunken and altered as to be hardly recognisable save by its connexions. Such was the case in one instance the writer has

seen, and in all his other cases the pancreas has been abnormal, usually contracted here and there, hard and knotty. In one case, however, the organ was enlarged and more succulent than usual, probably the first stage of the mischief. The occurrence of so important pancreatic changes in about one-half the cases of diabetes would seem to indicate more than a casual connexion with the disease. What this connexion really is remains unascertained. Klebs has associated the changes with disease of the celiac plexus. One result from the pancreatic mischief is inability to digest fat. This was seen in one of the cases referred to above. See PANCREAS, Diseases of.

The *kidneys* Rokitansky found diseased in 20 instances, but the changes were not uniform. Usually they presented the ordinary indications of hyperæmia, being enlarged, dark red, and full of blood. Occasionally there were signs of more extensive mischief, the substance of the kidney as well as its vessels and epithelium being involved, and the organ harder than natural. Often fatty changes occur. In one case of the writer's amyloid changes had begun in the Malpighian bodies.

The *lungs* are frequently diseased: in only 7 of his cases did Rokitansky find them normal; Dr. Dickinson only twice out of 27 instances. The changes included all stages, from acute or chronic pneumonia, to the formation of numerous cavities, or even to gangrene. In one of the writer's cases the whole of the upper lobe of one lung was converted into a huge cavity filled with solid and semifluid detritus, having no gangrenous odour. There had been no expectoration, and no hæmorrhage, though vessels were exposed.

The *stomach and intestinal canal* present little beyond the ordinary signs of recurrent or chronic catarrh—thickening, mammillation, and slaty pigmentation; erosions and ulcerations may, however, occur.

**SYMPTOMS.**—The following sketch comprehends the more characteristic clinical features of diabetes; but there are often important variations in individual cases, though a certain number of features are common to all: The patient, most likely a male between twenty and forty-five, when he comes before the physician has in all probability been suffering from the disease for some time; for it usually comes on insidiously. He may say that he has been in failing health for a varying period; that he has been very thirsty, and has passed much water, having to get up repeatedly for this purpose during the night. His appetite has been more than hearty; but his food seems to do him no good, for he has been constantly growing thinner, and he feels weak and ill. On closer examination it is found that he is daily passing as much perhaps as eight, ten, or more pints of urine, light in colour and of a peculiar sweetish

odour, of a high specific gravity, perhaps 1040°, and containing an abundance of sugar. The skin is dry and harsh; the tongue red and glazed or slightly furred; the mouth dry and clammy; the lips, teeth, and gums are covered with scanty, sticky mucus; the breath is often sweetish, or it may be unpleasant from the state of the mouth; the bowels are confined; and the countenance wears an expression of weariness and fatigue.

From this point the malady may progress in one or other of two directions. Under judicious management the symptoms may ameliorate. Often the first indication of improvement is a copious perspiration; the thirst diminishes; less urine is passed; the appetite is not so ravenous; the sugar decreases in quantity; and with it the specific gravity is lessened. Emaciation ceases, and the patient begins to regain weight. This auspicious commencement may, with time and care, end in a more or less complete return to health. Unfortunately there is another side to the picture, for, notwithstanding all our efforts, the patient often goes from bad to worse. We fail to reduce the quantity of sugar beyond a certain point. The appetite gets more and more voracious, especially for starchy articles of food, to obtain which the patient will sometimes lie or steal, and yet there is often no feeling of satiety. Sometimes the appetite fails, and then emaciation goes on still more rapidly. All sexual power and feeling have long ago been lost, the testes sometimes undergoing almost complete atrophy. The harsh, dry, and itchy skin becomes the seat of boils, or even of carbuncles. Often the sight is injured by cloudiness of the refractive media, especially of the lens, or by retinitis and other morbid changes. All this time the temperature is low, perhaps sub-normal; but towards the later stages of the malady it often rises. Such a rise indicates the accession of a formidable complication which might almost be said to be the natural termination of diabetes. This superadded mischief is a peculiar insidious kind of pneumonia, resembling acute pneumonic phthisis, and giving rise to local signs resembling those characteristic of that malady. The progress of this lung-mischief is rapid; the fever increases; and often there is irrepressible diarrhœa, sometimes of fatty-looking matter. As the end approaches, the sugar usually disappears from the urine, which may become albuminous and scanty. There may even be some œdema of the extremities. The end often comes swiftly, and without warning, by acute pneumonia, or by what, for want of a better name, we call diabetic coma, or more slowly by gradual exhaustion. After the onset of pulmonary symptoms it is never very long delayed. Such may be said to be the ordinary course of a well-marked case of diabetes. There are cases slighter, where the

history is different; but in both sets of cases there are certain symptoms, which demand further consideration than has just been given them.

*The Urine.*—The characteristic of the urine in diabetes is the presence in it of sugar in notable quantity, though this varies greatly in different cases. In the earliest and slightest forms of diabetes, small quantities of sugar may be passed now and again, as after a meal or the consumption of an unusual quantity of starchy or saccharine food; but the sugar may completely disappear in the interval, or may do so finally by the use of an animal diet. The sugar thus excreted is glucose of the kind called dextrose, from turning polarised light to the right, is readily soluble in water and alcohol, and easily ferments. When diabetic urine is allowed to stand in a warm place, fermentation soon sets in, gas being disengaged, and yeast deposited at the bottom of the vessel. The proportion of sugar to urine is usually from 8 to 12 per cent., but varies; the total quantity passed amounting to 20 or 25 ounces, or even more. Its presence in such proportion causes an increased specific gravity, though this is not invariably the case, inasmuch as it may vary from 1008 to 1060 or 1070. Along with the presence of sugar, we almost invariably find an increase in the quantity of the urine. But neither is this an invariable feature of diabetes, for sugar may exist with a normal quantity of urine, constituting the so-called *diabetes decipiens*. But in most cases there is a marked increase, the quantity passed amounting to eight, ten, or fifteen pints daily; and even the highest of these rates has been greatly exceeded. With an excessive amount of urine, its colour becomes lighter; sometimes it has a faint greenish tint, and when passed is quite clear. On standing there is no ordinary sediment, though, as already said, sporules of yeast may be deposited after a time. There is, on the whole, an excess—sometimes a large excess—of urea; and if the quantity of urine passed be small, deposits of uric acid or of the amorphous urates may appear. Very great inconvenience may arise in diabetic females, or even in males, by the arrest of saccharine urine about the external genitals, producing a raw or eczematous condition of the inside of the thighs and groins. The urine, being also almost invariably acid, is highly irritating to the raw and swollen parts. Enuresis is common, especially at night, and among diabetic children. Albumen makes its appearance sometimes in the progress of the disease, or in its later stages.

*The Digestive Organs.*—As the correlative of the unusual flow of urine we have also, as a marked and early symptom of diabetes, extreme thirst, a thirst, too, which cannot be satisfied, for apparently the more the patient drinks the greater is the thirst. This sensa-

tion is in some part due to the dry and clammy condition of the mouth, which it is difficult to relieve. Hunger, or even a voracious desire for food, is usually a prominent feature in diabetes, but is not invariably present, and in the later stages there may be complete loss of appetite, amounting to a loathing, especially for a restricted diet. It is often impossible to satisfy the intense craving for food, and should satiety be attained the sensation lasts but for a little time. The mouth is usually dry and parched, the saliva being scanty and tenacious. Often particles of food are retained about the teeth, and there putrefy, giving rise to unpleasant odours. The buccal fluid is almost always acid instead of alkaline, probably from the formation of lactic acid. The tongue is rarely perfectly natural. Seegen describes it as usually thickened and increased in volume, with fissures and glazed blood-red islands on its surface, which, however, may present a general coating. The teeth often fall out without pain, from the retraction of the gums, and are singularly liable to caries. Digestion is usually good, except during catarrh of the stomach, which is a rather frequent condition. Constipation is the rule in diabetes, often to a troublesome extent. This arises partly from the deficiency of water in the bowel; partly also from the small quantity of faecal residue from an exclusively meat diet. Diarrhoea, on the other hand, is not uncommon, and rapidly diminishes the patient's strength.

*General Symptoms.*—Emaciation is an early and marked symptom of diabetes, but not invariably so, for diabetes often occurs and persists in stout persons, without removing the obesity. These are cases of the more tractable kind, the patients being usually somewhat advanced in life. Their complete cure is seldom effected, but they do not seem to suffer greatly from the malady. Though the emaciation is in great part due to the removal of fat, and in part to the abstraction of water from the tissues, there seems to be also an actual waste of muscular substance, especially in the advanced stages of the disease. With this emaciation are associated weakness, weariness, and disinclination to exertion. These are often among the earliest symptoms of the disease, and may occur long before wasting is noticed, but increase markedly towards the close of the malady. From various causes (one being, doubtless, weakening of the heart), oedema of the lower extremities may occur, with or without albuminuria. Gangrene of the extremities, of the senile kind, has been observed.

*Respiratory Apparatus.*—With regard to the respiratory organs, a peculiar apple- or hay-like odour of the breath is sometimes observed, probably arising from the production of acetone mixed with alcohol. But the most

serious pulmonary symptoms are those of phthisis, resulting from a more or less chronic pneumonia. This mischief is usually indicated by a nightly rise in the temperature, but otherwise may remain for a time almost latent. It is most common in the young, and towards the end of the disease. The expectorated matters may contain sugar. Gangrene of the lung, or a form of necrosis of the lung-tissues, has been noticed. With this form of gangrene the sputum may be odourless.

*Skin.*—The skin is usually dry and scurfy, often extremely itchy; wounds inflicted by scratching heal with difficulty. Copious saccharine sweat is observed in a certain number of instances. There is a marked tendency to the formation of boils and carbuncles. Boils often occur early in the disease, and may give the clue to the existence of diabetes. Carbuncles occurring late in the disease may be the immediate cause of death.

*Nervous System and Special Senses.*—The sight is often affected in diabetes, most frequently by the formation of diabetic cataract. Operations in such cases do badly till the disease is cured, and are seldom tried, for the cataract occurs late in the disease, and advances rapidly. A form of retinitis not unlike that of albuminuria is sometimes found. Other forms of imperfect vision of uncertain origin occur in diabetes. They go by the general term of diabetic amblyopia.

Along with the physical, the mental powers fail, and the moral sentiments become blunted, which, to the friends of the patient, is not the least distressing feature of the malady.

*Genital Organs.*—Early in the history of the disease, all sexual appetite disappears, and sexual power soon fails in the male—but with improvement this may return. In the advanced stages amenorrhœa is not unusual amongst females.

**COMPLICATIONS.**—Several of the symptoms just described are regarded by some authorities as complications of diabetes rather than as belonging essentially to the disease. Such are especially diabetic cataract and amblyopia, boils and carbuncles, and the chronic pneumonia or phthisis in which diabetes so frequently ends.

Amongst the intercurrent diseases that are specially to be watched for and seriously regarded, one of the most important is albuminuria, the appearance of which may encourage a false prognosis, from the fall in specific gravity of the urine that attends it.

*Diabetic Coma.*—This is a not infrequent terminal symptom of diabetes. It is more liable to occur in the early stages of severe cases in young persons. It is especially noted after muscular exertion, and after the first adoption of an anti-diabetic diet. Constipation predisposes to its development. Cases of diabetic coma are divided into three groups. In the first group the attack begins with headache or epigastric pain and nausea, followed

by a great feeling of anxiety, or by restlessness and perhaps delirium, after which a peculiar dyspnoea sets in. The dyspnoea affects both inspiration and expiration, and no physical cause can be found for it on examination of the chest, and there is usually no cyanosis. Comatose symptoms then come on, which gradually deepen. The pulse is much quickened, and the temperature is abnormal. The coma generally ends fatally within forty-eight hours, and only a few cases have been known to recover. The symptoms of the second group closely resemble those of renal uræmia. The dyspnoea is not so well marked as in the preceding form. The patient complains of general weakness, becomes drowsy, lapses into a state of coma with low temperature and small pulse, and soon dies. The third form exhibits all the symptoms of acute alcoholic intoxication. The patient does not show the same muscular weakness as is observed in the two preceding forms. He is attacked suddenly by great excitement, staggering gait, and drunken delirium. Gradually drowsiness and coma develop as in the other forms, and death ensues. In all these forms, and during and preceding the attack, a peculiar sweet smell, resembling chloroform, is perceived in the breath. The urine has a similar odour; and it gives the so-called 'acetone reaction' with perchloride of iron—that is, when a solution of perchloride of iron is added in excess a deep red port-wine colour is produced.

The pathology of diabetic coma has been much debated. Kussmaul believed that the symptoms were due to the presence of acetone in the blood; but it has been shown that other substances besides acetone give the peculiar reaction with perchloride of iron. The blood of diabetics has been often observed to contain excessive quantities of fat. Drs. Sanders and Hamilton described a case of diabetic coma, in which much fat was found in the blood, and numerous fat-emboli were seen in the pulmonary capillaries; and they suggested that fat-emboli in the lungs and brain were the cause of the symptoms. Similar cases have been described by others. Drs. Dreschfeld and Taylor, however, were unable to find fat-emboli in seven cases examined by them, although the blood was exceedingly rich in fat.

**DIAGNOSIS.**—The diagnosis of diabetes depends on the discovery of sugar, in notable quantity, in the urine of the patient. But before the investigation for sugar is undertaken, there have usually been observed by the patient some of the early indications of diabetes, of which the following are the most frequent, though no one definite symptom invariably heralds the disease: (1) dryness of the mouth and thirst; (2) bodily weakness and gradual emaciation; (3) dryness of skin, with itching and a tendency to successive crops of boils; (4) urination in increased quantity, the urine being of a greenish-yellow tint, with the odour

described; (5) defects of vision. Occasionally the attention of the patient is drawn to the state of his urine by the attraction it presents to ants, flies, and other insects; or by the formation of white spots of sugar on his dress and boots left by the urine on evaporation.

We must not forget that in true diabetes we find sugar in notable quantity. There are often present in the urine other reducing agents (such as uric acid and colouring matters), so that the amount of sugar detected must be such as to admit of no doubt whatever. Again, the presence of the sugar must be persistent, hence a single examination will not suffice for diagnosis unless sugar be found in large quantity. It may so happen that the patient when seen is not passing sugar, from the influence of restricted diet, or from some other cause, such as pneumonia or other feverish condition. It is then best to examine the urine passed an hour or two after a meal, or even to permit the use of starchy food for a day or two, so that the constitutional proclivities of the patient may be the better ascertained. To determine the existence of sugar is not enough: it is always necessary to determine the quantity passed, so as to obtain a clue to the intensity of the disease, and to judge of the effects of treatment.

**Qualitative Testing for Sugar.**—The presence of sugar in the urine may be detected in various ways. When saccharine urine is mixed with yeast and kept in a warm place, it speedily ferments, with production of alcohol and evolution of carbonic acid; and as no other substance is capable of undergoing this transformation, the occurrence of fermentation with yeast is certain proof of the presence of sugar. The signs of fermentation are evolution of carbonic acid and the lowering of the specific gravity. By the latter of these two signs considerably less than one per cent. of sugar can be detected in the urine. The method of carrying out this test is fully described under the head of quantitative testing.

**Moore's or Heller's test.**—When saccharine urine is boiled with liquor potassæ the sugar is decomposed, and a compound is formed, giving its colour, black or brown, to the fluid. A convenient test-tube is filled one-third full with the urine, and an equal quantity of liquor potassæ is added. The two should then be well mixed by shaking, and the heat of a spirit-lamp applied to the upper portion of the mixed fluids. If sugar be present this portion will gradually darken, the tint assumed varying in depth according to the quantity of sugar present. This test is very convenient, but it is liable to several objections, notably these:—(a) It is far from delicate, requiring as much as three parts in a thousand, or a grain and a half of sugar to the ounce, to afford any satisfactory indication. (b) It is practically useless for quantitative purposes, though the plan has been

tried of comparing the colour produced with the colours of solutions containing known quantities of sugar, as is done in the Nessler process for substances producing ammonia and its allies. (c) It is liable to two notable fallacies. (1) High-coloured urine is always darkened in tint, sometimes blackened, by boiling with liquor potassæ. (2) Liquor potassæ very often contains lead, which is liable to be converted into black sulphide when boiled with caustic potash, if albumen or any organic matter be present in the urine. The former of these risks cannot well be obviated; the latter may, by first testing the purity of the liquor potassæ, and keeping it in green glass instead of white glass bottles.

**Indigo-carmin test.**—If a solution of indigo-carmin be rendered alkaline by carbonate of sodium, and boiled with a small quantity of grape-sugar, the indigo-blue becomes reduced to indigo-white, and causes the blue solution to assume a yellow colour. This test, originally introduced by Mulder, has been recommended by Dr. George Oliver, who makes use of it in the convenient form of test-papers. Two test-papers, one saturated with indigo-carmin, and the other with carbonate of sodium, are digested in a small quantity of water, the result being a clear blue solution. To this one drop of the suspected urine is added, and the mixtures boiled; if sugar be present the blue colour will successively give place to reddish-violet, different shades of red, and finally to a pale yellow tint. On standing, the solution re-absorbs oxygen from the air, and gradually returns to its original blue.

**Picric acid test.**—This test was introduced by Dr. George Johnson. The principle of the method depends on the power of grape-sugar to change a yellow solution of picric acid, in the presence of caustic potash, to a red solution of picramic acid—and the depth of the red colour varies with the amount of sugar present. Dr. Johnson has also applied this method for quantitative determination. The test is, however, less delicate than that of Fehling, to be presently described.

**Trommer's test.**—Equal volumes of the suspected urine and liquor potassæ are mixed together in a test-tube with a few drops of a 10 per cent. solution of sulphate of copper. Heat is then applied; and, if sugar be present, when the boiling-point is reached an orange-coloured precipitate of suboxide of copper is produced. This mode of applying the copper-test is, however, far inferior to the following.

**Fehling's test or method.**—This testing fluid may be procured ready-made, but the following is the formula for it, as slightly modified by Dr. Pavy, and fitted for daily use:—Five grains of sulphate of copper, ten grains of neutral tartrate of potassium, and two drachms of liquor potassæ. A more exact

formula will be given farther on. The fluid thus formed is of an intense blue colour, clear and bright. When the test-fluid is to be used, a small quantity of it should first be raised to the boiling-point, because by prolonged keeping the tartaric acid undergoes change, a substance being formed from it which is capable of reducing copper, and might give rise to confusion. But if on boiling the test-fluid no copper is thrown down, the suspected urine should be added drop by drop, the mixed fluid being at the boiling-point. If sugar be present in quantity, it will throw down the copper in the form of a red or orange precipitate. The quantity of urine added must never exceed the bulk of the test-fluid, and the upper portion of the fluid should be heated, so as to contrast with the lower portion. Should suboxide of copper be thrown down when the test-fluid is boiled, the fluid must be filtered before adding the suspected urine, or, still better, a new fluid be prepared. To obviate as far as possible such inconveniences, the cupric and alkaline fluids should be kept in separate bottles until about to be used.

When the quantity of sugar in the urine is very small, as may occur in ordinary diabetes after long fasting or the use of a rigidly restricted diet, or during an access of fever, still more minute precautions must be taken. The most important of these is to use a great excess of the test. When the copper solution is added drop by drop to a healthy urine, at a boiling heat, the blue colour is immediately discharged, although not a particle of sugar be present, and the urine assumes a deep amber tint. The degree to which urines exercise this decolorising property over Fehling's test varies with their concentration. A dense urine (sugar free) will discharge the colour from nearly its own bulk of Fehling's standard solution; but even the most dilute urines have a considerable power this way. To avoid this fallacy, and secure an excess of the test, the most certain method is to heat the solution first, as already recommended, and to add the suspected urine afterwards—and never to add more, but always something less, than the bulk of the test-solution. The mixture is then again raised to the boiling-point. It then changes (if sugar be present) to an intense opaque yellowish-green, and slowly a bright yellow deposit subsides. If the urine contain less than half a grain per cent. the precipitation does not take place immediately, but occurs as the liquid cools—in five, ten, or twenty minutes—and the manner of the change is peculiar. First the mixture loses its transparency and passes from a clear olive-green to a milky opacity. This green milky appearance is quite characteristic of sugar. Some of the natural urinary ingredients have a certain power of reducing the oxide of copper to a state of suboxide, and of becoming thereby a source of fallacy in

using this test for the detection of sugar. But this fallacy is easily avoided by not prolonging the ebullition. The fluid should simply be raised to the boiling-point, and then allowed to slowly cool. If such a proceeding fail to yield an indication of sugar, no amount of boiling will develop a trustworthy reaction. An increased delicacy can be imparted to this method by the following process, first suggested by Seegen. The urine is repeatedly filtered through animal charcoal until it is completely colourless. A little pure water is then passed through the filter, and to this water the test is applied in the usual way. An exceedingly minute trace of sugar (0.01 per cent.) can be detected by this procedure.

**Quantitative Testing for Sugar.**—*Fehling's volumetric method.*—This method, now in common use, is founded on the fact that the proportion in which sugar reduces copper is constant. One equivalent of grape-sugar decomposes exactly ten of sulphate of copper, or 180 parts by weight of grape-sugar decompose 1246.8 parts by weight of sulphate of copper. This being borne in mind, and a copper solution of known strength being used, it is easy to determine the quantity of sugar in any given specimen of urine. The quantities may be calculated according to the metric system, or by grains and minims. If we adopt the metric system, our fluid will consist of the following ingredients:—

Sulphate of Copper (crystals) 40 grammes.  
Tartrate of Potassium (neutral) 160 grammes.

Liquor Sodæ (sp. gr. 1.12) 750 grammes.

Water to 1154.5 cubic centimetres.

These should be carefully mixed; or, what is better, the copper and alkaline solutions are made separately, so that five cubic centimetres of each, or ten of the mixed fluids, will exactly decompose .05 gramme or 50 milligrammes of sugar.

According to the English system of measurement, Dr. Pavy's solution is the most convenient. It consists of sulphate of copper, 320 grains, dissolved in ten ounces of distilled water; and tartrate of potassium (neutral), 640 grains, with caustic potash 1280 grains, also dissolved in ten ounces of distilled water. These fluids may be kept separate or mixed. In mixing, the copper should be added to the alkaline solution, not *vice versa*, to prevent the formation of any precipitate. One hundred minims of this mixed fluid are decomposed by half a grain of sugar. Only a minim measure and a porcelain capsule, or other vessel which will stand heat, are required for Dr. Pavy's solution in the procedure, which is as follows: Most specimens of diabetic urine, containing too much sugar for accurate testing, first require dilution with water; and the most convenient degree of dilution is when one-tenth of the fluid is urine. Next put ten cubic centimetres of the metric copper solution, or one hundred minims of Pavy's

solution, carefully measured, in a small porcelain capsule. The fluid being deep blue is better for dilution, so as to diminish the intensity of tint. Of course this does not alter the quantity of copper present in it. The porcelain capsule with its contents is to be placed on an iron retort-stand, at such a level that the flame of a spirit-lamp will easily play on the capsule. Meanwhile a pipette, graduated from above downwards, either in minims or cubic centimetres, is filled up exactly to the 0 in the graduated scale with the diluted urine. When the solution of copper is boiling, the urine is added to it from the pipette, drop by drop, stirring carefully the while, until signs are shown of a decoloration of the cupric solution. The moment all the copper has been thrown down as suboxide, and all shade of blue or green has disappeared, the addition of the diluted urine is stopped, and the quantity already used read off on the graduated pipette. To ascertain the quantity of sugar in the urine is now a simple calculation. We know how much urine has been employed in reducing the 10 cubic centimetres, or the 100 minims, of the cupric fluid, but these measures represent exactly 50 milligrammes and half a grain of sugar respectively. The quantity contained in the diluted urine being hence deduced, multiply this by ten, to get the quantity contained in the urine as passed. Next multiply by the total quantity of urine passed in twenty-four hours, to ascertain the full amount of sugar passed in this period. In all such analyses the sample examined should be taken from the mixed urine passed during the whole twenty-four hours. That passed after prolonged fasting is the richest, that passed three or four hours after a meal the poorest, in sugar.

*Roberts's fermentation method.*—We have already noticed the fermentation method for demonstrating the existence of sugar in urine. Sir William Roberts has also devised from it a highly accurate quantitative process. It is as follows:—

Put about four ounces of the suspected urine into a clean eight- or twelve-ounce glass bottle. Introduce a piece of dry German yeast, about the size of a cob-nut, but divided into small pieces. Cork the bottle with a grooved cork to allow the escape of gas. Fill a companion bottle *quite full* with the urine. Cork quite tightly, and set the two side by side for twenty-four hours in a warm place—the mantelpiece will do. By the end of that time fermentation will probably have ceased, and the yeast fallen to the bottom. The specific gravity of the two specimens must now be carefully taken with an accurate urinometer, that of the unfermented bottle being taken as the standard. The fermented urine will have lost density from two causes: First, the sugar which gave the increased specific gravity has been destroyed; and, second, in its place have been formed alcohol, which is lighter

than water, and carbonic acid, which has escaped. *Every degree of specific gravity thus lost represents a grain of sugar in the ounce of urine.* Thus, if there is a loss of twenty-five degrees of specific gravity, the urine would contain twenty-five grains of sugar in each ounce. Multiply this by the total number of ounces passed, to get the amount of sugar discharged *per diem*. This plan is especially useful for noting the quantity of sugar passed day by day, and can be easily undertaken by the patient or his friends.

*Estimation by the polariscope.*—A plan of estimating sugar employed a good deal abroad depends upon the power of diabetetic sugar to turn the plane of polarisation to the right. The degree of rotation is in proportion to the quantity of sugar contained in the urine. The apparatus used commonly goes by the name of the Ventske-Soleil apparatus, from its inventors. It is best adapted for light-coloured urines. If the urine be deep in colour it requires to be diluted.

**PROGNOSIS, COURSE, AND TERMINATIONS.**—The prognosis of confirmed diabetes is ever unfavourable. Amelioration is common, but a perfect cure, save in exceptional cases, is rare. Diabetes coming on suddenly and from special causes, such as injury to the head, is more likely to do well than when more gradually developed. The younger the patient, the more sugar passed, the greater the emaciation and debility, the worse is the prognosis. Diabetes persistent with pure flesh diet is worse than if persistent only with a free starchy or saccharine diet. Loss of appetite and of digestive power, as indicated by the condition of the fæces, is of evil omen. When albumen appears in the urine in the course of diabetes, the specific gravity of the fluid diminishes, often giving rise to a false impression to the effect that, as the specific gravity has decreased, the diabetetic state must be improved. The only true test of recovery is the power of consuming ordinary mixed food with impunity; but Seegen says that out of two hundred cases he had never seen this result. But by rigid or limited dieting, the patient may live, and even enjoy life, for many years. Heredity, directly or collaterally, adds to the darkness of the outlook. Cases resulting from injury to the brain or other parts of the nervous system are often the most hopeful with which we have to deal, though by no means always so.

The course of diabetes is on the whole chronic. Most frequently it develops gradually, though in some cases suddenly; and it generally lasts from six months to three or four years. In stout elderly persons it may exist much longer, especially with good digestion and a limited dietary. On the other hand, Sir William Roberts records a case which proved fatal, after a well-marked onset, in eight days—the shortest period known to the writer. Another case was fatal in three weeks,

the patient being three years old. Some cases are recorded of death at still earlier periods, but these were most probably cases of long duration in an unnoticed shape suddenly developed into an aggravated form. A fatal termination of diabetes may be brought about, as already indicated, in various ways. One is by diabetic coma, not unlike the first stages of uræmia. In the advanced stages of diabetes, the strength being very low, comparatively slight causes may produce fatal effects.

**TREATMENT.**—Though we are ignorant on many points as regards diabetes, yet when we come to its treatment, we have these undoubted facts—that sugar cannot be formed out of nothing; and that the series of substances out of which it may be formed is limited. And though we cannot absolutely succeed in feeding the patient on substances which will not yield sugar, we can supply a nourishing diet furnishing sugar-forming materials in the scantiest proportion—an end best attained by a pure meat diet. But besides dietetic measures, other, though subsidiary, means may be taken to limit the formation of sugar by suitable hygienic and medicinal treatment. It is convenient to treat of these separately, though they should be employed in conjunction.

*Dietetic treatment.*—All authorities agree that meat should be the main constituent of the patient's food, and that starch, and cane and grape sugar, should be avoided, as well as the substances containing them. It is very important in constructing a diet scale to give the patient as much variety of form as possible, the basis remaining the same. As the diet of the patient is the main factor in his treatment, it is worth the practitioner's while to study the various changes and combinations of food which may be given with impunity. As regards vegetables, the rule is that all green vegetables, or the green parts of vegetables, may be eaten; for where chlorophyll is abundant, starch and sugar are commonly scanty; but this rule has important exceptions, which will readily occur to the reader. Thus the green parts of asparagus and celery may be used, whilst the white portion is highly saccharine. Globe artichokes may be used; Jerusalem artichokes are objectionable. Cabbage, endive, spinach, broccoli, Brussels sprouts, lettuce, spring onions, water-cress, mustard and garden cress, and celery may be used freely. Most fruits are forbidden, but nuts may be used if they can be digested. French beans may be used when quite green; when older the beans themselves must be removed. Haricot beans, peas, and all cereals; tapioca, sago, arrowroot, all forms of macaroni, potatoes, carrots, turnips, parsnips, and beetroot are in the forbidden list. Cheese, especially of the poorer kinds, may be used. Cream, butter, and other fatty articles may be used freely. To sweeten tea or coffee, gluside has been

suggested; but both will soon be found more refreshing without any sweetness. Cocoa made from the nibs can be used. Small quantities of cold tea without milk or sugar, with slices of lemon in it, will often be found palatable, cleansing the mouth and relieving thirst. Rinsing the mouth with iced water will often give more relief than a hearty draught. Slowly sucking ice is a very good plan for relieving thirst. All sweet drinks are in the forbidden list, including most sherries and ports, though some of the former may be found nearly sugar-free. Nearly all brandies contain sugar. Light French and German wines contain little or no sugar. Gingerbeer and lemonade are very objectionable; so are champagne, sweet beer, cider, porter and stout, rum and gin. Whisky is probably the best form of spirit. The use of alcohol in these cases has been gravely questioned. Where alcohol has not been used before the onset of the disease, the patient is probably better without it; but it is quite clear that in many cases it may be taken in moderation with advantage.

There is difficulty in procuring a substitute for bread and potatoes. Three imperfect substitutes are employed—namely, gluten bread, bran bread, and almond bread. Gluten bread, as introduced by Bouchardat, consists of flour out of which nearly, but not quite, all the starch has been washed. It is tough, and patients tire of it; better forms of gluten bread may be easily obtained in London. This last is palatable and nearly starch-free, but does not keep well. Bran bread is now often made, in the form of biscuits or cakes; the bran should always be washed nearly free of flour. Bran bread is hardly admissible when there is a tendency to diarrhœa, but may be useful in constipation. Almond cakes were first introduced by Dr. Pavy. They are rather rich to be eaten with meat, but used by themselves or with wine are excellent. Almond flour may now be obtained from which much of the oil has been expressed.

We pass over Dr. Donkin's skim-milk treatment of diabetes with the remark that in the hands of Dr. Donkin and some others the method has been successful; in the hands of many eminent physicians, both at home and abroad, it has done unmitigated harm. Probably the explanation of any successful cases may be found in the fact, that in certain conditions of diabetes, milk-sugar, as well as certain other substances, does not give rise to grape-sugar. If milk is to be used, butter-milk or sour milk will be best. The treatment by sugar or glycerine is self-condemned.

*Hygienic treatment.*—In this the first and most important point is regular exercise, especially riding or walking, short of fatigue. Bouchardat has strongly advocated gymnastic exercises. These would be useful in modera-

tion, especially in bad weather. Nothing is more grateful or beneficial to the skin after such exercise, or even without it, than a warm bath, temp. 80° to 90°, with a little common washing soda in it. In all cases the avoidance of wet and cold is important. Flannels, frequently changed, should be worn; and it is always safer to change the clothes after the slightest exposure to damp. Little may suffice to give rise to a fatal pneumonia.

*Medicinal treatment.*—This must be considered as subsidiary to dieting, but there are many cases where undoubted benefit results from drugs. Of those which have been found of real value, the foremost is opium. Yet there is no unanimity of opinion as to the mode in which opium acts, and its apparent effects are most discordant—in some cases reducing both sugar and urine without sleepiness, in others speedily giving rise to drowsiness and even apparently to coma. The varied susceptibility of different patients to this drug is very striking. Some will take 20 or even 50 or 60 grains a day with no apparent physiological effect beyond slight contraction of the pupil; others again cannot endure even a few grains. The writer's experience is decidedly in favour of opium. Morphine answers well in some cases. Codeine has been strongly recommended by Dr. Pavy as being the influential ingredient in the raw opium. He begins with doses of half a grain. Next to opium come alkalis, and especially some alkaline waters. Alkalis themselves may be given in various forms—as cream of tartar to relieve thirst, as citrate or bicarbonate of potassium, or, if there is a tendency to gastro-intestinal catarrh, as liquor potassæ, with or without a bitter. The waters in most repute in diabetes are those of Carlsbad, Vichy, Vals, Bethesda, and Contrexeville. An annual visit to Carlsbad, with the rigid system of regimen and dietary carried out under the resident physicians, often suffices, with due care, to keep the patient fairly well for many years.

Lactic acid has been strongly advocated by Cantani. His results seem good, but he also makes use of a most rigid dietary. Pepsin and rennet have been used, but without real advantage.

Of secondary remedial agents, the three most important are strychnine, iron, and cod-liver oil; these favour nutrition, and the cod-liver oil seems to improve the temperature somewhat. Laxatives, not purgatives, should be used for the troublesome costiveness. Mineral waters, castor oil, or alkaline purgatives suit best. Phosphorus in combination with iron as hypophosphite, taken in pills, has proved useful.

**MANAGEMENT OF A CASE OF DIABETES.**—By way of recapitulation we may say a few words here on the management of a case of diabetes. When a case comes under the care of the practitioner he must first ascertain the general state of the patient as a basis

for future comparison. The patient's weight must be carefully taken, and the state of his bowels noted. The quantity and characters of urine passed should be noted daily for a short time, the patient still consuming ordinary diet, unless there be some urgent reason to the contrary. After a day or two the restricted diet should be gradually commenced, sugar and potatoes being first cut off. Next the bread should go, being first of all cut down and used toasted hard or torrefied, and this should merge into the use of bran cakes or gluten bread. Above all things, the patient must not be disgusted with his food, for this favours the secret consumption of forbidden dainties. Week by week the weight should be taken; day by day the sugar estimated, as may easily be done by Roberts's method, and the whole arranged on a card so as to be seen at a glance. Warm baths, exercise, and the other adjuvants must be assiduously employed; and as soon as the effects of the limited diet are clearly marked, opium may be tried, if required. If well borne it should be used determinedly, and pushed to its physiological effects, as indicated by the contracted pupil. If the patient is seen daily, his diet should be regulated each day; if only at intervals, certain available changes should be indicated. When convalescence begins, and the urine has for some time been free from sugar, the diet may be gradually relaxed, beginning with substances containing little starch or sugar, gradually extending to bread in small quantity; potatoes should come last: sugar itself never. When, with every care, restriction of diet effects no diminution of sugar, or, if that be limited, emaciation and weakness rapidly go on, it will be a question whether it may not be best, as it often is, to return to a practically unrestricted diet.

ALEXANDER SILVER.

**DIAGNOSIS of Disease.**—See DISEASE, Diagnosis of.

**DIAPHORESIS** (διά, through; and φερέω, I convey).—The act of perspiring. The term is more generally applied to perspiration artificially induced.

**DIAPHORETICS** (διά, through; and φερέω, I convey).

**DEFINITION.**—Remedies which increase the secretion of sweat. When the increase is so great as to cause the perspiration to stand in beads upon the surface, they are usually termed *sudorifics*.

**ENUMERATION.**—The principal diaphoretic measures are—The Vapour Bath, Turkish Bath, and Wet Pack; Warm Drinks; Warm Clothing; Jaborandi, Pilocarpine; preparations of Antimony; Ipecacuanha; Opium and Morphine with their preparations; Sarsaparilla, Guaiacum, Serpentry, Sassafras, Senega, Mezereon, Camphor; Sulphur; Ammonia and its Carbonate, Acetate, and

Citrate; Alcohol; Ethers (especially Nitrous Ether); and Chloroform.

**ACTION.**—The secretion of sweat usually consists of two parts, namely, a free supply of blood to the sweat-glands, and the abstraction from it of the materials for sweat by the cells of the gland. These two processes sometimes occur independently of each other. In fevers the supply of blood to the glands is abundant, but they do not secrete; and a similar condition is observed in belladonna-poisoning. Belladonna and atropine possess the power of paralysing the secreting nerves of the sweat-glands, just as they do those of the salivary glands, and thus the skin remains dry, although the cutaneous vessels are much dilated. In collapse the cutaneous glands secrete a cold sweat profusely, although the supply of blood to them is deficient.

The secreting cells appear to be under the influence of nerves, by exciting which secretion occurs. The centres for the secreting nerves of the sweat-glands appear to be situated in the spinal cord, and in the medulla oblongata. The fibres seem to run in the same path as the vaso-motor nerves. The secretory nerves of the sweat-glands may be excited directly by stimulation of the nervous trunks in which they run; and the sweat-centres may also be reflexly excited by irritation of various sensory nerves. Certain substances, such as nicotine and carbonic acid, seem to stimulate the sweat-centres; whilst other drugs, such as pilocarpine, appear to act upon the peripheral terminations of the secretory nerves in the sweat-glands themselves. Several remedies, at the same time that they excite secretion, likewise increase the flow of blood through the skin, rendering it redder, warmer, and more vascular. Others, again, excite the secretion at the same time that they diminish the cutaneous circulation. Diaphoretics have therefore been divided into two classes, the former kind being termed *stimulant*, and the latter *sedative* diaphoretics. The exact mode in which each drug already enumerated produces diaphoresis has not yet been ascertained; but antimony, ipecacuanha, and jaborandi are classed as sedative diaphoretics, and all the others as stimulating ones. The supply of blood and the secretion are both increased by the application of warmth, by the ingestion of warm fluids, and by the action of jaborandi.

**USES.**—Diaphoretics are employed to increase the flow of blood to the surface, and possibly to aid the elimination of excrementitious products in internal congestion, such as catarrh of the respiratory passages or digestive tract, and in febrile conditions generally. In fevers, the cutaneous circulation is generally active, and the so-called sedative diaphoretics are then most useful. Diaphoretics are also used to increase the

elimination of water by the skin, and thus lessen the accumulation of fluid in dropsy, or to relieve other excreting organs, such as the kidneys in albuminuria and diabetes insipidus, or the intestines in diarrhoea. In these cases stimulant diaphoretics are indicated.

T. LAUDER BRUNTON.

### DIAPHRAGM, Diseases of the.

The diaphragm is an important part of the respiratory apparatus, in relation to the movements of breathing, and by no means receives the attention from a pathological and clinical point of view which it deserves. Only a brief outline of its affections can be given within the limits of the present article; and for practical purposes they may be conveniently discussed according to the following arrangement:—

1. PHYSICAL INTERFERENCE WITH THE DIAPHRAGM, AND DISPLACEMENT.

2. FUNCTIONAL DISORDERS. (a) Paralysis. (b) Spasm.

3. ORGANIC LESIONS. (a) Injury, Perforation, and Rupture—Diaphragmatic Hernia. (b) Inflammation, acute or chronic. (c) Muscular Rheumatism. (d) Atrophy and Degeneration. (e) Morbid Formations.

1. PHYSICAL INTERFERENCE WITH THE DIAPHRAGM, AND DISPLACEMENT.—The diaphragm is frequently interfered with by morbid conditions within the chest or abdomen, which impede its movements, displace it more or less, either upwards or downwards, or render it tense and stretched. The entire structure may be thus affected, or only a portion of it, such as one lateral half or its central part. The chief thoracic conditions by which the diaphragm may be thus affected are pleuritic effusion, empyema, or pneumothorax, emphysema of the lungs, abundant pericardial effusion, enlargements of the heart, and tumours within the chest. Chronic contracting affections of the lung and pleura tend to draw it upwards, by a process of suction. Pleuritic adhesions, considered independently, have rather a depressing influence than otherwise (Walshe). The principal abdominal conditions which may raise the diaphragm are a distended stomach, tympanites, ascites, peritonitis, pregnancy, large faecal accumulations, and tumours or enlarged organs which attain considerable dimensions, especially ovarian, hepatic, splenic, or renal tumours. It sometimes happens that the diaphragm is interfered with both from its thoracic and its abdominal aspects. Tight lacing may materially embarrass and alter the position of this structure.

**SYMPTOMS.**—The symptoms induced by this mechanical interference are readily explained by its effects. A sense of uneasiness and discomfort is often experienced around the lower part of the chest, amounting sometimes to considerable tension or tightness.

There is not any actual pain, but in some instances, where the diaphragm is much pushed down, the patient complains of a painful sensation referred to the ensiform cartilage, as if the attachment of the diaphragm at this point were being severely dragged upon. The act of respiration is more or less impeded, and this often seems to be the cause of the discomfort experienced. A sensation frequently complained of by patients is that they cannot take a full breath. Respiration may be much hurried, or oppressed and laboured; and not uncommonly the normal relation between the thoracic and abdominal movements is markedly altered, as observed on inspection and palpation, and the diaphragm may so act as to draw in the lower part of the chest-walls in inspiration. Occasionally a kind of spasmodic cough seems to be excited by the tension of the diaphragm produced by certain conditions. The act of coughing is also frequently rendered more or less difficult and ineffectual.

The actual position of the different parts of the diaphragm in a particular case can only be determined by carefully noting the physical signs afforded by the structures in contact with it, on its thoracic and abdominal aspects respectively.

2. **FUNCTIONAL DISORDERS.**—The affections of the diaphragm included within this group are (*a*) **Paralysis**; and (*b*) **Spasm**. It will be understood that in the cases now under consideration there is no structural change in the diaphragm itself.

*a. Paralysis.*—The diaphragm is completely paralysed when the spinal cord is destroyed at the origin of the phrenic nerve, whether as the result of injury or disease. If one or both phrenic nerves be cut across, destroyed by disease, or even severely compressed, the same effect will be produced, either one lateral half or the whole of the diaphragm being paralysed, according as one or both nerves are involved. This structure may also be implicated in diphtheritic paralysis; lead-palsy; and hysteria. Diaphragmatic paralysis has also been attributed to cold. *See PHRENIC NERVE, Diseases of.*

**SYMPTOMS.**—Where paralysis of the whole diaphragm is suddenly produced, death speedily ensues from the grave impediment to the respiratory function resulting therefrom. If it is brought about gradually, or if only part of the structure is involved, the effects are seen in more or less interference with this function, and with the acts in which respiration is concerned. Thus there will be a subjective sensation of a want of power to breathe; while the respiratory movements may be hurried and shallow. It has been noted, however, that, when the patient is at rest, breathing is often quite easy, but any exertion causes it at once to become frequent, and brings the extraordinary muscles of respiration into play. Physical examination

shows that, if a deep inspiration be taken, the epigastrium and hypochondria sink in instead of protruding, while during expiration these regions bulge out again. If only one half of the diaphragm is affected, this abnormal movement is unilateral. Coughing and sneezing cannot be performed efficiently, and sputa cannot be easily expelled; while the abdominal acts for which a tense diaphragm is required, such as defæcation or vomiting, are also ineffectual or impracticable. A peculiar dysphonia has been described, in which the voice loses its power as the day advances, becoming at last a mere cracked whisper. The lower parts of the lungs tend to be more or less collapsed and congested; and if bronchitis sets in, the patient is in grave danger.

*b. Spasm.*—The diaphragm may be the seat either of clonic or tonic spasm or cramp. The disorder may depend upon disease of the nerve-centre at the origin of the phrenic nerves; irritation of these nerves in their course; direct excitation of the diaphragm; or reflex causes. Tonic spasm is most strikingly observed in cases of tetanus; of poisoning by strychnine; or of hydrophobia. A form of asthmatic attack has also been attributed to this condition of the diaphragm; and it may result from immoderate laughter.

**SYMPTOMS.**—The symptoms will vary in different cases. Tonic contraction of the diaphragm gives rise to severe pain, and a sense of constriction in the corresponding region, which may come on in paroxysms; clonic spasms also originate painful sensations after a time, which may become very considerable. Hiccough is probably due mainly to a clonic spasm of the diaphragm (*see HICCOUGH*). If this structure should become rigidly fixed, respiration is gravely interfered with, and the patient soon presents the phenomena of apnoea, which will end fatally if the spasm is not relieved. In the form of asthma supposed to be due to diaphragmatic spasm, expiration is very difficult and greatly prolonged, inspiration being short and abrupt; the lungs are distended; great distress is felt; and there may be signs of impending death from suffocation. A spasmodic cough may be due to clonic spasm of the diaphragm.

3. **ORGANIC LESIONS.**—These may be briefly considered in the order in which they were enumerated at the commencement of this article.

*a. Injury, Perforation, and Rupture — Diaphragmatic Hernia.*—The diaphragm may be perforated, lacerated, or ruptured in connexion with various forms of injury, such as crushing accidents, fractured ribs, penetrating wounds, or gunshot injuries. Should the patient recover, a permanent opening may be left. Rupture of the diaphragm may also occur from violent strain, as of persistent retching, or during parturition; and from the effort to conceal or

suppress the cries of pain in parturition. In medical practice perforation of this structure may be met with as a congenital condition; as the result of the bursting of some fluid-collection through it, such as an empyema, pulmonary abscess, a sub-diaphragmatic, hepatic, renal, or other abscess, or a hydatid-cyst; or from its destruction in the progress of some organic lesion, such as malignant disease, an aneurysm, or a chronic gastric ulcer. It may occasionally occur independently of these causes, owing to the yielding of a weak portion of the diaphragm, especially between the attachment to the ensiform cartilage and the seventh rib. Congenital perforation is rare, but a considerable portion of the diaphragm may be thus deficient. The size and other characters of the perforation differ much in different cases. If it is produced by the opening through the diaphragm of a fluid-accumulation, this fluid escapes from the abdominal into the thoracic cavity, or *vice versâ*, the latter being exceptional; in other instances portions of the thoracic or abdominal organs pass through the perforation, constituting forms of *diaphragmatic hernia*. The writer had the opportunity of observing a remarkable instance in which the entire stomach had passed through an opening in the diaphragm into the cavity of the chest. In a case reported by Dr. Little, of Dublin, the diaphragm presented an almost circular opening, well-defined, sharp, with somewhat thick edges, and through this opening passed a hernia consisting of peritoneum containing some omentum, and about fifteen inches of the transverse and descending colon. The stomach and colon most frequently pass through the diaphragm. In rare instances the small intestines, spleen, or other structure form part of the hernia.

**SYMPTOMS.**—To recognise clinically a perforation or rupture of the diaphragm is generally no easy matter. In cases of sudden and extensive rupture, there will probably be grave collapse and speedy death, and the patient may present marked risus sardonius. Often there are no symptoms referable to the diaphragm, though there may be signs indicating that its functions are more or less impeded. The occurrence of sudden perforation may be known from the previous existence of some condition likely to cause this event, such as empyema, or an abdominal abscess; the supervention of acute pain, accompanied with indications of shock or collapse, and the disappearance or modification of the signs of the original morbid condition; followed by the development of phenomena revealing that fluid has passed through the diaphragm, and accumulated in the thoracic or abdominal cavity, as the case may be, or that some secondary affection has been set up as the result of the perforation, such as peritonitis or pleurisy. A fluid collection may, however,

penetrate the diaphragm without giving rise to any very evident disturbance. When an organ passes through this structure, the symptoms present, if any, are more likely to be associated with this organ than with the diaphragm, and physical examination may possibly detect the displacement. In the case of hernia of the stomach already referred to, the chief symptom was urgent vomiting, which occurred immediately after taking any food or drink.

**b. Inflammation—Diaphragmitis.**—The serous covering of the diaphragm, either on its thoracic or abdominal aspect, is not uncommonly involved in cases of acute pleurisy or peritonitis respectively, and the inflammatory process may penetrate its structure. It may also be involved by extension from pericarditis. Inflammation of the substance of the diaphragm may further arise from injury, direct irritation, pyæmia, or without any evident cause. The anatomical conditions observed are increased vascularity and sometimes ecchymosis; the formation of lymph upon its surfaces, or exudation into its substance; softening and degeneration of its muscular tissue or central tendon; or, in rare instances, suppuration, an abscess forming in the substance of the diaphragm, or pus collecting under one or other of its serous coverings. Ulceration and gangrene have been noticed in exceptional instances. Chronic inflammation of the diaphragm may occur, leading to a fibroid change in its muscular portions, either by extension from neighbouring structures, or as the result of chronic local irritation.

**SYMPTOMS.**—The symptoms of acute inflammation of the diaphragm are generally very obscure and ill-defined. The condition may be indicated by severe pain in the region of this structure, of burning and constrictive character, obviously increased by breathing, so that the respiration becomes instinctively thoracic, as well as hurried and shallow; and also much aggravated by coughing, defæcation, or any other act which disturbs the diaphragm, as well as by movements of the trunk, and pressure over the epigastrium. Sighing, hiccough, painful dysphagia, risus sardonius, and violent delirium are other symptoms described. The movements of respiration will indicate more or less complete paralysis of the diaphragm. The patient will probably be much distressed. More or less pyrexia will be observed. If an abscess should form, this might burst either into the chest or abdomen, and thus lead to secondary pleurisy or peritonitis. The inflammation might also spread to the pericardium. Chronic inflammation and its consequences may possibly be suspected from a want of free movement in the diaphragm, associated with conditions likely to originate this change; but it could scarcely be recognised with any certainty.

c. **Muscular Rheumatism.**—The diaphragm may be involved in this complaint, whatever its nature may be; probably it is attended with structural changes in the tissues. The affection is characterised by pain referred to the diaphragm, which may be very acute when it is in any way brought into play. So long as it is kept at rest, there may be no discomfort, but deep breathing causes considerable pain, so that the respiration is carried on in a shallow manner, and may be entirely thoracic. Such acts as coughing or defæcation cause much pain and a sense of aching.

d. **Atrophy and Degeneration.**—The diaphragm may be involved in the course of progressive muscular atrophy; it may also be atrophied from causes which produce general wasting; or undergo senile atrophy and degeneration; or be similarly affected from local causes, such as interference with its blood-supply from vascular degeneration, want of action, or after chronic inflammation. Fatty and fibroid degeneration are the chief forms met with. Brawny induration has been noticed in scurvy. These conditions tend to give rise to more or less evident interference with the functions of the diaphragm, which in extreme cases would amount to their total cessation, diaphragmatic breathing being rendered impossible, the symptoms being then the same as when the diaphragm is paralysed. In cases of progressive muscular atrophy the fatal termination may arise from this cause. There would not be any pain, but uncomfortable sensations might arise from the impeded respiration.

e. **Morbid Formations.**—The diaphragm is occasionally the seat of malignant disease, being usually involved by extension from some neighbouring structure. Syphilitic and other non-malignant solid growths have in rare instances been found in it. Parasitic formations may also occur in it—namely, hydatids, cysticercus, and trichina spiralis. Tubercle is occasionally found in the diaphragm. Possibly malignant disease might be indicated by signs of impeded diaphragmatic movements, with localised pain, accompanying indications of cancer in other parts. The implication of the diaphragm in trichinosis may also be recognised in some instances by severe pains, spasmodic contractions, and serious interference with diaphragmatic respiration. In most cases, however, the presence of any morbid growth in connexion with the diaphragm cannot be diagnosed during life, and is only discovered at the *post-mortem* examination.

**TREATMENT.**—But little can be done in most cases in the way of direct treatment in connexion with affections of the diaphragm. One of the most obvious indications is to get rid, if possible, of any condition which is mechanically displacing it, or impeding its movements, and preventing it from perform-

ing its functions. In the next place, any disease of which the condition of the diaphragm is but a part must receive due attention, such as progressive muscular atrophy, centric nervous disease, diphtheritic paralysis, lead-poisoning, hysteria, or trichinosis. Collapse or shock due to a sudden diaphragmatic lesion must be treated on the usual principles. Painful affections might be relieved by local applications of dry heat, fomentations, support by a bandage, or anodynes; and if acute inflammation is suspected, a few leeches might be applied. Paralysis or spasm of the diaphragm may call for the employment of electrical treatment, applied through the phrenic nerve; and various remedies are found useful in hiccough. See PHRENIC NERVE, Diseases of; and HICCOUGH.  
FREDERICK T. ROBERTS.

**DIAPHRAGMATIC HERNIA.** —  
See DIAPHRAGM, Diseases of the.

**DIAPHRAGMITIS.** — Inflammation of the diaphragm. See DIAPHRAGM, Diseases of the.

**DIARRHŒA** (*διάρρœα*, I flow away).—  
SYNON.: *Defluxio*; *Alvi Fluxus*; Purging; Fr. *Cours de Ventre*; *Dévoïement*; Ger. *Der Durchfall*; *Bauchfluss*; *Durchlauf*.

**DEFINITION.**—A frequent and profuse discharge of loose or of fluid alvine evacuations, without tenesmus, mainly due to increased peristalsis of the large intestine.

**ÆTIOLGY.**—The causes *predisposing* to diarrhœa are individual peculiarity; childhood—especially the period of first dentition; the climacteric period; and hereditary or acquired weakness of the digestive organs.—The *exciting* causes may be thus classified: 1. *Direct irritation of the intestines* by: (a) *Food* in excess, or of improper quality—for example, salted meat, shell-fish, sour unripe fruit and vegetables, diseased, decomposed, or imperfectly masticated; the products of faulty digestion prematurely passing the pylorus; imperfectly elaborated and fermenting chyme; impure water, such as that containing from 3 to 10 grains of putrescent animal matter per gallon (Parkes); or imperfectly fermented malt liquors. Acute diarrhœa, especially in hand-fed infants, is very frequently caused by fermentation of milk either previous to or after ingestion—the irritant effect being due either to undigested casein, to the products of acid or alkaline fermentation, or to the formation of poisonous ptomaines or the presence of bacteria. (b) *Purgative medicines and irritant poisons.* (c) *Bile*, excessive or acrid. (d) *Fæces*, retained. (e) *Entozoa*—lumbriçi, tæniæ, trichinæ, and *entophyta*—mycosis enteralis (Buhl and others). (f) The contents of a *ruptured abscess* or *hydatid-cyst*. (g) *Intestinal lesion*—such as tubercular or other ulceration.

2. *Defective hygiene*.—Diarrhœa may arise from the dwelling being damp, cold, dark, and unventilated; or from foul emanations from decaying organic, especially animal matter, sewage, or fœcal collections. 3. *Chills, climatic variations, &c.* Diarrhœa has been attributed to insufficient clothing; sudden exposure to cold and damp; chills, as from wet feet, and damp bed or clothing; and rapid variations of temperature, such as hot days and cold nights. 4. *Nervous disturbances*, for example, depressing emotions—fright, grief; neuralgia, hepatalgia (Trousseau), dentition, and other causes of reflex disorder. 5. *Defective absorption with augmented peristalsis* of the small intestines, so that the food is passed unaltered—*hæmorrhagic diarrhœa*. 6. *Symptomatic* in various morbid states, for instance, in passive congestion of the portal vein from disease of the liver, heart, or lungs; peritonitis, especially puerperal; organic disease of the intestines—ulceration (simple, stercoral, typhoid, tubercular, cancerous), lardaceous degeneration, enteritis, acute or chronic; cholera; typhoid fever; dysentery; occasionally in pyæmia, measles, scarlatina, confluent small-pox, malaria, gout, Bright's disease (its later stages), and in anæmia and exhaustion, as from over-lactation, phthisis, cancer, Addison's disease, Hodgkin's disease, exophthalmic goitre, leukæmia, and other affections.

Frequently diarrhœa arises from the combined action of several exciting causes, as when the disease is epidemic during summer and autumn. Pollution of air, water, and food by foul emanations from organic matter decomposing in very hot weather, overcrowding, food (and especially fruit) in a state of incipient decay, excessive heat, and chills, may then collectively determine the result. In children the exalted irritability of the nervous system during dentition predisposes to diarrhœa from slight determining causes. In several forms of diarrhœa fermentation of the intestinal contents plays an important part—*e.g.* in infantile and summer diarrhœa.

**DESCRIPTION AND VARIETIES.**—Diarrhœa may be broadly divided into the *acute* or occasional, and the *chronic* forms; and the numerous clinical and pathological peculiarities of different cases are conveniently grouped into typical varieties. The general effects, varying according to the intensity and duration of the flux, are mainly these:—Emaciation, and, in children, also arrest of growth—the weight either diminishing or ceasing to be progressive; anæmia, indicating defective hæmatisis; desiccation of the tissues from the rapid draining of serum from the blood—hence the thirst, and the very concentrated, acid, and even albuminous urine observed when there is a copious watery outflow from the bowels, as in choleraic and similar forms of diarrhœa.

It will be expedient to describe briefly the principal forms of diarrhœa.

1. **Irritative Diarrhœa.**—**SYNON.:** *Diarrhœa crapulosa* (Cullen).—Simple flux from direct irritation of the intestines is the most common variety of diarrhœa. The evacuations, usually preceded by severe griping pains, are at first feculent and usually fetid and sour, then watery. In children (especially hand-fed) they are often like pale clay or putty, or they contain dense masses of undigested casein before being loose; after evacuation they frequently become greenish, like chopped spinach, from contact with very concentrated acid urine converting the brown colouring matter of the bile into green biliverdin; or they are dark green when passed, and may be so acrid as to excoriate the anus, the genitals, the inner parts of the thighs, and even the heels. Fever is usually absent.

Diarrhœa from irritation is frequently a preliminary stage of the inflammatory, dysenteric and choleraic varieties.

2. **Inflammatory Diarrhœa.**—**SYNON.:** *Diarrhœa serosa*.—When the causes of simple irritation excite inflammation of the mucous membrane of the bowels, fever sets in, and the diarrhœa increases. Usually the evacuations become more serous, and contain shreds of fibrin or mucus or pus. Before the attack passes off, the large bowel is apt to be the main seat of inflammation; then the motions are scanty, frequent, more mucous or glairy, contain streaks of blood, and are passed with severe straining. At the same time the skin is hot and dry.

3. **Choleraic Diarrhœa.**—**SYNON.:** Choleraic diarrhœa; Thermic diarrhœa. This form prevails mostly in hot weather. The onset, indicated by vomiting and purging, is usually sudden. At first the vomited matters are mucous and bile-tinted, and the dejections are feculent—both quickly, however, becoming more and more abundant, watery, and colourless. The copious and incessant outflow of serum may in a short time, and especially in children, induce a striking resemblance to the symptoms of Asiatic cholera—a drawn, sunken, and cyanotic appearance, loss of temperature, scanty secretion of urine, insatiable thirst, and cramps. Even in extreme cases, however, the fluids from the stomach and bowels are rarely free from bile, and are not so like rice-water as in true cholera. The collapsed algid condition, as a rule, rapidly gives place to recovery in previously healthy adults, while it is apt to become fatal in delicate children, children prematurely weaned, the debilitated, and the aged. Rarely, the cold stage being outlived, the patient becomes hot, and passes into a state of stupor, with either bilious vomiting or purging and tympanites—the typhoid stage. In children death is almost invariably if the cold stage exceeds twenty-four hours.

4. **Nervous Diarrhœa.**—The peristaltic movements, and the activity of the glands, of the alimentary canal, are often increased by causes operating through the nervous system. Diarrhœa from mental, and especially emotional, perturbation, is the most common example. Even a chronic looseness may be maintained by debility of the nervous system, induced by worry and anxiety. Exalted innervation of the bowels may be natural, a proneness to diarrhœa from slight exciting causes having always existed; or acquired, when, for instance, a flux once established is apt to be maintained. The unstable nervous system of the periods of rapid development and of the climacteric change predisposes to it. It is often an important factor in chronic diarrhœa. The intestinal nerve-centres may become so sensitive (as in delicate children) that every meal, however small, may induce an immediate call to stool, the motions being liquid or pultaceous, and pale, but otherwise healthy. The peristaltic movements may be even so increased as to hurry the food through the stomach and bowels, so that it appears unchanged in the stools. Time is not allowed for digestion or absorption to be even begun. This form has been termed *Diarrhœa lienterica*, and is most frequent in children before the period of the second dentition. The increased tonicity of the muscular fibres of the alimentary tract may have resulted from previous inflammation of the mucous surface, or is the propagation upwards of some irritation (ulcer, inflammation, &c.) of the mucous membrane of the rectum; or it arises from the products of imperfect primary digestion entering the duodenum. Indigestion is the usual cause in adults. The appetite is, as a rule, voracious; and debility may become extreme. In painful or difficult dentition, diarrhœa arises from irritation of the nerves of the stomach and bowels; digestion is arrested; and the contents of the alimentary canal become acid from fermentation, and are ejected by vomiting and purging.

5. **Vicarious Diarrhœa.**—Embarrassment or suppression of the functions of the skin, kidneys, or lungs may lead to the bowels performing additional excretory work. The flux thus set up is salutary, because compensatory. Diarrhœa from chills (suppressed perspiration) is a common instance, while that from renal and pulmonary causes is less frequently observed, and may be misconstrued by the practitioner. Inasmuch as diarrhœa usually diminishes the quantity of urine, even sometimes to the verge of suppression, the reverse of this clinical fact may be easily overlooked or misinterpreted. Even when forewarned, the observer may at times—especially when the urine is free from albumen—find it difficult to determine whether the diarrhœa is a cause or an effect of imperfect renal elimination: a distinction

having all-important bearings on the treatment. The uræmic and eliminatory character of it may be easily decided when the kidneys are known to be diseased; not so, however, when the only thing ascertainable is scanty—may be albuminous—urine, or total suppression of urine in an elderly patient. In such a case there may or may not be organic disease of the kidneys, and still the diarrhœa may be uræmic, inasmuch as it may depend on 'renal inadequacy.' Diarrhœa from pulmonary embarrassment generally affords relief to breathing and cough. The chronic looseness of some gouty patients is also eliminatory: when it is checked, gout is apt to advance, and the health to suffer.

6. **Diarrhœa from Mechanical Congestion.**—Draining of serum into the bowels is a common result of overloading of the portal vein from an impediment to the flow of blood, either in the vein itself, the vena cava, or the right side of the heart.

7. **Chronic Diarrhœa.**—**SYNON.:** Cachectic diarrhœa.—Chronic diarrhœa is frequently, if not generally, unconnected with intestinal lesions. It may be maintained by chronic catarrh of the intestines, or by an exhausted and impoverished state of the system, as in inanition, either from insufficiency of food or from enfeebled digestion, or in chronic wasting diseases, such as syphilis, malaria, or scurvy. The flux increasing, the debility on which it depends thereby perpetuates itself, and this vicious circle tends more and more to destroy life by anæmia and exhaustion, and even after apparent recovery the diarrhœa has a strong disposition to relapse. These clinical features of chronic diarrhœa are well illustrated by the malady which, from the paleness of the stools, is commonly known in India as 'White Flux'—a result of deterioration of health by climate and malaria (*see* PSILLOSIS). When accompanied by fever and night-sweats, chronic diarrhœa is nearly always due to tubercularisation.

**DIAGNOSIS.**—The different forms of diarrhœa may be readily distinguished from each other by a careful consideration of the causes and symptoms. The diseases most apt to be mistaken for diarrhœa are epidemic cholera, dysentery, and mucous irritation of the bowels from retention of fæces.

(a) *Cholera*, in its less definite forms, may resemble bilious diarrhœa and choleraic diarrhœa. The probability in favour of it may be determined by the absence of ordinary causes of diarrhœa, the paleness and watery character of the stools, tormina being slight or absent, the suppression of urine, and the early exhaustion. The presence of bile in the stools is always in favour of diarrhœa. Vomiting is more frequent in cholera; when it occurs in diarrhœa the vomited matter usually

contains bile and undigested food, while in cholera it is a colourless fluid.

(b) *Dysentery* is usually characterised by fever, tormina, and tenesmus, and frequent scanty muco-sanguinolent evacuations. Sometimes, however, in the early stage, the motions are copious, watery, and feculent, as in ordinary diarrhœa; but the presence of tormina and tenesmus, and tenderness in the regions of the cæcum and sigmoid flexure, indicate the dysenteric nature of the disease. Chronic diarrhœa may be distinguished from chronic dysentery by the absence of a history of acute dysentery, and of mucus and tenesmus, and by the less frequent discharge of blood in the evacuations.

(c) *Mucous irritation of the bowels* from retention of feces may induce a condition resembling diarrhœa—frequent, thin, muco-feculent evacuations, which are, however, shown, on inquiry, to be somewhat scanty, and voided with straining. See FÆCES, RETENTION OF.

TREATMENT.—(a) *Diet and Hygiene*.—In acute or occasional attacks of diarrhœa, everything should be taken in small quantity, and tepid or cold, never hot. Farinacea—arrowroot, sago, rice, tapioca, flour, and the like—are useful, and may be taken in milk, or in chicken or mutton broth, or weak beef-tea. Animal broths—and especially beef-tea—when concentrated, or in large quantity, are apt to aggravate diarrhœa. Demulcent drinks—white of egg in water or milk, rice or barley or arrowroot water; and astringent liquids—infusion of dried whortleberries or roasted acorns, red light wines—may be given. Brandy is often of service, and may be given in an aromatic water or with the farinacea. Lime-water with milk is in many cases of much value.

Rest in bed secures a uniform warmth of skin, and favours the cessation of diarrhœa.

In children, errors of feeding should be corrected. Lumps of casein in the motions may be prevented by reducing the quantity of milk, or by adding thin arrowroot or barley-water, with or without lime-water or bicarbonate of sodium, to the milk; and by regulating the time between meals, providing a wet nurse, or substituting the milk of the goat or ass for that of the cow. In infantile diarrhœa the milk should be previously boiled and diluted—one part in two or three of barley-water—and given regularly every hour in small quantities (two or three teaspoonfuls). The feeding-bottle, when not in use, should be kept in a weak solution of permanganate of potassium. Sometimes, however, milk in any form must be given up, when barley-water or thin rice-water, sweetened by saccharin or milk-sugar, may be substituted. The abdomen should be protected by a flannel bandage, and the feet and legs by warm clothing. See INFANTS, Diseases of; and DENTITION, Disorders of.

Inasmuch as in chronic diarrhœa the flux is perpetuated by the debility and anæmia which it induces, and by the activity of intestinal digestion, it has become a leading principle of treatment to prescribe food rich in materials for the construction of the blood and the tissues, and almost wholly disposed of by the stomach. Hence the happy results frequently observed from a diet exclusively animal, either raw or lightly cooked, the digestion of which may be aided by hydrochloric acid, alone or with pepsin. Individual peculiarity may be gratified, and variety obtained from the use of mutton, veal, chicken, pigeon, and game. Beef, the tough parts of veal, and pork are, as a rule, to be avoided. Milk and farinacea are gradually permitted during the progress towards recovery, but the period during which they should be interdicted may require to be very prolonged—even months. The treatment with raw meat, strongly advocated by Trousseau and Niemeyer, has been successfully applied to nearly every variety of chronic diarrhœa, but especially to that obstinate form occurring from the time of weaning to the close of the first dentition. The meat may be pounded into a pulp or finely minced; then mixed with salt, sugar, fruit jelly, or conserve of roses; or diffused through clear gravy soup, or chocolate made with water or wine. Or the juice may be extracted from it by pressure. Notwithstanding the prohibition of other food, it is best to begin with a small quantity, and to increase it gradually. The only drink allowable is water containing white of egg. Trousseau found opium in small doses, chalk, and bismuth, at and between meals, to assist this regimen. When a restricted animal diet cannot be digested, causes loathing, or aggravates the flux, other varieties of food may be added, and the feeding should be as generous and varied as possible, and adapted to the digestion of the individual. Articles of diet appearing undigested in the motions should be avoided. Low and damp situations should be exchanged for dry and open ones. Warm clothing, flannel next the skin, and flannel waist-belts should be worn. Chronic diarrhœa (lienteric and chronic tropical diarrhœa) has also been successfully treated by a strict milk diet and rest; and by rest along with massage.<sup>1</sup>

(b) *Medicinal Treatment*.—The kind and degree of interference required should first be decided in each case of diarrhœa. A routine prescription of astringents is much to be deprecated. When the flux is moderate and salutary—for example, removing undigested or indigestible materials or irritating secretions, relieving an engorged portal vein, or supplementing a suppressed secretion—it may be left uncontrolled by medicine, or may be encouraged by laxatives, such as castor oil, rhubarb, or a saline aperient,

<sup>1</sup> Eccles, *Practitioner*, Dec. 1890 and Jan. 1891.

combined with a mild sedative, such as henbane or opium. As a rule, the treatment of diarrhœa should begin by removing irritating substances from the alimentary canal by laxatives, guarded by small doses of opium; and astringents, such as chalk-mixture with kino, catechu, hæmatoxylym, and opium, should be held in reserve. The laxative may increase the flux, which, however, soon subsides. Trousseau advocated the use of salines—sodii sulphas, soda tartarata, magnesi sulphas—in progressively decreasing doses, dissolved in a small bulk of water, in the morning, fasting, while others prefer castor oil, or rhubarb. Castor oil is by far the most useful remedy for children, as well as for adults. For the former it should be emulsified in gum and syrup, for the latter in yolk of egg; and as occasion requires it may be combined with a small opiate, for example, compound tincture of camphor, or the wine or tincture of opium, in proper doses.

In *choleric form* or *summer diarrhœa*, the best results are obtained from castor oil guarded by a small dose of laudanum at the commencement, and repeated if the disease is severe; while astringents and opiates alone are withheld until the bowels are relieved of offensive materials, as in the later stages, the stools being copious and watery, griping and distension of the abdomen absent, and the tongue clean. Vomiting should be encouraged by copious draughts of warm water, and, if need be, by emetics of mustard or ipecacuanha. In children, when the motions are colourless, profuse, and incessant, it is best to give hydragryrum cum cretâ in small doses every hour or two, and a very small enema of starch, containing plumbi acetas or cupri sulphas, with laudanum, which may be repeated if necessary. Remedies which are believed to arrest abnormal intestinal fermentation (such as naphthaline, calomel in small repeated doses, perchloride of mercury, potassio-mercuric iodide, carbolic acid, resorcin, salol, salicylate of sodium) have lately been employed with success in infantile and summer diarrhœa. Dr. Luff has obtained the most gratifying results from potassio-mercuric iodide, preferring the following formula: R Liquoris hydrargyri perchloridi ℥xij.; potassii iodidi gr.  $\frac{3}{4}$ ; chloral hydratis gr. j.; aquæ ad ʒj.; to be given every four hours to infants up to six months, and the dose to be doubled for children of more than one year old.<sup>1</sup> In the cold stage there have been recommended mustard baths (for twelve or fifteen minutes, several times a day); emetics (ipecacuanha 2 to 3 grains twice or three times in twenty-four hours); diffusible stimulants (ether in syrup every hour or half-hour). In the stage of reaction, saline aperients or calomel in small doses may be given; white of egg in water as a drink throughout; and, vomiting having ceased, and diarrhœa

being established, bismuth, chalk or lime-water.

In *nervous diarrhœa* the first indication is to allay reflex excitability by the bromides; or, these failing, by opium. When diarrhœa is excited by food, the dose should be given shortly before meals. In *lienteric diarrhœa* arsenic is invaluable. Mal-digestion should be met by hydrochloric acid, bismuth with alkalis, or other appropriate remedies, according to the indications. Occasional doses of castor oil—alone, or with bismuth or small doses of opium or henbane—are useful in clearing away fermentescible matters, which are apt to maintain an irritable state of the bowels. Astringents should only be prescribed after the failure of these or similar measures.

In *vicarious diarrhœa* the skin should be made to act freely by warm baths, or hot air or vapour baths. In renal inadequacy counter-irritation across the loins, digitalis, and nitrate of potassium may be likewise indicated. The diarrhœa should not be arrested or even checked unless it be profuse and exhausting, especially after restoring or augmenting the action of the skin and the kidneys; it is sometimes advisable to nurse and encourage it.

*Diarrhœa from passive congestion* of the portal vein is to be met by treating the cause, for example, disease of the heart, by digitalis, iron, and other remedies.

The flux of *chronic diarrhœa* (lienteric and the chronic tropical diarrhœa known as sprue or psilosis) cannot, as a rule, be stopped altogether by astringents only—the evacuations while thus retained may decompose, and induce flatulence and colic, or fever. The general health should be restored and anæmia removed; the secretions will then generally improve and the diarrhœa subside. Tonics—iron, arsenic, quinine, strychnine—may be aided by astringents—mineral acids, opium, bismuth, chalk, or hæmatoxylym. The best preparations of iron are iron-alum—3 to 5 grains, and liquor ferri pernitratiss—10 to 40 minims. Ipecacuanha and taraxacum are useful when the skin and liver are inactive: from 1 to 3 grains of powdered ipecacuanha may be given night and morning. Podophyllum—2 or 3 minims of a solution of 1 grain in 1 drachm of rectified spirit three or four times a day—is indicated when the motions are watery, pale or high-coloured, and passed with severe cutting pains. Saline purgatives in the early morning have been recommended—2 drachms of sulphate of sodium, sulphate of magnesium, or soda tartarata on the first day; then 1 drachm for fourteen days, dissolved in a small bulk of water, with avoidance of fluids after the dose. Lately Mr. Begg has reported successful results in the treatment of chronic tropical diarrhœa by santonin (the yellow crystals) gr. v., well mixed in a teaspoonful of olive oil, given in the early

<sup>1</sup> *Lancet*, Dec. 20, 1890.

morning or at bedtime for six days.<sup>1</sup> The profuse sweating and colliquative diarrhœa of hectic is best met by hæmatoxylum and diluted sulphuric acid, or opium with astringent mineral salts—nitrate of silver, sulphate of copper, or acetate of lead—given by the mouth or rectum.

Suppressed secretions, particular cachexiæ, disturbed innervation, congestion of the portal vein, and organic diseases of the intestines, form special indications for treatment when diarrhœa is present.

GEORGE OLIVER.

**DIATHESIS** (*διαθήμη*, I dispose).—A morbid constitution, predisposing to the development of a particular disease. See CONSTITUTION, with which, in a somewhat more limited sense, this term is synonymous.

**DIATHETIC DISEASES.**—Constitutional diseases. See CONSTITUTIONAL DISEASES.

**DICROTISM** (*δῖς*, double; and *κρότος*, a stroke) is a term applied to the second great wave of the pulse. This dicrotic wave, or dicrotism, is due to a second expansion of the artery which occurs during the diastole of the ventricle. The *pulsus bis feriens* of old authors was a pulse in which a second beat became perceptible to the finger; an occurrence observed occasionally as an antecedent of hæmorrhage, and also in the course of fevers. The second beat perceived by the finger is not always the true dicrotic wave, but may in some cases be an exaggerated tidal wave. This is the wave perceived in the high arterial tension sometimes antecedent to hæmorrhage. Dicrotism is favoured by a low state of arterial tension, by elasticity of the arterial coats, and by quick and strong ventricular contractions. It is central in its origin, and is a secondary wave of pressure produced in the blood-column by the elastic recoil of the aorta after its distension by the blood injected at each ventricular systole. The recoil of the aorta causes the wave to spring towards the periphery from the closed aortic valves as a *point d'appui*. See PULSE.

B. WALTER FOSTER.

**DIET.**—DEFINITION.—The term 'diet' as here employed may be understood to express the regulation of food to the requirements of health and the treatment of disease.

**GENERAL PRINCIPLES.**—In order to sustain life, a diet must consist of a proper apportionment of the following alimentary principles:—

1. Nitrogenous principles.
2. Non-nitrogenous principles (fats, carbohydrates, &c.)
3. Inorganic materials (saline matters and water).

Whilst these principles hold different relative positions of value, the absence or deficiency

<sup>1</sup> *Practitioner*, Jan. 1891.

of either group will render a diet unfit for the support of life. Milk, the product provided by Nature as the sole article of sustenance during the early period of the life of mammals, may be regarded as furnishing us with a typical dietetic representative of all these principles. The egg also holds a like position, and, as all the parts of the young animal are evolved from it, must needs comprise all the materials for the development and growth of the body.

The required principles are contained in food derived from both the animal and vegetable kingdoms, and the diet may be drawn from either; but, looking to man's general inclination and the conformation of his digestive apparatus, it may be assumed that a mixed diet is that which is designed in the plan of Nature for his subsistence, and it is that upon which he attains the highest state of physical development and intellectual vigour.

Animal food, being identical in composition with the body to be nourished by it, is in a state to be more easily appropriated than vegetable food. It also appeases hunger more thoroughly and satisfies longer: in other words, it gives, as general experience will confirm, a feeling of greater stay to the stomach. Animal food possesses stimulant properties which have sufficed in certain instances, as after starvation, and in those accustomed to a vegetable diet, to produce a state allied to intoxication. This stimulating effect is further illustrated by looking at the relative character of animal and vegetable feeders. Liebig says that it is essentially their food which makes carnivorous animals in general bolder and more combative than the herbivora which are their prey. He then relates that a bear kept at the Anatomical Museum of Giessen showed a quiet gentle nature as long as it was fed upon bread, but a few days' feeding on meat made it vicious and dangerous.

The standard diet framed by Moleschott has been accepted as furnishing a model of what may be considered the requisite proportion of alimentary principles for maintaining health in a person of average stature under exposure to a temperate climate, and the performance of a moderate amount of muscular work. It runs as follows:—

*Alimentary substances in a dry state required daily.*

Dry Food	In ounces avoird.
Albuminous matter . . . . .	4·587
Fatty matter . . . . .	2·964
Carbohydrates . . . . .	14·250
Salts . . . . .	1·058
	<hr/>
	22·859

This, it will be seen, furnishes a supply of about 23 ounces of dry solid matter, of which

one-fifth is nitrogenous. If we reckon that ordinary food contains about 50 per cent. of water, then 23 ounces will correspond to 46 ounces of solid food in the condition in which it is consumed. To complete the alimentary ingesta, a further quantity of from 50 ounces to 80 ounces of water may be assumed to be required to be taken daily under some form or other.

For a life of inactivity, it must be stated that a much smaller amount of food will suffice. The diet, for instance, which is ordinarily supplied to the patients of Guy's Hospital, and which suffices to satisfy and properly sustain them, only contains about 30 ounces of solid food, equivalent to about 17 ounces of water-free material.

It has been mentioned that there are reasons for regarding a mixed diet of animal and vegetable food as best adapted to our nature, and it may probably be considered that the most suitable admixture contains about one-fourth or rather more of animal food.

Looked at from the following point of view, it will be seen that an admixture of animal and vegetable food more economically supplies what is wanted than either kind taken alone, unless the adjustment should be made with the proper apportionment of fat as a representative of a non-nitrogenous article. It is estimated that for a man of medium stature, and performing a moderate amount of work, about 300 grains of nitrogen and 4,800 grains of carbon are required to be introduced daily into the system with the food, to compensate for the outgoing of these elements that occurs. Now this is yielded, as nearly as possible, in the case of both elements, by 2 lbs. of bread and  $\frac{3}{4}$  lb. of meat—that is, 44 ounces of solid food, of which about one-fourth consists of animal matter. If the lean of meat only were consumed, rather over 6 lbs. would be needed to furnish the requisite amount of carbon, and there would be a very large surplus of unutilisable nitrogen; whilst if bread only were taken, the amount necessary to supply the requisite quantity of nitrogen would be rather more than 4 lbs., and this contains nearly double the amount of carbon wanted.

In order to preserve health it is necessary that a portion of the food consumed should be in the fresh state, and this applies to both animal and vegetable food. There may be no lack of quantity, and yet disease and death may be induced by inattention to this fact. Affections of the scorbutic class are produced, which can only be checked and removed by the supply of fresh food or the juice of some kind of succulent vegetable or fruit. The efficacy of lemon and lime juice, for instance, is well known in the prevention and cure of scurvy.

Climate influences the demand for food, and instinct leads to the adaptation of diet to the

requirements that exist. Not only is there a correspondence between the amount of food required and the inclination for taking it, but the nature of the food selected in different countries varies and stands in harmony with that which is most in conformity with what is needed. The dwellers in the arctic regions, besides consuming a large quantity of food, partake of that kind which abounds in the most efficient form of heat-generating material—namely, oleaginous matter. In the tropics, on the other hand, it is upon vegetable products, largely charged with principles belonging to the carbohydrate group, that the native inhabitants mainly subsist.

Labour necessitates a supply of food in proportion to the amount of work done. The employer finds that the appetite of a workman may be taken as a measure of capacity for work—in other words, that a falling off of the appetite means a diminished capacity for the performance of labour.

Until recently it was considered, in accordance with the teaching of Liebig, that muscular and nervous action resulted from an oxidation of muscular and nervous tissue, and that according to the amount of action occurring, so was there created a demand for the supply of nitrogenous alimentary principles to replace the oxidised material. It is now held, however, that the non-nitrogenous elements of food contribute, as well as the nitrogenous, to the production of muscular and nervous force. Fick and Wislicenus undertook a known amount of work upon a non-nitrogenous diet, and proved that the oxidation of their muscular tissue, as measured by the amount of nitrogen voided with the urine, sufficed only for the production of a small proportion of the force expended in the accomplishment of the work performed. The muscles, in reality, appear to stand in the position of instruments for effecting the conversion of the chemical energy evolved by the oxidation of combustible matter into working power. Fats and carbohydrates can furnish the combustible matter required, and, under ordinary circumstances, probably do largely, if not chiefly, supply it. Nitrogenous matter can do so likewise, but it has to undergo a preparatory metamorphosis for effecting the separation of nitrogen in a suitable form for elimination. It must be said, however, that experience shows that hard work is best performed under a liberal supply of nitrogen-containing food. The explanation of this probably is that it leads to a better-nourished condition of the muscles and of the body generally. Under the use, for instance, of animal food, which is characterised by its richness in nitrogenous matter, the muscles are observed to be firmer and richer in solid constituents than under subsistence upon vegetable food.

Persons who lead a sedentary and indoor

life naturally require less food than those engaged in active work, and less should be consumed by them to prevent the system becoming clogged with effete products, which act perniciously in various ways upon the body. The food should also be largely constituted of non-nitrogenous principles, as these tax the excretory organs less than the nitrogenous.

The diet of *infants* is a branch of dietetics the importance of which can scarcely be overrated. The proper food during the first period of infancy is that which has been provided by Nature for the young of mammals—namely, milk. Up to about the eighth month the infant is designed to be sustained solely by its parent's milk. The teeth, which ordinarily begin to show themselves about this time, indicate that some solid matter should now be consumed, and one of the farinaceous products will be the most suitable with which to commence. Bread, baked flour, plain biscuit, or one of the numerous kinds of nursery biscuits that are made, may be employed for a time as a supplement to the former food. At about the tenth month the mother, who ought previously to have commenced lessening her own supply, should now cease it altogether. As the child advances through its second year, and the teeth become more developed, meat, preceded for awhile by gravy, may be given. If the mother cannot suckle her child, and if a wet nurse, whose supply stands next best to that of the mother, cannot be provided, the milk of one of the lower animals should be obtained, and that of the cow gives the nearest approach to what is wanted. Cow's milk, however, is richer in all its solid constituent principles than woman's, and the addition of a solution of sugar or—what is more in conformity with the natural supply—sugar of milk (lactose), in the proportion of an ounce to three-quarters of a pint of water, is needed to bring the two into closer approximation. The milk of the goat is even richer in solid constituents than that of the cow, and therefore stands somewhat further removed from that of the human subject. If, however, it is not adapted for infants, it is highly useful for improving the condition of badly nourished children, and is sometimes employed for this purpose.

**THERAPEUTICAL APPLICATIONS.**—The application of the principles of dietetics may be successfully brought into use in the treatment of corpulency and thinness. A diet rich in nitrogenous matter, conjoined with exercise, promotes the growth of muscle, but the fat undergoes no increase. The conditions most conducive to an increased accumulation of fat are a diet rich in either fat or carbohydrates (provided the requisite amount of nitrogenous matter be present for affording what is wanted for the nutritive operations of life), exposure to a warm atmosphere, and inactive habits. A supply of fat in a

direct manner leads to an increased deposition of fat in the system, but the carbohydrates require in the first place to undergo assimilative change before they can be applied in the same direction.

The details of the dietary to be prescribed where the aim is to produce increased stoutness and an improved condition of the body, should comprise such articles as fat meats, butter, cream, milk, cocoa, chocolate, bread, potatoes, farinaceous and flour puddings, oatmeal porridge, sugar and sweets, sweet wines, porter, stout, and ales.

The converse mode of dieting is necessary for reducing stoutness. Mr. Banting, by his noted system of dieting, reduced his weight from 14 st. 6 lbs. to 11 st. 2 lbs. in about a year. Besides altering, however, the character of his food, he limited the quantity in a manner that must have contributed an important share towards producing the effect observed—not more than twenty-two to twenty-six ounces of solid food (corresponding with eleven to thirteen ounces of water-free material) being consumed, according to his statement, in the twenty-four hours.

As a guide to the corpulent, it may be said that the fat of meat, butter, cream, sugar and sweets, pastry, puddings, farinaceous articles—as rice, sago, tapioca, &c., potatoes, carrots, parsnips, beetroot, sweet ales, porter, stout, port wine, and all sweet wines, should be avoided, or only very sparingly consumed. Wheaten bread should only be partaken of moderately, and brown bread is to some extent better than white. The gluten biscuits which are prepared for the diabetic may, on account of their comparative freedom from starch, be advantageously used as a substitute for bread in the treatment of obesity. The articles that may be taken to the extent of satisfying a natural appetite are lean meat, poultry, game, eggs, green vegetables, succulent fruits, light wines, dry sherry, and spirits. Milk should only be taken sparingly.

Holding the position that food does in relation to the operations of life, the art of dietetics not only bears on the maintenance of health, but is capable of being turned to advantageous account as a therapeutic agency; and it is not too much to say that success in the treatment of disease is oftentimes dependent upon a display of judicious management in regard to food.

In the therapeutic application of dietetics the maxim should be held in view that, whilst the particular requirements are secured, there should otherwise be no greater deviation from what is natural than the special circumstances of the case demand.

The quantity of food consumed may require to be regulated as well as its nature. The quantity administered at a time should stand in relation to the power of digesting it; and to properly compensate for a diminished capacity for taking quantity there should be

a corresponding increase in the frequency of administration. 'Little and often' is the maxim to be followed upon many occasions, and much will sometimes depend upon the strictness with which it is acted up to; for, apart from harmonising with what is wanted, upon the principle that has just been referred to, it meets the defective aptitude that exists in sickness for sustaining any lengthened duration of abstinence from food.

In febrile, acute inflammatory, and other conditions where there is a failure of digestive power, the food administered should be such as not to tax the stomach, and should therefore consist of liquid materials. Solid matter by remaining undigested—and solids of an animal nature are particularly likely to do so—would act as a source of irritation in the stomach, and only serve to aggravate the condition of the patient. The articles under such circumstances to be selected from are beef-tea, mutton-, veal-, or chicken-broth, whey, calf's-foot and other kinds of jelly, arrowroot and such-like farinaceous articles, barley water, rice mucilage, gum water, fruit jelly, and the juice of fruits, as of lemons, oranges, &c., made into drinks. Where a little latitude is allowable, the employment of milk and of eggs in a fluid form may be sanctioned. As circumstances permit, an advance may be made to solid substances which do not throw much work on the stomach, as rice, sago, tapioca, bread and custard puddings, and stale bread or toast sopped. Next may be allowed fish, beginning with whiting. As power becomes restored, calves' feet, chicken, game, and butcher's meat—mutton to begin with—may be permitted to follow.

In cases of ordinary dyspepsia the aim of the physician should be rather directed to raising, by appropriate treatment, the digestive capacity to the level of digesting light but ordinary food, than to reducing the food to an adjustment with a low standard of digestive power. Of butcher's meat, mutton is almost invariably found to be the most suitable; chicken and game are allowable, also white fish (boiled or broiled), particularly whiting, sole, flounder, and plaice. Stale bread, dry toast, plain biscuits, floury potatoes, rice, and the various farinaceous articles form the kind of food derived from the vegetable kingdom to be selected. Green and other succulent vegetables, it is found, are more apt to create flatulence than other kinds of food, and articles belonging to the cabbage tribe are particularly to be regarded as obnoxious to those who have a tendency to this form of derangement.

Much depends in cases of weak digestion upon the state in which the food reaches the stomach. Thorough mastication affords great assistance to the performance of digestion; and when the teeth are bad, the food should be finely minced, or otherwise minutely divided, before being eaten. Regularity in

the periods of taking food tends to promote the orderly working of the digestive organs. An interval of more than four or five hours' duration between the meals is to be avoided, as it acts perniciously in several ways. By inducing an exhausted state of the system it diminishes the energy of the digestive organs, and whilst having this effect it at the same time calls for the periodical exercise of increased energy, on account of the larger amount of food which must be taken at each meal to compensate for the length of time that is allowed to elapse between them.

In cases of ulcer of the stomach, acute gastric catarrh, and vomiting, the food must be selected from that which is nutritious and which at the same time taxes least the digestive powers. Milk—and this is often better borne after being boiled—milk and water, or milk and soda-water, will frequently be found to be tolerated when other articles excite irritation and are returned. Sometimes the milk may be advantageously mixed with isinglass, arrowroot, ground rice, or biscuit powder.

In dysentery and other forms of ulcerative disease of the intestine, scrupulous attention must be paid to diet. The food should consist of articles which are known to exert the least stimulant and irritant action on the mucous membrane and muscular coat of the alimentary canal, and those which best meet the demand in question are such as milk, isinglass, and the various farinaceous products, amongst which rice is pre-eminently valuable. Next to these come eggs, white fish, white-fleshed poultry, fresh game, and fresh meat. Salted and dried meats are highly objectionable, and fruits and succulent vegetables, with the exception of a floury potato, should be strictly shunned. *See PEP-TONISED FOOD.*

The development of gout is known to be favoured by the consumption of a highly nitrogenised diet, especially if conjoined with sedentary habits. With those who have already experienced symptoms of the disease, and those also who have grounds for apprehending its invasion, it is important that an excess of nitrogenous food should be avoided. The diet should be simple, in order that the temptation may be avoided of eating too much, and should at the same time be adjusted to the mode of life. The principle to observe is that the higher the degree of inactivity the greater ought to be the preponderance of food derived from the vegetable kingdom.

Even of more importance than what is eaten is what is drunk, where the question of gout is concerned; and observation shows that it is not distilled spirits, but the stronger wines and malt liquors, which favour the production of the disorder. Nothing is more potent than port wine in leading to the production of gout, and a few years' liberal indulgence in it has often been known to be

instrumental in bringing on the disease where no family predisposition had existed. Dry sherry and the light wines, as claret, hock, &c., may be drunk, certainly in moderation, with comparatively little or no fear of inducing the disease, although any kind of wine appears capable of sometimes acting as the exciting cause of a paroxysm where the gouty disposition is already established. Stout, porter, and the stronger ales, especially those that have become hard from age, rank next to port wine in their power of predisposing to gout. As regards the light bitter beers, which are so extensively used at the present time, the same must be said of them as of the light wines, viz. that with little, if any, disposition to induce the disease, they nevertheless appear capable of sometimes exciting its manifestation in a gouty subject. A pure spirit, as whisky, hollands, or brandy, diluted with water, often forms the only kind of alcoholic drink that is found to agree with those who are suffering from gout.

In Bright's disease with threatening uræmic poisoning it is a point of consideration to diminish as far as practicable the amount of excretory matter to be eliminated by the kidneys. The fats and carbohydrates throw no work upon the kidneys. Their products of destruction escape through another channel. Nitrogenous matters, on the other hand, undergo metamorphosis in the system, and yield nitrogen-containing compounds—chiefly urea—to escape by the kidneys. In this way the kidneys become taxed by nitrogenous food, and, to lessen the work demanded of them, reason suggests that the diet should preponderate in food derived from the vegetable kingdom.

In diabetes mellitus there is a want of assimilative power over the saccharine and starchy principles of food. Whilst these principles become utilised and lost sight of when ingested by a healthy person, in the system of the diabetic they fail to become consumed, but pass off under the form of sugar in the urine, giving rise to severity of symptoms in proportion to the amount of sugar escaping. Much may be done towards subduing the symptoms of the disease by a properly arranged dietetic scheme, and the principle upon which it requires to be framed is the exclusion, as far as practicable, both from what is eaten and what is drunk, of articles containing saccharine or starchy matters.

Observation has shown that the reaction of the urine is susceptible of being influenced by the character of the food. The effect of animal food is to increase the acidity of the secretion, whilst that of vegetable food is to diminish it, and even, it may be, to produce alkalinity. Hence, on persons affected with the lithic-acid diathesis, benefit is conferred by a plan of diet in which animal food is

limited, and succulent vegetables and fruits, with the light wines, as claret, hock, &c., are freely supplied. On the other hand, with the phosphatic diathesis, the converse principle of action should be adopted.

F. W. PAVY.

**DIGESTION, Disorders of.**—The function of digestion is of a physico-chemical nature, being compounded of certain muscular acts, and of certain processes exercised by the digestive fluids on the ingesta, which are thereby converted into a fluid and diffusible state.

Any interference with the due performance of the several components of the function will lead to indigestion; and though it may for convenience be desirable to consider these disturbances separately, it must be remembered that the occurrence of one condition is apt to be quickly associated with another, and hence the forms of dyspepsia as they usually present themselves are of a complex nature, however simple the primary fault may have been. It appears to the writer to be very undesirable to restrict the application of the term *indigestion* or its synonym *dyspepsia* to the perverted actions of any one part of the alimentary system, such as the stomach, or to any one special morbid state, such as gastric or intestinal catarrh; but rather that it should include all departures from normal function which any part of the digestive tract may present. Nor is it possible to consider irregularities of digestion only from the point of view of the organs immediately concerned. Complicated as our organism is, disturbances of other functions will speedily make themselves felt in the one under consideration; and failures in the absorption of the digested food, or in its subsequent metabolic changes and elimination, will tell back sooner or later on that process which is, strictly speaking, limited to its preparation.

**CAUSES OF DYSPEPSIA.**—Dyspepsia may be immediately traced to (1) *the food*; (2) *disturbances of the so-called mechanical processes*—namely, the muscular acts, solution, &c.; (3) *deficiencies in the chemical changes* exercised by the digestive secretions; or (4) *imperfect absorption* of digested products. It is to be understood that the three last-mentioned groups of causes which bring about indigestion are determined by certain gross or minute abnormal changes in the structural tissues of the organs concerned, as described in the article DIGESTIVE ORGANS, Diseases of the.

**1. Imperfections of Food.**—Imperfections of food, whether in quality or quantity, are among the most frequent causes of digestive disorders. A thorough knowledge of the principles of dietetics is essential, that the errors may be recognised and remedied. Since our digestive capability is limited, it

is obvious that, when those limits are overstepped, the domain of disease is entered upon; and although no very absolute lines may be laid down for universal application, the general rules for quantity and kind are capable of being stated. *See* DIET.

(a) *Deficiency of Food.*—Except under rare conditions, such as famine, &c., this is not a common cause of disease. Of the signs and symptoms of starvation it is not needed here to treat. But there are frequent occasions when, with no deficiency in the total bulk of food taken, there is yet a serious want in one or perhaps more of the needful alimentary principles, and this is especially liable to occur in the feeding of children. Setting aside those gross cases of cruelty, when infants and the youngest children are fed almost from their birth with bread, broth, or even meat, there are still too often to be met with children whose diet-scale is almost entirely wanting in nitrogenous matter. Fed chiefly upon infants' foods, the latter consisting of little more than starchy material, their tissues are ill-formed for the want of proteids, which, during the period of growth, are required in a larger relative proportion. The relationship of rickets to prolonged suckling, with the accompanying deficiency in necessary food-stuffs, is now generally recognised.

The effect of a deficiency of food is a general state of malnutrition, in which any hereditary tendencies to disease that may exist have a more favourable field for development. There is a gradual diminution in the weight of the body, and an imperfect performance of its functions, as indicated by muscular weakness, mental lassitude, &c.

The specially dyspeptic symptoms referable to a want of food are, mainly, perversions of sensation, such as a feeling of sinking, emptiness, or even pain at the epigastrium, or generally in the abdomen. Constipation is frequent from lack of stimulus to normal peristalsis, and it is probable that the digestive secretions are defective in quality and quantity, thereby causing imperfect digestion of the food that is taken. A weak digestion is not seldom due to want of the needful stimulation of sufficient food.

The deficiency in food taken may result not so much from a defective supply of nutriment, as from a disinclination to eat, a common symptom of many diseases, especially febrile states; self-imposed fasting too frequent or prolonged; the anorexia of the hysterical temperament; or from obstruction to the entrance of food into the stomach from stricture of the œsophagus. Or the appetite may be impaired by over-indulgence in alcohol or tobacco. *See* APPETITE.

(3) *Excess of Food.*—There is very little doubt but that more food is daily in the habit of being taken than is actually required to restore the tissue-waste, most active lives

being led on an amount far below what is ordinarily regarded as being necessary; and there is equally little doubt that much of what is taken is not in the most digestible form. It is open to question whether the appetite would be satisfied by the ingestion of merely sufficient to balance the waste, particularly if the gross bulk of the food taken were diminished by the removal, as far as possible, of all indigestible matters, leaving little more than the needful alimentary principles. At the same time it must not be forgotten that the appetite is very easily controlled by custom, and determination can in time overcome a vicious habit.

An habitual excess of food, at least in this country, usually errs in the disproportionate amount of nitrogenous matter it contains. Remembering the relatively small quantity of this principle that is essential, and in what a number of the ordinary articles of diet it occurs, this statement will be the more readily accepted. Since all the proteid principles require, to fit them for absorption into the blood, a considerable amount of chemical alteration, and, physiologically speaking, there is good reason to believe that the subsequent metabolic changes of these matters, when absorbed, are more complex than those undergone by fats and amyloids, it would follow that the organs concerned in effecting these changes are very prone to suffer from overwork and its sequelæ. Also many of the substances resulting from the metabolism of nitrogenous matter are liable to become positive poisons in the economy, and the proper elimination of such materials is specially provided for by such organs as the kidneys and skin. The frequency with which these organs become the seat of disease may at least indicate the probability of errors of diet being an important factor in determining the morbid changes, especially as considerable relief is often the result of a restriction of nitrogenous food. There can be little doubt but that the large group of diseases associated with failure in elimination of nitrogenous waste has for a prominent cause an habitual excess of nitrogenous food.

The results of an excessive ingestion of food are as numerous as they are diverse. In many cases there does not seem to be either impairment of health or shortening of life. In some obesity and in others leanness ensues. In a large majority of individuals whose food is much in excess of their wants, particularly if the exercise taken be but little, there are variable symptoms of indigestion, such as a general feeling of lassitude and want of energy, both muscular and mental, a liability to headaches chiefly frontal, constipation, or more rarely diarrhœa, high-coloured urine depositing abundance of urates, a general disposition to sleep, various skin-eruptions, particularly acne, and not

infrequently a feeble heart's action from commencing fatty degeneration of its substance. Any or all of these symptoms may exist, and may be more or less completely relieved by a restricted diet. It is impossible to lay down any exact rules for the quantity of food that should be daily consumed; though it is desirable to remember that the tendency is to take too much, at the same time that age, season of year, and occupation are all circumstances determining variations both in quantity and kind.

(7) *Improper Food.*—Setting aside those extreme cases of perverted appetite occasionally seen in the hysterical condition, there yet remains a very constant violation of the dietetic proprieties. These errors may be classed under the following heads:—1. Substances which are indigestible; either essentially so, or from imperfect preparation (cooking, &c.) 2. Substances which, though digestible, are innutritious or even poisonous. In the first group are included such bodies as the pips and seeds as well as the skins and rinds of fruits, the husks of corn and bran, the stalks and fibres of leaves, and gristle, elastic tissue, and hairs in animal food. For the reducing of these to a fluid and diffusible condition no chemical arrangement exists in the human organism, and they are voided very much in the same state as they are swallowed. Many articles of diet depend in great part for their digestibility on their proper preparation by division, cooking, &c. Thus most vegetables when taken in the raw state are but imperfectly digested, and such nutritious food as potatoes becomes when uncooked positively harmful. The apparent value of raw green vegetables, as lettuce, endive, cress, &c., would seem to depend on the peculiar condition of their mineral constituents, rather than on the vegetable tissues. Such substances as the above-mentioned are apt to produce perversions of digestion in virtue of the mechanical irritation they give rise to, indicated by more or less pain of a gripping character (colic), and frequently accompanied by diarrhœa. The constant indigestion of the more formidable may even set up a gastro-enteritis, acute or chronic. Occasionally articles of food, such as brown bread, oatmeal porridge, &c., are taken for the very aperient action they induce, owing to the irritating nature of the indigestible husks they contain. Symptoms of acute dyspepsia very frequently follow the taking of meat foods enveloped in greasy sauces, since the fat, being undigested in the stomach, prevents the action of the gastric juice on the proteid matter, which then passes on into the intestines, setting up irritation like any other indigestible substance. The most interesting among those articles of diet which, though easily digested, may be poisonous, are those producing their effects only on certain individuals. Such, for example, are certain

mushrooms, shell-fish, or indeed any fish. Remarkable cases are authentically recorded of serious and even fatal results following their ingestion. The symptoms may be those of an acute gastro-enteritis; or, as is very frequently the case, an urticaria is the result, with or without swelling of the eyes and throat. Severe nervous prostration has been met with occasionally. Be it understood that other people have partaken of the same diet with no ill results. The writer is acquainted with a gentleman who for many years was unable to remain in the room when fish of any kind was on the table, its presence inducing severe vomiting, abdominal pain, and general illness; and although the effects are now but slight from the mere smell of such food, very marked symptoms follow on partaking of any. The most digestible and nutritious articles of food may determine indigestion when taken too hot or too cold.

Finally must be included those substances which accidentally find their way into the alimentary canal with the food, as entozoa, ergot of rye, pins, needles, coins, buttons, &c., or lead and other poisons off the hands of workers in them, all of which give rise to definite and for the most part characteristic symptoms.

**2. Irregularities of the Mechanism of Digestion.**—The motor factors of the digestive process depend for their due and normal performance on the integrity of the muscular tissue, the nerve-centres, and the connecting nerves. The several stages of the entire process are mastication, deglutition, the churning movements of the stomach, the peristaltic action of the intestines, and defæcation. Each of these is liable to impairment, in the direction of increased activity, or of deficiency (paralysis), due either to lesions, or to reflex stimulation of the nerve-centres whence the motor stimuli emanate, of the nerve-fibres by which these stimuli are conveyed, or of the muscular tissue by which the movements are performed. Not infrequently more than one of these tissues may be at fault.

Lastly, obstructions to the movements may be caused by tumours, cicatrices, adhesions to adjacent structures, &c. Irregularities of mastication, deglutition, and defæcation are fully considered elsewhere.

(1) *Paralysis.*—Apart from possible structural affections of the intrinsic nervous mechanism of the gastro-intestinal tract—Auerbach's plexus—of which, indeed, little is known, arrest of the peristaltic action of the gullet, stomach, or intestines is undoubtedly often associated with diseased conditions of the central nervous organs; but the exact connexion is far from being satisfactorily determined. Those lesions which interfere with the action of the vagus nerve, and remove its accelerating influence over the peri-

staltic movements, have been regarded as most likely to bring about this condition; but stimulation of the splanchnic nerves, by which inhibitory impulses reach the stomach and bowels, will produce the same result. The nervous exhaustion induced by long fasting, continued vomiting, previous violent peristalsis from purgatives, hysteria, and such diseases as chronic anæmia, typhus, and puerperal fever; also over-brainwork, with the attendant altered conditions of cerebral vascularity, and extremes of blood temperature in pyrexia, or great cold, have been found to be accompanied with symptoms indicating loss of power of the muscular coat of the bowel. Possibly in these cases the result is due to arrest of the vagus influence.

Paralysis of the stomach and intestines is a frequent result of affection of these organs themselves. Inflammation of the peritoneal or mucous coats, with the subsequent infiltration of the muscular coat with the inflammatory products, materially diminishes the power of the contractile tissue. Degeneration of the organs, particularly the lardaceous variety, which, commencing in the mucous, subsequently invades the muscular coat, obviously interferes with the movements. Over-distension from whatever cause will also enfeeble the contractile power of the muscular fibres of the stomach or intestine, whilst moderate distension is the normal stimulus to their activity. If, as is supposed, the energetic contractions of the pyloric part of the stomach, whereby the chyme is propelled into the duodenum, be normally excited by the acidity of the gastric juice, it may be expected that any diminution of this acidity will lead to deficient propulsion of the contents, and their undue retention in the stomach. The movements of the alimentary canal may be considerably lessened by the administration of certain drugs, such as opium, which diminish the excitability of the augmentor centres, or stimulate the inhibitory control.

The results of these various paralytic affections are in most cases sufficiently apparent. The palsied lips and cheeks and tongue tell their own tale by the half-opened mouth, the dribbling saliva, and the cheeks distended with food which cannot be kept between the teeth. When the fauces and pharynx are affected, the painful efforts at swallowing, the rejection of food through the nose, and the passage of food into the larynx, are signs not to be mistaken. Paralysis of the stomach and intestines is mainly recognised by the constipation from inability of the canal to propel its contents, and by symptoms of dyspepsia, such as flatulence due to deficient gastric secretion from want of the requisite stimulation afforded by the churning of the stomach-contents, and to consequent delayed digestion, leading to abnormal fermentative changes. A paralytic condition of the

sphincter ani will be indicated by an inability to retain the fæces. It should not be forgotten that, normally, the gastric movements cease during sleep.

(2) *Excessive activity*.—Excessive activity of the muscular structures of the alimentary canal will be manifested by an increased peristalsis of the stomach and intestine, or by tonic spasms of limited regions. The former condition, by hurrying along the contents at the expense of their proper digestion and absorption, is an effective cause of diarrhoea. The latter, which is altogether abnormal and not a mere exaggeration of a healthy action, will, in proportion to its severity and extent, determine obstruction to the passage of the food. The spasmodic affections are almost always associated with pain—intestinal cramp, colic, tormina, &c.; and varying degrees of discomfort usually accompany exalted peristalsis.

The causes leading to these states may, as in paralysis, be referred (a) to the cerebro-spinal centres, or (b) to the local neuro-muscular structures. Excluding spasms of the muscles of mastication, deglutition, and defæcation, which are elsewhere treated of, increased gastro-intestinal movements (a) may follow on (i.) emotional states; (ii.) some structural lesions of brain and spinal cord, such as basilar meningitis and locomotor ataxy; (iii.) the increased excitability of the neuro-muscular apparatus caused by drugs—as strychnine, and septic poisons; (iv.) cutaneous irritation, as from cold, reflexly stimulating the accelerator nerves; affections of other viscera, especially uterine and ovarian, may act in a similar manner. Whilst the first- and last-mentioned causes tend to cause increased peristalsis of the canal, the other causes mentioned rather lead to tonic spasm.

(b) Causes which may be regarded as exciting gastro-intestinal movements by their local effect may be grouped thus: (i.) irritation due to the character of the contents, such as its indigestible nature, acidity, extreme coldness or even excessive bulk, foreign bodies in the food, as pins, buttons, &c., irritant drugs, or worms; (ii.) drugs which appear to act directly on the gastro-intestinal plexuses and muscle-fibres, as lead, nicotine, &c.; (iii.) increased irritability of the mucous surface, as from inflammation or ulceration, thereby favouring the action of the ordinary stimulants to contraction. Certain blood-states, such as that occurring in the uric-acid diathesis, anæmia, or any other condition in which there is a deficiency of circulating oxygen and overcharge of carbonic acid, are liable to excite spasm or increased peristalsis, probably acting on the nerve-centres concerned, as well as directly on the tissues of the canal itself. Spasm of the pylorus, described as of occasional occurrence, is of theoretic rather than practical interest; a paralytic state of this sphincter is probably

more common. The normal tonicity of the sphincter ani may give place to painful spasm, a condition which is very apt to complicate fissure and ulcer of the anus.

(3) *Mechanical difficulties.*—The due performance of the mechanism of digestion may be interfered with by alterations in the condition of the alimentary canal caused by various kinds of obstruction or dilatation. Thus deglutition may be rendered difficult or even impossible by a swollen tongue or tonsils, post-pharyngeal abscess, tumours of the oesophagus or larynx, or new-growths situated at the cardiac aperture of the stomach. The various obstructive diseases of the pylorus and intestines will obviously interfere with the proper passage of the contents, and in those dilatations of the canal which are liable to develop above a stricture, the food accumulates and is delayed in its passage. The adhesion of coils of the bowels to each other or to adjacent structures is a further source of imperfect movement.

(4) *Diseases of the teeth.*—Lastly, the subdivision of the solid food, so necessary for the effective action of the digestive juices, is only imperfectly performed when the teeth are deficient in number or are carious, and to this cause a large proportion of cases of dyspepsia may be fairly assigned.

So marked a perversion of the mechanism of digestion as vomiting is more fitly described by itself, though it is a very frequent symptom of indigestion.

3. *Imperfections in the Chemical Changes.*—Our knowledge of the normal chemistry of digestion, much as it has advanced of late, is still very far from complete; and, in face of our ignorance, but little can be said of the conditions existing in disease. Yet there are certainly no departures from the healthy working of the body so common as are those associated with the digestion of the food.

The various secretions, whose office it is to convert into a fluid and diffusible form those alimentary principles which cannot be absorbed without such preparation, are formed from the blood by the salivary, gastric, pancreatic, hepatic, and intestinal glands. It is clear that, for these juices to be secreted in proper quantity or of proper composition, the blood, no less than the secreting cells, must be in a healthy condition, and the trophic influence of the nervous centres upon the latter must be unimpaired. If the circulating fluid be laden with imperfectly secreted products of tissue-change, or if it be charged with poison, of whatever origin, it is not to be expected that a normal secretion is to be obtained from it; whilst, on the other hand, a degenerated secreting epithelium is unable to perform a function intimately dependent on the integrity of its protoplasm. Of necessity these two factors—blood and cells—react on one another; any flaw in the one is reciprocated by the other, and thus becomes

intensified by mutual interdependence. Experiment leads us to ascribe the efficacy of these juices in the changes they effect to the existence in them of certain so-called ferments, whilst the result they bring about is mainly one of hydration. How far the various mineral constituents of the secretions aid in the process is uncertain, but at least their presence cannot be dispensed with. In this way, the insoluble starches of our food are converted by the saliva, the pancreatic, and possibly the intestinal juices, into soluble and diffusible sugars; the various proteids are rendered capable of absorption into the blood, by the gastric and pancreatic juices, and perhaps also the succus entericus, being changed into bodies known as peptones. The fats are prepared for absorption by the bile and pancreatic juice, by being in part reduced to a sufficiently minute state of subdivision (emulsion) to permit of their passage through the tissue-interstices, and partly by being chemically altered into soaps. However closely we may imitate the separate actions of these fluids in our test-tubes and laboratories, the conditions are undoubtedly much more complicated in the alimentary canal, where so many changes are simultaneously going on, and so many sets of products are formed.

The various secretions may be morbidly affected in respect to their quantity or composition; alterations which must of necessity profoundly modify the digestive process, though with our present imperfect knowledge we may not be able to refer with accuracy any particular symptoms of indigestion to a special change in either of the secretions.

The *saliva* may be increased in amount (*see SALIVATION*), the fluid being poor in solids and ferment. It may be difficult to trace any definite digestive troubles to this condition, since there is no evidence that the carbohydrates are insufficiently converted; but it is probable that the quantity of slightly alkaline fluid swallowed may interfere with the changes in the stomach. The general wasting which frequently follows on this state may be in part attributable to the anorexia which is so commonly associated with it. Nor can a diminution in the saliva, such as occurs in fever, be held responsible for any special dyspeptic symptom. Should the fluids of the mouth become acid, as from lactic or other fermentation of food therein, a stomatitis of varying severity may be induced, so commonly seen in infants.

Within the past few years considerable attention has been paid to the behaviour of the *gastric juice* in disease, and much valuable information has been obtained, mainly in reference to the acid constituents. The want of a reliable test for the acids has been felt, but for all ordinary practical purposes, so far as present investigation has gone, Gunzburg's reagent would seem to be suffi-

cient for the detection of hydrochloric acid. This is an orange-brown fluid composed of 2 grms. of phloroglucin and 1 gm. of vanillin dissolved in 30 c.c. of absolute alcohol. A few drops of this, gently heated in a porcelain dish with a similar quantity of the fluid obtained by filtering the vomit (or gastric contents withdrawn by stomach-pump or syphon-tube), give a deep crimson crystalline deposit if HCl be present. Lactic acid is detected by it deepening the faint yellow tint of a very dilute solution of ferric chloride. It is now ascertained that during the earliest stage of gastric digestion of an ordinary meal, lactic acid derived by fermentation of the carbohydrates or directly from the ingesta, is alone to be found; later the hydrochloric acid becomes manifest, and at the end of digestion should be the only acid obtainable. Examination of the vomited or withdrawn gastric contents will determine the presence or absence of these acids, and their quantitative analysis may be also approximately estimated. Repeated observations have now shown that the HCl is diminished in amount in pyrexia, anæmia, certain general cachectic states, some gastric neuroses, and in chronic gastric catarrh, whilst it cannot be detected at all in most cases of gastric cancer and atrophic and degenerated states of the mucous membrane. As a means of diagnosis, therefore, the examination of the vomit in this particular becomes of great importance (*see* STOMACH, Diseases of). An increase in the amount of HCl in the stomach may be due to actual hyper-secretion following on the introduction of food, and also to an accumulation of the juice during fasting. Normally there should be no gastric juice in the empty stomach; but in some nervous diseases, such as hysteria and tabes, large quantities of fluid containing a marked excess of HCl may be vomited, independently of food, with epigastric pain and acid eructations. This condition is said to lead to gastro-ectasis and predispose to gastric ulcer. The more frequent cause, however, of 'acidity' is an excess of lactic acid from fermentation of the sugars present, and under these circumstances the HCl is diminished in amount and does not replace the former as it normally should. Butyric, acetic, and other acids of the series may also occur as results of fermentation of the stomach contents. Facts concerning the variation in quality or quantity of the pepsin are wanting, but it would appear to vary with the hydrochloric acid; and the same may be said of the rennet ferment. The efficacy of the gastric juice is impaired by the presence in the stomach of strong alcohol or of bile, which precipitates the pepsin; and an excess of mucus, such as occurs in gastric catarrh, acts prejudicially by preventing the food coming in sufficiently close contact with the secreting surface. During sleep, also, the gastric secretion is diminished.

Notwithstanding that the normal *pancreatic juice* effects active digestive changes in the proteid, fat, and carbohydrate constituents of the food, the results which follow its deficiency in quantity or quality cannot be indicated with precision; possibly its absence may be in part compensated for by the bile and intestinal secretion. It is not to be forgotten that its effective working largely depends on the previous gastric digestion having been properly carried out. There is no doubt but that in many cases, when the pancreatic fluid is completely cut off from the duodenum, the digestion of fats is very considerably interfered with, as shown by the character of the stools, and the rapid emaciation of the patient; but this result is not invariable, and is certainly most marked when the bile is also wanting. In fever the secretion is said to be diminished.

An excessive secretion of *bile* is not clinically recognised except as the result of the administration of certain drugs, but a deficiency or complete absence is of common occurrence in those morbid states associated with obstruction of the bile-ducts, and, to a less degree, in fever. The dyspeptic symptoms directly referable to this state are specially due to the impaired fat digestion, as evidenced by the clayey fœtid stools. Abnormal fermentations, manifested by flatulence, may also take place. The absence of bile further exerts a prejudicial effect on the course of proteid digestion, from the failure to neutralise the acid chyme and consequent precipitation of peptones and arrest of peptic fermentation. The exact changes which ensue are unknown, but there is little doubt that the duodenal digestion of proteids is seriously interfered with by the absence of bile.

So little is known of the action of the *intestinal juice* in health, that nothing can with certainty be affirmed of the part it may play in disease. It is really doubtful which of the alimentary principles it can affect, or whether its action is not limited to effecting further changes in the already partially digested food-stuffs. An excess of the secretion appears to occur, and to form the bulk of the fluid in the diarrhœa of cholera, which may be compared to the salivation and gastric hyper-secretion of certain nerve-states.

In addition to the changes in the ingesta effected by the above-mentioned secretions, there is reason to believe that similar changes of a solvent character may be brought about by the action of *micro-organisms* in the stomach and intestines. Recent observations have demonstrated that numbers of these fungi are normal inhabitants of different parts of the alimentary canal, and that certain species are peculiar to special regions. Originally introduced *ab extrâ*, they propagate enormously, at the same time being subject

to great variation in number, and to some extent in kind, with the food taken. These microbes would seem for the most part to play a minor part in ordinary digestion; although the lactic-acid fermentation of carbohydrates in the stomach, and certain changes in the proteids in the duodenum, resulting in the formation of such bodies as indol, are to be ascribed to their agency. How far, however, such changes are normal and essential is uncertain; but there is no doubt they may be very readily induced and very considerably extended—such as the butyric- and acetic-acid fermentations. In this way may be formed other products of putrefactive decomposition—ptomaines and tox-albumens—often of a poisonous character, and mainly responsible for many remote dyspeptic symptoms; as well as accumulations of such gases as carbonic acid, hydrogen, sulphuretted hydrogen, and marsh-gas, productive of all degrees of flatulence. The exact conditions which permit these abnormal fermentations are not known with certainty. Delay in the propulsion of the gastro-intestinal contents is probably an important factor; and still more so is the proper composition and quantity of the different digestive secretions, by which the activity of many toxic organisms is undoubtedly held in check. Diseases such as cholera and enteric fever, due to specific germs, are not ordinarily accounted as 'disorders of digestion,' though many of their most marked symptoms as well as anatomical lesions are connected with the digestive system.

**4. Deficient Absorption of the Digested Products.**—This may reasonably be supposed to cause indigestion, as it certainly does the subsequent impairment of nutrition. The accumulation in the stomach and intestines of materials which should have been gradually absorbed into the blood or chyle vessels must interfere with the healthy action of the digestive secretions, much in the same way, probably, as the alcoholic fermentation of sugar is arrested in time by the presence of the very products of such fermentation.

Data for estimating the normal rate of absorption from the alimentary mucous membrane of the ordinary results of digestion are very imperfect; and, until they are more clearly ascertained, it is not possible to diagnose with certainty this functional defect, though its existence may often be inferred with much probability, and may suggest modifications in the size if not in the quality of the meals taken.

Conditions upon which an impaired absorption of the digesta may be assumed to depend, are (i.) failure in converting the food to a diffusible state from inefficiency of the secretions; (ii.) impaired peristalsis, whereby the materials are not brought into sufficiently close contact with the mucous mem-

brane; (iii.) deficient circulation of the blood and lymph in the chylopoietic area, and consequent hindering of osmosis; (iv.) disease of the vessel-walls, impairing their permeability; and (v.) degeneration of the epithelium, with the same result.

**SYMPTOMS OF DYSPEPSIA.**—The almost numberless symptoms which indicate the perverted functions above described, and are primarily dependent on morbid structural changes in the organs concerned, may be conveniently grouped into *local* and *remote*. Many so-called dyspeptic symptoms, however, are more properly referable to subsequent perversions of metabolism than to actual digestive imperfections.

The *local* symptoms—those, that is, which are connected directly with the affected structures—are, perversions of sensation; constipation or diarrhœa; vomiting; pyrosis and acidity; hæmatemesis; flatulence; eructation and foul breath; salivation or dry mouth; morbid states of the tongue; abnormal character of the stools. Excepting the first-mentioned, these receive detailed description in the present work under their respective headings.

Ordinarily we are unconscious of the process of digestion, but in disease the function may be accompanied by alterations of sensation varying from a mere sense of weight and discomfort in the abdomen to the severe spasmodic pain of colic. Such, however, are not constant, for very definite indigestion may exist without the patient ever complaining of abdominal sensations. The ingestion of food may be followed by a feeling of abnormal repletion, or of emptiness with craving for food; or there may be heartburn, an ill-defined sense of burning felt in the epigastrium or over the chest or extending to the throat; or positive pain, or tenderness over some tolerably definite area. Sensations as of excessive movements of the bowels, of sinking, or of tightness across the abdomen are of frequent occurrence.

Among the numerous *remote* symptoms are pain in shoulder, back, or limbs; headache—frontal, occipital, and vertical; vertigo and giddiness; muscæ volitantes; tinnitus aurium; cramps in the limbs; muscular weakness; palpitation, cardiac irregularity, flushings, and anginous attacks; cough and singultus; impaired appetite; all degrees of mental perversion, from irritability to apathy and hypochondriasis; drowsiness or insomnia; a peculiar sallow, muddy-looking skin; various cutaneous eruptions, chiefly papular; jaundice; and abnormalities of urine. Mal-digestion may also determine a condition of fever, which may be so severe as to reach the typhoid state and end in coma. Such general perversions of nutrition as wasting and obesity may result from indigestion. Many of these symptoms may be regarded as reflex in causation, such as

distal pains and cough, the afferent nerve-path for which is probably the vagus; others are doubtless to be attributed to actual want of proper materials for the nourishment of the tissues, owing to their improper preparation in the alimentary canal; and others again are possibly toxic in character, due to poisonous substances produced in the course of the perverted digestion.

The extreme diversity of these symptoms is remarkable: there is scarcely a function of the body that may not be implicated in the disturbance of digestion, and contribute its share to the total symptoms of any given case. The degree to which these manifestations may be present is, moreover, most varied: in one patient the malady may be mainly represented by a headache, in another the evidences of illness are most numerous. And, again, the different ways in which these symptoms are associated in different cases are almost as many as the cases themselves. It is impossible, therefore, to attempt any description of a case of indigestion which shall have other than the most limited application; and since the symptoms set forth find their fuller explanation elsewhere in this work, the bare enumeration of them must suffice here. It is important to remember that many of the symptoms mentioned are indicative of disordered digestion are of not infrequent occurrence in the course of maladies quite distinct, or at least primarily so, from digestive diseases: headache, vomiting, cough, palpitation, &c., are illustrations of this. So that, although the recognition of the symptoms be not difficult, care and experience are needful to ascribe them to dyspepsia. But supposing that causes other than indigestion have been excluded, there still remains the task of referring the manifestations of disease which any case may present to their causal perversions in the digestive process: to determine, that is to say, what may be the morbid change which has taken place in the secretions and consequent chemical processes, or the disturbances of motility, or may be the error of diet; and, if possible, to infer beyond these conditions the underlying structural lesions. The intimate interdependence of the various factors of digestion renders the discrimination of the primary fault, from the symptoms present, a matter of exceeding difficulty; and it is this which makes the accurate diagnosis of a case of dyspepsia so uncertain, and its treatment often so empirical. Many of the signs and symptoms, without doubt, indicate with tolerable certainty the region of the alimentary system which is at fault, and may even suggest the nature of the disease; but how far, and in what manner, morbid change in one part of the canal, or at one stage of the digestive process, may determine subsequent changes, and what the exact nature of these may be, and their relation to the symptoms manifested, are data at present

almost unknown. The progressive character of the function of digestion—that is to say, the continuous series of stages, whereby the later ones are dependent on those preceding—renders affections of this system very different in their detection from those of other organs. Alterations in the physical characters of the digestive organs, such as a dilated stomach or an enlarged liver, may usually be ascertained with accuracy, and some symptoms may be reasonably asserted to follow from these conditions, whilst their relation to other morbid manifestations is often of doubtful inference. Modifications in the quantity or quality of the digestive secretions may be demonstrated with certainty, but their connexion with many of the symptoms which may be present is often most obscure. And, again, there may be most persistent and serious disturbance of digestion, with severe discomfort to the patient, and it may be quite uncertain which organ is primarily at fault, and still more doubtful what the real morbid change is. So far as individual symptoms may be referred to definite diseases of the various digestive organs, this is attempted in the articles treating of the diseases of the stomach, intestines, liver, pancreas, &c., which are complementary to this section.

TREATMENT.—The essential basis for the successful treatment of indigestion is the recognition of the cause. Oftener perhaps than may be supposed this is a removable one; in every case it must be well sought for, and corrected if possible. A carefully regulated diet, both as regards ordinary food and special idiosyncrasies, is all-important, and the means from which much good is to be expected, both by way of prevention and cure. To lay down a requisite diet entails a general knowledge of the average composition of food-stuffs and of the changes which they normally undergo in the process of digestion, together with the nature of the influence exerted by various ingredients of the food upon the digestion of the others—*e.g.* ‘the retarding effect of tea and coffee on peptic digestion’ (Roberts). Whether or not any given article of diet should or should not be allowed must depend upon its digestibility in the given case, apart from what may be its value under healthy circumstances, and also upon what harmful by-products of digestion it may give rise to under the diseased conditions which may be present. Exact information on several of these points is not yet available, and at the same time it is to be remembered that articles of food which normally are regarded as of the most digestible and innocent character may at times seriously disagree, whilst most indigestible articles may be taken with impunity. The writer is convinced that, whilst fully recognising the great importance of diet, it is nevertheless the case that not a few

dyspeptics may trace their ailment to a long-continued adhesion to a strict diet which might theoretically fulfil all physiological requirements, and that many cases of indigestion may be considerably benefited by a laxity which might not always be justified on physiological grounds. As useful adjuncts to dietetic treatment are the various artificial partially or wholly digested food-stuffs, such as maltines, peptones, and emulsions of different kinds. Such details of general hygiene as exercise, bathing, occupation—mental and bodily, change of scene and air, require attention scarcely, if at all, secondary to diet. Undoubtedly, much may be done with drugs, both in relieving the symptoms and in treating the conditions on which the symptoms may depend. The latter are met by such means as abstinence from food for periods, with consequent rest to the organs concerned, the requisite nutriment being supplied *per rectum*; by artificially digested aliment, thus helping the digestive juices; and by supplying the elements of the secretions—acids, alkalis, and ferments, when there is reason to suppose they may be deficient. Irregularities of movements of the canal may be remedied by such drugs as strychnine, nux vomica, belladonna, and opium, or more active aperients or astringents. Pain and other sensory disturbances of the stomach or intestines may be relieved by alkalis, bismuth, hydrocyanic acid, morphine, or opium. Among the long list of drugs whose value is assured in different cases of dyspepsia are arsenic, iron, the vegetable bitters, silver, creasote, charcoal, valerian, the hyposulphites, and the various carminatives.

**CONCLUSION.**—In the foregoing remarks no attempt has been made to enter into a detailed description of the various symptoms of disordered digestion, or to do more than indicate very generally the treatment to be followed. Such subjects are left to the diseases treated of in their respective sections. Nor has it been thought desirable in this article to treat the subject from the ill-defined point of view of ‘varieties of dyspepsia.’ Rather it has been sought to bring the matter of indigestion within the limits of an anatomico-physiological basis, since it is only on such lines that the protean symptoms of dyspepsia can be accurately defined. At the same time, whilst for clearness the various causes have been made to assume a somewhat tabular form, it is not intended that the interdependence of these states should be overlooked, or to suggest that one only of the causes mentioned is at work in any given case. The complexity and harmony of our functions alike forbid such a mistake being made. Yet for that mental analysis which the formation of a diagnosis presupposes, some such scheme as the foregoing is essential, no less than for the adoption of a rational treatment.

W. H. ALLCHIN.

**DIGESTIVE ORGANS, Diseases of the.**—The organs comprised in the digestive system have for their function the preparation of the solid and fluid ingesta of the body, so as to fit them for absorption into the blood. Some of the food requires little or even no such preparation; some needs considerable treatment, both physical and chemical. To effect this object it would appear to follow that there should be some receptacle or series of receptacles into which the food may readily be taken, and from which the worthless residue may escape, provided with muscular structures to ensure a movement of its contents. It would further follow that there should be certain organs communicating with the foregoing, whose function it should be to prepare those materials necessary to effect the required chemical changes in the food; and, lastly, that some arrangement should exist to permit of the ready absorption of the digestive materials. Such requirements we find supplied in the alimentary canal, with its terminal apertures, and its continuous muscular coat so arranged as to maintain a progressive advance of the contained food, though with varying degrees of speed—for some lengths, as through the gullet, without any arrest; through others, as the stomach, with considerable delay. Into this canal open numerous glands (mucous, salivary, gastric, intestinal, hepatic, and pancreatic), the secretions of which play each their special part in the conversion of the food to a fluid and diffusible state. From an anatomical, and indeed a genetic, point of view, these glands may be regarded as more or less complicated diverticula of the mucous surface. In order that the food when so treated may gain a ready entrance into the blood, the surface of the canal in contact with the digesting food—mucous membrane—offers various modifications—villi, &c.—to facilitate the process of absorption. Lastly, in beings so complex in structure as man, there is need for some controlling influence to bring the operation of this system of organs into harmony with the actions of other and interdependent systems. Such power of co-ordination is exercised *viâ* the nervous system, sympathetic and cerebro-spinal.

By the expression ‘diseases of the digestive organs’ is meant, departures from the normal structure of the tissues of which these organs are composed.

The constructive tissues of the alimentary organs are:—(1) the Epithelial; (2) the Connective, including the Lymphoid; (3) the Muscular; (4) the Nervous; and (5) a compound texture—the Vascular. Each of these is subject to its own perversions, either alone or in common with others.

**ÆTIOLOGY.**—If we consider diseases to be altered functions dependent on altered structure, the latter being determined by some perversion in the normal stimuli to nutrition,

either hereditary or acquired, we shall at once recognise that the opportunities for abnormal stimulation in the case of the digestive organs are most numerous. Communicating with the external world, and continuously subject to the admission of foreign matter, we have in the character of the ingesta abundant sources of disease. Toxic agents, living and dead, find ready entrance; and excesses in quantity of food, no less than imperfection in its quality, alike serve to produce those departures from the normal structure on which perversions of function depend. Furthermore, the tissues of the alimentary viscera are, equally with those of the body generally, subject to those more obscure hereditary influences which determine irregularities in structure and their sequence. And, finally, arrests in development of organs, not infrequent in those under consideration, complete the list of possible diseases to which the alimentary system is liable.

Nor is this system independent of morbid conditions affecting other organs. So complicated as is the human body, it is impossible that disease should for long be limited to one region. Sooner or later the functions which are acting the one with the other, to constitute the harmonious working of healthy life, will feel the effects of the one that is out of gear, and will respond each in its own manner to the abnormal condition of its environment. A disease primarily located in the nervous system will produce an effect in the working of the nutritive functions, none the less real because the exact lesion cannot as yet be determined. Failures in elimination of the products of tissue-waste, from structural diseases of the excretory glands, must tell back on the organs concerned in the preparation and elaboration of the ingesta; and such conditions constitute a frequent cause of disorders of digestion. The causes may be thus tabulated:—

#### A. Hereditary.

1. Arrests in development of tissues and organs.
2. Abnormal nutritive stimuli, determining new-growths, &c.

#### B. Acquired.

1. Poisons.
2. Imperfections in quantity or quality of normal ingesta.
3. Failure of excretory functions, with consequent circulation of an impure blood, and malnutrition of tissues.
4. Tropic disturbances acting *via* the nervous system.
5. Traumatic.

**VARIETIES OF DISEASE OF THE DIGESTIVE ORGANS.**—There is scarcely any form of diseased structure that is not to be met with in the tissues comprising the digestive organs. Since almost every variety of texture is found in them, and there is so extensive a liability

to the causes of disease, this result is only to be expected.

#### I. Affections of the Vascular State.

Regarded collectively, the organs of digestion present several points worthy of remark in respect to their blood-supply. First, the arrangement of the vessels is such as to ensure a very extensive, and at the same time very direct, supply. The arteries to those alimentary organs situate in the abdomen are almost all primary branches of the aorta; and this, together with the numerous and free anastomoses between them, reduces to a minimum the chance of failure in circulation. Secondly, the blood from the same area is all collected into one large vein—the portal—and after circulating through the liver is carried by one—the hepatic—directly into the inferior vena cava, close to the right auricle. Such an arrangement, whilst considerably reducing the speed of the blood-flow through the hepatic capillaries, offers a double chance—*viz.* in the liver and in the heart—of producing a general state of congestion of the alimentary organs. Thirdly, the existence of such an organ as the spleen, which by its position and structure, as well as its periodic rhythmic contractions, allows of great variation in the amount of blood it contains, will materially affect the extent of vascularity of the digestive organs. And, lastly, the alimentary organs probably undergo, within normal physiological limits, a wider variation in amount of blood than does any other system. The *post-mortem* appearances of the vascularity of the canal, both normal and morbid, are most deceptive, and give little or no indication of what actually exists during life. This is due in great part to the constriction of the vessels after death, and a consequent bloodless state of the tissues.

1. *Hyperæmia.*—*Hyperæmia* is here taken to mean an excess of blood in the arterial side of the capillaries. How far a determination of blood to the alimentary canal may exist, unaccompanied by any change in the tissues, is a matter of doubt: it rarely if ever leads to any recognisable morbid state. In the normal process of digestion this condition obtains, but with it there is an alteration in the glandular epithelia, if in no other tissue-elements. It is conceivable, however, that a vaso-motor paralysis with consequent fluxion may occur, and such may be the case in certain mental states, as indicated by diarrhœa. The majority of circumstances that produce *hyperæmia* do not stop at that point, but bring about a state of catarrh and inflammation, in which the epithelial and connective tissues are also engaged.

Among the digestive glands, the liver undoubtedly manifests states of simple *hyperæmia* without any appreciable changes in the hepatic tissues. Excessive feeding, irritants such as spices and alcohol, hot climates and

malaria, suppressed menstruation, have all been recognised as producing temporary enlargements of the liver from active vascular engorgement, although without doubt these causes if continued lead to structural affections.

2. *Congestion*.—An excess of blood primarily in the venous side of the capillaries, brought about by some impediment to the return of the blood in the veins, has little or no analogy with any normal physiological action. As a condition of disease it is more important and far more common than the preceding.

There are two chief causes leading to its occurrence: (A) It may be part of a general congestion due to obstruction at the right side of the heart from tricuspid dilatation. Owing to the very direct communication of the veins of the chylipoietic viscera and the right auricle, these organs are among the first to experience the effects of the cardiac obstruction. (B) Obstruction through the portal circulation in the liver, either by compression of the portal capillaries by cirrhosis, &c., or by pressure on the portal trunk by enlarged glands, tumours, &c., will directly produce congestion of the gastro-intestinal tract and pancreas. Congestion of the alimentary canal and glands, when due to either of these causes, is in the main progressive in its nature, though occasionally liable to temporary variations in degree.

As a rule, changes which are strictly limited to alterations in the fulness of the vessels are not to be detected with certainty after death; but extreme conditions, especially if associated with any hæmorrhage into the sub-epithelial tissue, may present *post-mortem* appearances often mistaken for irritant poisoning.

3. *Results of Increased Vasculature*.—(a) *Hæmorrhage*. Over-fulness of the capillaries, from whatever cause, is liable to lead to extravasation of blood, either by diapedesis of the corpuscles and transfusion of the fluid part of the blood, or from actual rupture of the vessels. It is much more common and far more extensive in venous congestion than in arterial hyperæmia. It must not be too readily assumed *post mortem* that either of these conditions alone is the cause of the hæmorrhage, since minute ulcers of the mucous membrane communicating with main vessels have been met with. Dependent on the cause and situation, the effused blood may vary in colour from bright red to a coffee-ground appearance. Hæmorrhage may be also due to altered states of the blood, such as purpura and scurvy. (b) *Œdema*. An over-distension of the vessels, especially of the veins, if it be at all persistent, is invariably accompanied by an effusion of serum into the substance of the viscera themselves, and into the alimentary canal, in the latter case producing diarrhœa. (c) Tissues the seat of a chronic congestion in time undergo

certain structural changes as the result of their impaired nutrition, resulting in *atrophy* or *degeneration*; or they may be characterised by the presence of an excessive amount of connective tissue, containing fewer protoplasmic elements than normal, and exhibiting a marked tendency to contract. This *fibroid substitution* may occur throughout the entire digestive system, but is particularly noticeable in the stomach, intestines, liver, and pancreas. (d) Another result of a long-continued congestion is to develop—often to an enormous extent—collateral circulations, especially at the points of communication between the vessels of the portal and systemic areas; e.g. the connexions between the gastric and œsophageal veins around the cardiac orifice, and by the latter with the phrenic, intercostal, and azygos veins, and thence with the inferior vena cava, or the junctions between the hæmorrhoidal veins and branches of the internal iliacs. Considerable compensation is thereby very frequently established, but the over-distension to which the vessels are subject especially favours their rupture.

4. *Anæmia*.—The alimentary viscera, in common with the rest of the body, may share in a general bloodlessness due to excessive loss or extreme malnutrition from wasting disease, &c. A deficiency of blood limited to these organs is not clinically met with.

5. *Infarctions*.—As compared with the brain, spleen, and kidneys, the organs of the alimentary system would appear to be less prone to suffer from emboli and thrombi, or at all events from the effects of these obstructions. A partial exception to this general statement must be made in the case of the liver, which is a frequent seat of abscess determined by the arrest in the portal capillaries of septic particles taken up by the portal radicals in dysentery, &c. Any of the vessels of the liver may be occluded by emboli, or thrombosis following on endarteritis or endophlebitis, but they are conditions of very rare occurrence. The superior mesenteric artery has been known to be blocked by an embolus, with consequent infarction and profuse intestinal hæmorrhage. Minute emboli in the gastric arterioles, or thrombosis from degeneration, atheromatous or hyaline, of these vessels is an undoubted cause of ulcer of the stomach, though not the only one.

II. *Structural Affections*.—1. *Inflammation*.—This term is applied to express those changes which take place in the nutrition of a tissue subsequent to the application of some abnormal stimulus which shall not have been sufficiently powerful to produce destruction. The changes in the structural elements of the textures result partly in the production of some material which is unlike the normal constituents of the part affected, and partly also in certain destructive phenomena. One or other of these phases may predominate, as suppuration and abscess or

ulceration, &c. Certain variations present themselves in the nature of the new-formed material, and also in the general course of the process, constituting forms of inflammation, as simple catarrhal, diphtheritic, phlegmonous, &c. In vascular tissues there are, in addition to the tissue-changes, certain alterations in the circulation in the affected region, commencing with hyperæmia and leading to a variable amount of stasis and œdema.

Inflammation as it affects the alimentary tract offers no exception to this description. It is rare for the muscular tissue to share in the process, which is practically limited to the epithelium, mucosa, and submucosa. The transition, so far as anatomical appearances are concerned, from the normal state of activity of the organs, with their increased vascularity and cloudy appearance of the epithelial cells, to that of simple inflammation or catarrh, is but a step marked by no abrupt line.

Different sections of the entire canal appear to be more prone to certain forms of inflammation than are others. It is unusual for the whole length to be involved, and certain regions, notably the jejunum, are but exceptionally affected. The mouth, pharynx, stomach, lower part of the ileum, and colon are the usual seats. *Catarrhal* inflammation, characterised by intense redness and swelling of the mucous membrane, with an excessive secretion, mucous or serous, and desquamation of the epithelial cells, is of commonest occurrence almost throughout the canal (*see* STOMATITIS; STOMACH, Diseases of; and INTESTINES, Diseases of). When but limited areas are affected and the cause is removed, this condition runs an acute course and tends to recovery; but when the greater part of the canal is involved and the cause is of specific virulence, the effects are more serious. This is the case in cholera, whether of the Asiatic or English type, the anatomical lesion of which is essentially an intestinal catarrh. It is noticeable that the epithelia of this tract are but little prone to manifest a purulent inflammation, as is so characteristic of some other mucous surfaces, such as the nasal, bronchial, and vaginal. *Phlegmonous* inflammation, probably bacterial in origin, corresponding to erysipelas of the skin, and distinguished by the formation of pus in the mucosa and submucosa, represents a more severe inflammatory process, accompanied by a more severe constitutional disturbance. It is of most frequent occurrence in the fauces and tonsils, where it is common (*see* TONSILS, Diseases of), and far more rarely in the stomach, where it constitutes a very serious disease. Under the term *membranous* or *pellicular* are included those forms of inflammation in which the new material assumes the form of a false membrane on or in the mucous surface. This is represented in the mildest degree by aphthæ,

which characterise a special form of stomatitis, and are small patches of coagulated exudation from the blood involving leucocytes and the deeper layers of the stratified oral epithelium. After a day or two's duration they break down and are thrown off, leaving an abrasion or not according to the extent to which the tissues are included in the patch. The more severe forms of inflammation associated with formation of false membrane are termed *croupous* and *diphtheritic*, words which have been extensively misapplied, with much resulting confusion. The former denotes that form of inflammatory product which consists of a coagulated fibrinous exudation effused among the necrosed epithelial cells and into the mucosa, together with many leucocytes and a few red blood-cells; in the latter, the necrosis of the tissue-elements is more extensive and the adherence of the membrane is more intimate. The fauces are by far the most frequent seats of these states, but patches of false membrane are of occasional occurrence in the stomach, and more commonly on the edges of the sacculæ of the large intestine. It is doubtful, however, whether such membranes are diphtheritic in the clinical sense of the term, however closely they may correspond in anatomical characters.

An acute or sub-acute attack of catarrhal inflammation of the stomach or intestines is likely, if the cause persist, to become chronic, the structural features of which are especially an atrophy of the epithelium, with or without much fibroid thickening of the mucosa. The wasting of the epithelium is often very marked, and when the villi also become shrunken the surface of the mucous membrane appears smooth and glazed over large areas. In young children this atrophy appears frequently to follow a single attack of acute intestinal catarrh. Some forms of stomatitis, though persisting a long time, can scarcely be said to be chronic in the sense of leading to permanent structural changes; the utmost that occurs is a thickening of the epithelium, which usually disappears in time. The fibroid overgrowth may affect the stomach or parts of the intestine tolerably uniformly, or not infrequently form polypoid elevations.

Certain peculiarities in the inflammations of special regions, such as the cæcum, colon, and rectum, are fully described elsewhere. *See* CÆCUM, Diseases of; RECTUM, Diseases of; and TYPHLITIS.

The accessory glands (salivary, pancreas, and liver), which genetically are but diverticula of the primitive digestion-tube, essentially consist in the adult state of a glandular epithelium arranged in various ways and supported by a connective-tissue stroma which carries vessels, lymphatics, and nerves. In structure, therefore, they correspond to the mucous membrane of the alimentary canal,

and, like it, they are subject, though much more rarely, to similar inflammatory changes. The communicating ducts of these organs with the mouth and duodenum respectively are lined by a mucous membrane continuous with that of the cavities into which they open. Catarrh of the ducts, usually caused by extension or by the irritation of calculi, may be acute or chronic, and may lead, by the retention of the secretions from the blocked lumen, to inflammation and other changes in the proper gland tissue.

The causes of inflammatory affections of the digestive organs are very numerous. Among those which may be regarded as predisposing are (i.) the exposure of the mucous membrane to injury or irritation from injurious ingesta; (ii.) age, which confers special liability, as witness the frequency of stomatitis and gastro-enteric catarrh in children; (iii.) season, such as extremes of heat and excessive moisture, which are most active in determining epidemics of gastro-intestinal inflammations; (iv.) morbid conditions of the mucous membrane, more especially chronic congestion and degeneration, which favour the development of the catarrhal state. Exciting causes are (i.) the nature of the ingesta, whether mechanically or chemically irritating, corrosive poisons, toxic micro-organisms, ptomaines, and other bodies which may occur in the course of mal-digestion; (ii.) catching cold; (iii.) dentition; (iv.) wounds or malpositions of the bowels, herniæ, &c., impacted fæces; (v.) extension from the peritoneum; (vi.) the irritation of new-growths; (vii.) secondary to many acute febrile states, and some chronic diseases, such as phthisis and diabetes.

Among the chief results of inflammation are:—

(a) *Abscess*.—This may occur in any part of the submucous tissue, in the so-called phlegmonous and pyæmic inflammations, but is of most common occurrence in the gums—gumboil, in the tonsils, and in the liver; often in the latter situation it is the result of inflammation determined by absorption into the mesenteric veins of septic particles from dysenteric ulceration.

(b) *Ulceration*.—The mucous membrane of the alimentary canal is particularly liable to this morbid process. Some preference is exhibited for the different forms of ulcer by certain regions of the canal, and a difference exists in the tendency to perforate the entire thickness of the tube, those of shorter duration frequently producing this result, whilst the chronic ulcers are usually accompanied by a slow formation of indurated connective tissue, which proceeds *pari passu* with the destructive process, and is especially likely to institute adhesions between the canal and adjacent organs. Ulcers are met with in the salivary, hepatic, and pancreatic ducts, very frequently as a sequence of in-

flammation determined by the passage of calculi.

The ulcers which are usually acute in their course are:—(1) Simple or catarrhal. (2) Aphthous. These forms, though they may occur in any part of the mucous membrane, are far more commonly situated on the gums, cheeks, tongue, and palate. (3) Acute specific ulcerations: as diphtheritic and scarlatinal, mainly affecting the fauces; or typhoid, limited to the jejunum and ileum, and originating in the solitary and agminated glands. (4) Dysenteric.

The ulcers that are commonly chronic in their course are:—(1) Gastric. (2) Tubercular, which may occur in any part of the canal, but are usually limited to the same situation as the typhoid. (3) Syphilitic, most common in the mouth, fauces, and rectum. (4) Cancerous. (5) Dysenteric and chronic catarrhal.

(γ) *Sloughing and Gangrene*.—The inflammatory state may be so intense as to lead to molar death of the area affected, with the production of slough. This often follows scarlatinal inflammation of the fauces, and the surface of the large intestine in dysentery is frequently covered by large and numerous sloughs.

Gangrene is almost entirely limited to the mouth in children, when it produces the condition termed noma. The cheeks are usually affected first, the process rapidly involving the gums, jaws, &c. Portions of bowel that have been strangulated readily become gangrenous.

*Post-mortem* softening and destruction of the stomach and intestines is frequently met with, and is due to an actual digestion of the viscera by the gastric juice, which, thus escaping from the stomach, may cause destruction of adjacent organs. It is usually met with when death has occurred during the process of gastric digestion, and is more common in infants, possibly from the greater acidity of the products of digestion.

2. *Hypertrophy*.—A general overgrowth of the normal tissues of the digestive organs is practically unknown. Certain parts may manifest this condition, notably the muscular tissue of parts of the canal above an obstruction. Patches of hypertrophied epithelium are seen in the mouth following on persistent mechanical irritation of broken teeth, &c., or as the result of syphilis. The overgrowth may be limited to the epithelium of the lingual papilla. Considerable hyperplasia of the sub-epithelial connective tissue may be caused by a chronic inflammation of any part of the canal. The liver is described as being occasionally hypertrophied in certain cases of diabetes.

3. *Atrophy*.—The alimentary organs may share in the general atrophy and wasting of old age or inanition. This condition is apt to follow disease of certain parts, especially the epithelia in chronic inflammation, or

may be seen in the thinning and shrinking of the stomach and intestines beyond an obstruction or an artificial anus, or when these organs are permanently dilated. Pressure on the organs, as by tight-lacing, &c., may lead to the same result.

4. *Degenerations.*—Those morbid processes to which the term ‘degeneration’ is applied, and which essentially consist in the conversion of the tissues into materials of a less complex chemical composition than normal, associated with a diminished vital activity, may affect any or all of the structural elements of which the digestive organs are composed. Albuminoid infiltration or cloudy swelling is the invariable accompaniment of inflammation of the epithelial and muscular tissues. Fatty degeneration is a further result of inflammation or of other malnutrition, and has been found in the gastric mucous membrane, as well as in the muscular coat at various parts of the canal. Although not the commonest organs to be so affected, yet not infrequently the intestine and stomach are the seat of lardaceous degeneration, not always limited to the vessels, but affecting also the epithelial, fibrous, and muscular coats. The liver is especially liable to undergo degeneration, both fatty and lardaceous.

Deposition of pigment may be found in the deeper epithelial strata of the mouth and intestines in Addison’s disease, in the gastric and intestinal mucous membrane as the result of changes in effused blood associated with chronic inflammation, and in the liver in certain cases of blood diseases and intermittent fevers. The sulphides of such metals as lead may be deposited in the mucous membrane of mouth or intestines in lines and spots of black or bluish appearance.

5. Changes in or retention of the secretions of the various glands may result in the production of *calculi*—salivary, pancreatic, or biliary.

6. *New-growths.*—There is scarcely any known form of neoplasm which may not be found in some region or another of the alimentary tract.

The new-growths limited to the epithelial coat, or commencing in it, are condylomata, papillomata, and various forms of carcinoma.

In the fibrous tissue occur sarcoma, fibroma, myeloid, adenoid, gumma, enchondroma, and lipoma.

In addition there may be polypi or tumours of the mucous membrane; myxoma; muscular-tissue tumour; cysts; vascular growths, such as nævi and hæmorrhoids; and the infective granulomata, viz. tubercle, lupus, and syphilis.

7. *Traumatic.*—Certain parts of the alimentary tract are, from their position, more liable than others to external injury. Incised and punctured wounds of the mouth, œsophagus, stomach, intestines, and liver are of occasional occurrence, and rupture of the

abdominal viscera is sometimes met with. The ingestion of corrosive substances may produce destruction of certain parts of the canal, and wounds may be determined by foreign bodies, as pins, fish-bones, &c., which have been swallowed.

III. *Malformations and Malpositions.*—1. *Hereditary.*—Of these the most important are hare-lip; cleft palate; macroglossia; fistulous communication between the pharynx and the exterior, or between the gullet and trachea; intestinal cæca; imperforate anus; and herniæ.

2. *Acquired.*—Malformation and malposition of the viscera may follow from disease. Communications between the stomach and intestines, or between different coils of intestine, or between the gall-bladder and the gut, may result from chronic ulceration. Many herniæ are not developed until long after birth, from violent strains, &c.

Stricture of various parts of the canal is frequently associated with the healing of ulcers, and with new-growths. Dilatation of the canal is apt to occur on the proximal, and contraction on the distal side of such strictures. The intestines may be considerably displaced from adhesions following peritonitis. Twists (volvulus), intussusception, internal strangulations, and prolapsus ani, are more or less common affections of the intestines.

IV. *Abnormal Contents.*—*Concretions.*—These are found occasionally in the sacculæ of the large intestine, cæcum, appendix, and, more rarely, stomach and small intestine. They usually are made up of concentric layers of earthy and organic matter, the former being principally phosphates of lime and magnesia. They frequently have as a nucleus some foreign body. Similar bodies have been met with, formed of chalk, magnesia, or oxide of bismuth, which have been swallowed. Much larger masses, mainly composed of densely felted hairs or vegetable husks and fibres, with earthy and fecal matter, are sometimes seen; gasteroliths are generally of this variety (see CALCULI). Foreign bodies, such as pins, bones, fruit-stones, coins, &c., may also lodge in the alimentary canal.

*Parasites.*—The alimentary organs are infested by numerous forms of micro-organisms, belonging for the most part to the vegetable kingdom. The greater number are comprised in the Schizomycetes, or bacteria, but a few, such as the Actinomycetes, found in the mouth and liver, are included in the division Hyphomycetes, or moulds; and as an example of the Blastomycetes, or yeasts, may be mentioned the thrush-fungus, *Saccharomyces albicans*.

These all obtain entrance to the canal from the exterior, chiefly with the food, and the majority give rise to no symptoms of disease provided proper cleanliness be observed. That many propagate and flourish in different parts of the tube is certain, and some appear to take a definite share in converting the food

to a diffusible state, especially in the intestines, and to that extent are true digestive agents. The ferments which set up the lactic-acid change in the stomach are to be considered as normal, whilst those which develop butyric acid are clearly abnormal. The true pathogenic organisms, such as those producing tubercle, typhoid fever, diphtheria, and cholera, develop as they may find their surroundings suitable; but it is also probable that some forms of gastro-intestinal disturbance, such as summer diarrhoea of infants, are due to the activities of bacteria, which produce toxic substances—ptomaines—in the course of putrefactive changes set up by them in the intestinal contents.

A few species of Protozoa have been met with in the intestines, such as *Amœba coli*, *Paramœcium coli*, *Cercomonas intestinalis*, and *Psorospermia*. The other and larger parasites belong to the flat or round worms, and include the various species of *Tœnia* and *Bothriocephalus* among the former, *Ascaris lumbricoïdes*, *Oxyuris vermicularis*, *Trichocephalus dispar*, and *Ankylostoma duodenale* among the latter. See ENTOMOZOA.

*Gases.*—The gases normally occurring in the stomach or intestines, whether they be swallowed with the food or formed during fermentation in the canal, are often much increased in amount, especially carbonic acid, sulphuretted hydrogen, and marsh gas, the last named being inflammable, and occasionally eructated in considerable quantity.

W. H. ALLCHIN.

### DILATATION (*dilato*, I enlarge).

*ÆTIOLOGY.*—Dilatation of any of the cavities, tubes, or orifices of the body may either result from increased pressure from within, or from diminution in the resisting power of the walls of the tubes or cavities. These two causes are frequently combined, and, indeed, the latter is often the result of a long continuance of the former. Increased pressure from within may be due either to increased secretion of the normal contents of the cavity, or to some other effusion into it. This is the usual cause of dilatation of the closed cavities of the body; we have examples in the ventricles of the brain, the pericardium, the synovial cavities, the bursæ, the follicles of the thyroid body in cystic goitre, and the Graafian vesicles in some forms of ovarian dropsy. In the various tubes of the body, increased pressure from within may arise from obstruction, and the consequent accumulation behind the seat of obstruction of the substances which it is the function of the tubes to transmit.

*VARIETIES AND CHARACTERS.*—1. *Cystic dilatation.*—In tubes which begin by blind extremities the result of dilatation is generally the formation of a cyst, and this is the usual mode of origin of the large class of *retention-cysts*, or *cystic dilatation*. We have ex-

amples in the sebaceous cysts, in the cysts of mucous membranes, due to the ducts of the mucous glands becoming obstructed by the products of catarrhal inflammation, in cysts of the kidney formed by dilatations of the Malpighian capsules and uniriferous tubules, and in dilatations of the gall-bladder, and of the pelvis of the kidney.

2. *Uniform or cylindrical dilatation.*—In tubes not beginning by blind extremities, the effect of the obstruction is usually to produce a *uniform* or *cylindrical*, and not a cystic dilatation; though sometimes one part of the wall will yield, and so cause a diverticulum or sacculus. These uniform and cylindrical dilatations may occur in all the tubes of the body. They are met with in the œsophagus and all parts of the digestive canal, in the heart, veins, bladder, ureters, bile-ducts, &c. This form of dilatation may be attended either with thickening and hypertrophy, or with thinning and atrophy of the walls. Usually when the tubes are in the main muscular, hypertrophy occurs, from increased exercise of the muscular fibres in their efforts to overcome the obstruction; but when the walls are mainly fibrous or elastic, they generally become atrophied and thinned.

3. *Compensatory or collateral dilatation.* Another form of dilatation from increased internal pressure may be termed *compensatory* or *collateral* dilatation; it is produced by the tubes having to transmit an increased quantity of fluid in consequence of the obstruction of other channels. Besides the blood-vessels, we may meet with examples of compensatory dilatation in one ureter when the other is blocked, and in the bronchial tubes and other parts. Resembling this form in its mode of origin is the dilatation caused by tubes having to transmit substances of too large a calibre, as, for example, in the passage of calculi down the gall-duct and ureters.

4. *Dilatation from changes in the walls.* The last class of dilatations consists of those due to diminished power of resistance in the walls of the tubes or cavities. The most important examples of this class occur in the circulatory and respiratory systems: in the heart from fatty degeneration; in the arteries from atheromatous changes. In the respiratory organs it occurs both in the bronchial tubes and in the air-cells. Here, however, the loss of resisting power is itself usually caused by prolonged increased pressure from within; which in the air-cells, as their walls are elastic and not muscular, rapidly causes atrophy, and subsequent dilatation.

W. CAYLEY.

### DILUENTS (*diluo*, I wash or dilute).

*DEFINITION.*—Remedies which increase the proportion of fluid in the blood.

*ENUMERATION.*—Water is the only real diluent. It is given for this purpose in various

forms—soups, *tisanes*, barley water, toast and water, milk, lemonade, aerated waters, &c.—to quench thirst, and increase secretion.

USES.—Diluents are employed to lessen thirst, as in fever and diabetes, and to remove the products of tissue-waste. As the thirst may depend upon local dryness of the throat, as well as upon general want of fluid in the system, the power of water to quench thirst may be greatly increased by adding to it a little vegetable or mineral acid, or some aromatic, such as lemon or orange peel, which will stimulate the flow of saliva, and thus tend to keep the mouth moist after the liquid itself has been swallowed. The thirst-quenching power of water is also aided by the addition of mucilaginous substances, such as oatmeal, or linseed tea, which, leaving a mucilaginous coat on the inside of the mouth and pharynx, retard evaporation, and thus lessen the dryness of the mucous membrane. The free use of water, and especially drinking hot water on rising in the morning, or on going to bed at night, and between meals, is useful in gout, chronic rheumatism, and biliary lithiasis, lessening or preventing the occurrence of acute attacks.

T. LAUDER BRUNTON.

**DIPHThERIA** (*διφθέρη*, a skin).—  
SYNON. : Fr. *Diphthérite*; *Diphthérie*; Ger. *Diphtheritis*; *Diphtherie*.

DEFINITION.—An acute, specific, contagious and infectious, and often epidemic, general disease; accompanied by pyrexia and great weakness; having as its local manifestation inflammation of various mucous membranes, particularly those of the throat and larynx, and the formation upon them, or upon external wounds, of ulcers covered by a fibrinous false membrane; and often followed by paralysis of varied distribution.

HISTORY.—From the writings of the most ancient physicians, it is evident that the disease we know as diphtheria existed in the earliest ages, and then had main features differing little, if at all, from those with which it presents itself to us now. Hippocrates, Aretæus, Galen, Celsus, and Aëtius, have all left fragmentary accounts of a disease prevalent in their time, which serve for its identification with diphtheria. From the sixteenth century onwards, since which time medical observation has been more fully recorded, we have reliable descriptions of epidemic diphtheria occurring at times in various parts of Europe, which leave no doubt as to the nature of the disease, and show that essentially it had not changed for centuries. Bretonneau, in a work published in 1826, gave an account of his experience of the disease, beginning with an epidemic in Tours in 1818; and from this description dates the copious modern literature upon the subject. Bretonneau, too, gave the modern name to the disease, by calling it *diphthérite*, and later *diphthérie*, in allusion to the production of

membranes or skins—a special character of the malady. The most severe outbreak of diphtheria which has occurred in England was that which attained its height about the year 1858. But while later epidemics may not have been so severe as this, their occurrence has become more frequent, and, as will be discussed in the section on Pathology, the disease is now endemic in our large towns. In the article upon CROUP, the writer has given his reasons for believing that at least a large majority of cases classed under this head are really true diphtheria having its primary seat in the larynx. But membranous croup and diphtheria are even yet considered by some physicians as distinct diseases, and in former times this view was much more prevalent. Consequently, many of the outbreaks of 'croup' in Scotland and England must be considered as epidemics of diphtheria. It is noteworthy, too, that the recent returns of the Registrar-General show an increase in the number of cases of diphtheria and a decrease in those of croup, doubtless from the recognition of most cases of the latter disease as examples of laryngeal diphtheria.

INCUBATION.—After exposure to the poison of diphtheria a certain interval elapses before definite phenomena of the disease appear, whether these be of the nature of general symptoms or the beginnings of the local lesion. This period of incubation varies greatly. It is much shorter after direct inoculation than when the poison is taken into the system by other channels. When animals are inoculated with diphtheritic poison, signs of the disease may appear in about twelve hours, or may be delayed as long as three days. In the human subject such inoculation occasionally takes place, as when diphtheritic membrane comes in contact with an open wound in another patient, or when instruments which have been used for an operation upon a diphtheritic subject have not been thoroughly cleansed and disinfected before being used for other purposes. In such cases the period of incubation resembles that seen in the experiments upon animals, but may be prolonged to four days. When, however, the poison is received in other and more indirect ways, the incubation time is much more variable, and probably its ordinary limits are from two to eight days. Yet certainly the lower limit is frequently anticipated, for there is no reason to doubt the accuracy of many published cases, in which the disease began a few hours only after exposure to infection. Cases are on record, too, where the incubation seemed to have been much longer, but these are more doubtful. Not only may it be suggested that a second exposure had occurred, but it is likely that, where this can be disproved, the poison was not received into the body at the time of exposure, but was held by fomites

until a period much nearer to the outbreak, when the patient was in a more favourable state to receive the disease.

A difficulty in determining the period of incubation is caused by the invasion of the disease being not infrequently insidious. During the incubation, while there may be no symptoms at all, there may be mere indefinite feelings of malaise, not to be distinguished from the early symptoms of invasion in a mild attack.

There is reason to believe that the more intense the poison, or the more liable the patient, the shorter is the period of incubation.

**SYMPTOMS.**—*Invasion.*—According to the severity of the disease in its clinical aspects, three separate types of diphtheria may be distinguished—the benign, the ordinary, and the malignant. These are not natural orders, but relative terms. Numerous cases occur which throughout their course may be classified as belonging to one or other of these groups. Yet, on the other hand, instances are also numerous which show a transition from one to the other group, which, beginning, for example, after the benign type, afterwards show the most serious symptoms or sequelæ of the disease; or which, presenting only ordinary symptoms at the onset, rapidly assume the features of the malignant type.

1. It will be convenient to describe in the first place the symptoms of the disease as it is *ordinarily* met with.

In such cases, after a period of incubation, the patient, as a rule gradually, but often almost suddenly, feels ill. He has wandering aches and pains, disinclination for exertion, slight headache, chilliness, loss of appetite, nausea—and rarely even vomiting, thirst, and the general discomfort of a febrile condition. The temperature of the body is raised, but not to a great degree—in most cases, at the onset, to not more than 100° or 101° F. At this time, however, the local symptoms of the disease appear in the form of soreness and tenderness of the throat, and slight enlargement of the glands at the angles of the jaws. The fauces, tonsils, soft palate, and pharynx are swollen, and show a diffuse dusky redness. The pulse and the respiration are hurried, in accordance with the general febrile state. The urine presents the usual febrile condition.

The symptoms rapidly increase in severity. The bodily weakness becomes extreme, and anæmia soon presents itself. The glands, not only those at the angles of the jaws, but their lymphatic connexions also, become greatly swollen, painful, and tender; and their covering of skin may be reddened. The pulse is frequent, feeble, and of low arterial tension; the respirations are more hurried. The local disorder now becomes characteristic. The tonsils themselves assume a paler aspect, but are surrounded by the reddened mucous

membrane. Soon a white haziness appears in patches on the tonsils, the soft palate, or the pharynx. While the swelling of the parts already mentioned greatly increases, the haziness becomes more pronounced, until in disseminated patches a distinct false membrane is seen, slightly raised from the surrounding parts, yellowish-white in colour, ragged in outline, and surrounded by a zone of congestion distinguishable even from the general redness of the neighbourhood. At first the membrane can be detached from the underlying parts; and when removed it leaves an intensely red surface behind. But very soon it becomes more adherent, and its removal is only attained by laceration of the mucous membrane and the exposure of a bleeding ulcerated surface, on which a new layer of false membrane is formed in a short time. The membrane spreads, and may cover the whole of the fauces and pharynx. When this occurs there is usually so much swelling of the parts that scarcely any opening of the fauces can be seen. Further, the aspect of the membrane changes. From being yellowish-white, thin, and delicate, it becomes thicker, of a brownish tint, firm and leathery in texture. The difference in colour is due to admixture with decomposed blood-pigment, the entanglement of atmospheric particles, and drying of the exudation. Signs of coryza are present, and from the nostrils a thin acrid discharge issues.

The tongue is dry, coated with a white or brownish fur, and red at the tip. Appetite is entirely lost, and as a rare symptom there may be vomiting. Swallowing is difficult and painful. The bowels are, as a rule, constipated. The temperature becomes higher, but does not attain the extreme degree seen in many other fevers. Its ordinary height is between 102° and 103° F. The examination of the chest at this time, provided the case is uncomplicated, reveals either nothing at all, or only the rhonchi and râles of bronchial congestion. The urine is still febrile, and in a large number of cases contains albumen. Slight delirium may occur, but is not a frequent symptom in the ordinary type.

When such cases tend to a favourable issue, the duration of the disease is variable. Even after such a moderately severe form as that just described, the symptoms may begin to decline at about the fourth day; the patches of false membrane, after separating, cease to re-form; and the patient rapidly resumes his ordinary health. Usually, however, both the general and the local symptoms are prolonged for a much longer period, say up to the twelfth or fourteenth day, when slow convalescence begins.

Such is a description of the form in which diphtheria ordinarily presents itself, without any of the numerous complications which may occur. Many of the symptoms, how-

ever, require a more detailed description, and this will be given below.

2. The *benign* form is so mild that a considerable number of patients pass through it with so little feeling of illness that they consider it unnecessary to consult a medical man at all. There can be no doubt that such ambulant cases are important factors in spreading the disease. The patient suffers from but slight malaise and weakness, with loss of appetite and slight fever. The urine is usually not albuminous. No complaint whatever may be made of soreness of throat; and for this reason the nature of the complaint is frequently overlooked, even by medical men. Generally, however, there is some soreness in swallowing, and a little enlargement of the glands at the angles of the jaws. On examination, a few white patches of false membrane, easily detached, will be seen on the tonsils, rarely on the soft palate or pharynx. Rarely, with only mild general symptoms, very extensive deposit may be seen in the throat. In a few days the patches disappear, either by being coughed out or by gradual dissolution; no more form; the symptoms, slight as they were, decline; and the patient rapidly resumes his usual health. During the whole period of the disease it is possible for the patient to walk about, and even attend to his usual duties. But this is very undesirable, not only because such cases are as capable of spreading infection as more severe ones, but because, however slight may be the condition at the onset, at any time more serious symptoms may arise, and in this, the benign, form there is no exemption from the most dangerous complications and sequelæ of the disease, such as spread of the membrane to the larynx, cardiac failure, and paralysis.

3. The *malignant* form of diphtheria is characterised by severity rather of the general symptoms than of the local lesions. Commonly, from the first the symptoms are severe, though the early features of the case may be those of the ordinary or even of the benign form. There is extreme prostration; the pulse is rapid, feeble, and of low tension; and the respirations are hurried and shallow. The skin is dry, pungent to the touch, and of a generally dusky hue. The tongue is dry and brown. Very soon the 'typhoid' state develops, and there may be sometimes a muttering delirium, though in most cases this is absent. A hæmorrhagic diathesis is a frequent phenomenon in this condition, and is shown by the occurrence of hæmorrhages from various mucous membranes, or beneath the conjunctiva, and by the appearance of a purpuric rash on the skin. The temperature may be highly febrile, or not unduly raised. The urine is albuminous. While the symptoms of the throat-lesion are not very evident, yet its signs are characteristic. The false membrane is soft, pultaceous, and very dark—

sometimes black, in colour. It is often extensive; and the tissues around are sloughing or gangrenous. A foul smell comes from the putrifying mass at the back of the throat. In most cases, too, the false membrane has spread to the nose; and a sanious discharge, also of foul smell, issues from the nostrils, and by its irritation excoriates the skin around with which it comes into contact. The glands of the neck are always much enlarged and inflamed. Such a condition in most cases ends fatally in a short time.

This general description of the three types of diphtheria requires to be supplemented by a detailed account of some of the symptoms.

*Formation and Spread of the False Membrane.*—The presence of the false membrane is the characteristic feature of diphtheria, and that from which it derives its name. It is the special local manifestation of the disease. But, as will be clear from the descriptions given, in diphtheria we have to deal with general symptoms which, in point of severity, may have no relation to the extent of the local lesion. In the benign form we may sometimes find the general symptoms very slight, or practically absent, while a large quantity of membrane may be seen in the throat. In the malignant form we may have the most severe general symptoms when the local phenomena, severe it may be, are not so prominent as the signs of general disease. The question then arises whether we may occasionally meet with general symptoms of diphtheritic fever, not only without signs of any local lesion, but without the appearance of any false membrane, and with merely congestion and swelling of the fauces, having no specialised character. It is in the experience of clinicians that this occurs, but certainly very rarely. It is possible that, in the course of an epidemic of diphtheria, instances of the benign form may be met with without more local lesion than the early congestion described; yet in such cases a slight formation of false membrane may easily be overlooked. Because of the existence of such a condition, Virchow originally described a catarrhal form of diphtheria, in which no membrane at all was formed. But it is beyond doubt that the poison of diphtheria may act so powerfully upon the system as to, in extremely rare cases, kill the patient before the local lesion has time to develop. It is only such cases as these which can without fear of error be used for argument; but they have been recorded by such competent observers that it must be admitted that diphtheria may at times occur without any specialised local lesion.

In by far the majority of cases, however, false membrane does appear. Generally it occurs first on some part of the throat—either on the tonsils, soft palate, or the back of the pharynx. Usually, too, it is seen in one or more spots simultaneously on both sides, and

most commonly on the tonsils. Yet, occasionally, at an early period but one side may be attacked, but soon, however, an affection of the opposite side follows. The membrane can, at quite its early appearance, be detached with but slight difficulty, leaving a very red or hæmorrhagic surface behind. But very soon its connexions to the subjacent parts become much firmer; it can be removed only by some force, and leaves behind a rough, bleeding, and ulcerated surface. In all but extremely slight cases, a new formation of membrane then occurs; and this may be repeated many times if the disease continues. The detachment of membrane also takes place naturally, not only at the termination of the case, but during its height, new membranes being again formed. In this way, the patient may expel from the mouth, usually by coughing, large quantities of the false membrane, in small or large shreds. Where the larynx and trachea are attacked, complete casts of these cavities may be thus expectorated; and in still more serious cases even moulds of the smaller bronchi are occasionally coughed up.

In the early stages, the membrane appears as a thin whitish deposit, which rapidly thickens. The colour, too, soon changes, passing through stages of grey, greyish-yellow, yellow, brown, and even black. Its consistence increases, becoming in most cases very firm, and resembling shreds of kid-leather. In the malignant form, the membrane becomes softened, and even pultaceous. Its colour then is still darker, and the membrane becomes mixed with blood or the products of its decomposition.

The isolated patches in which the membrane first appears soon spread and amalgamate, until the whole of the throat, as seen on opening the mouth, is entirely covered. But the affection spreads farther. One of the most dangerous complications of diphtheria, and also one of the most frequent, is the spread of the membrane to the larynx. This is more frequent in children; and, while dangerous in adults, is still more so in children, because of the greater ease with which it diminishes the lumen of the glottis. The signs by which the complication may be diagnosed are the following. The patient feels soreness in the larynx, and is greatly troubled by cough. The breathing becomes first wheezy, then stridulous, and always more hurried than before. The cough is harsh; the voice is hoarse and husky, and finally lost. The patient, if a child, clutches at the throat as if to remove an obstruction. Soon the narrowing of the larynx interferes greatly with the entrance of air into the lungs. All the extraordinary muscles of inspiration are brought into action. The lower ribs cease to move outwards, and later on are depressed at each inspiration. The supra-clavicular spaces are similarly de-

pressed. These signs are more obvious in children: in very young subjects, and especially in those affected by rickets, the whole chest-wall may be depressed as each breath is drawn. Signs of cyanosis may now be observed in blueness of the lips and cheeks; but more commonly the face is pallid. The pulse is very rapid. The temperature is no more raised than it was before, and may indeed be only slightly above the normal. The forehead, and even the whole body, becomes covered with a cold clammy sweat. If unrelieved by operation, this condition soon ends in death, occasionally preceded, in young subjects, by convulsions.

The opinions of observers of great experience vary as to the period of the disease at which extension of membrane from the pharynx to the trachea is most likely to occur. Sir William Jenner stated that he had never known this complication commence after the expiration of the first week of the disease. Oertel, on the other hand, speaks of its occurrence as late as the thirteenth day; and Hensch saw it begin fourteen days after the commencement of the primary disease. Sir William Jenner's statement, however, is correct for the majority of cases; and we may place the usual time of appearance of secondary laryngeal symptoms at three to six days after the onset of the diphtheria. It is rather in the mild and in the ordinary forms than in the malignant that this complication may be looked for. Yet its appearance is a sign of extreme danger; most of the deaths from diphtheria are due to this cause. Sir William Jenner again states that he had seen death occur twelve hours after the onset of laryngeal symptoms, and had never known it delayed more than five days; and his experience agreed with that of Bretonneau.

The membrane frequently spreads to the nares also. This is almost invariably the case in the malignant, but occurs, too, in the ordinary form. The symptoms of the complication are snoring or snuffling respiration, and the discharge of an acrid fluid from the nostrils, which excoriates the surrounding skin. The excoriations may in their turn become covered by diphtheritic membrane when the disease is severe. Shreds of membrane, too, may be discharged from the nostrils. Epistaxis is of frequent occurrence. Sometimes, as stated in the account of malignant diphtheria, this is due to the general tendency to hæmorrhage found in septic diseases, but it is also a sign of invasion of the nasal mucous membrane. From the nose the lacrymal ducts, and even the conjunctivæ, may be infected, leading occasionally to destruction of the eyeball.

Spreading from the throat, the membranous formation easily reaches the upper pharynx, and by way of this proceeds to the middle ear. Great pain and sense of distension are thereby produced, and severe *otitis media*

with destruction of the parts, or even *otitis interna* and *externa*, and necrosis of the bone, may result.

The œsophagus, too, may be affected in the whole of its length, and even the stomach and intestines, as will be mentioned in the pathological description.

Sometimes false membrane may appear in various parts of the mouth, probably by the infection of abrasions of surface.

In most cases, as above described, the membrane makes its first appearance in the throat. Yet numerous cases are now on record in which it has first been seen elsewhere.

The primary laryngeal form of the disease is well recognised, and has been known by the name of Diphtheritic Croup. The general symptoms of the disease are then the same as in the ordinary form, but signs of the trouble in the larynx occur early and before any affection of the pharynx can be detected. With the laryngoscope, if the patient will allow of it being used, false membrane, having the ordinary appearance, can be seen covering the posterior surface of the epiglottis, the whole of the interior of the larynx, and even of the trachea. The affection may remain localised in the larynx until death, or may extend thence to the pharynx and other parts.

A primary nasal form is rare, but sometimes met with. Here the coryzal symptoms and the muco-purulent or sanious discharge from the nostrils occur early in the disease; and, when the affection is advanced, false membrane may be seen near the anterior nares. From the posterior nares the lesion may spread to the pharynx, as in the laryngeal form. Such cases are often overlooked in their early stages, because of the obscure position of the characteristic membrane. They are generally severe in character.

External wounds or abrasions may be secondarily infected and covered by false membrane in the course of the ordinary disease. But, on the other hand, there happen cases in which not only has the first local appearance of the disease been seen in an external wound, but the inoculation of such a wound has been the means of infecting the general system. Thus diphtheria has originated from the use of an infected instrument in the operation of circumcision, diphtheritic membrane first developing on the wound. Dr. Hill has recently described cases in which the primary lesion was on the perinæum. Dr. Braxton Hicks and others have also described cases where the first appearance of the false membrane was on some part of the female genital organs, doubtless as the result of accidental inoculation.

*Affection of the Glands.*—The glands beneath the jaw are swollen and tender in all but very exceptional cases of diphtheria. In

most they are so large as to be easily seen; in nearly all they can be felt. But in by far the larger majority of cases the swelling of the glands is a very marked feature. In severe cases, too, it is not only the glands beneath the jaw, but also all those of the neck, on both sides, though the affection of one side may predominate. The skin covering them is swollen, reddened, and œdematous. Sometimes, too, the tissue lying between the glands is greatly inflamed; and in a few cases a brawny hardness, extending from the jaw to the clavicle and involving apparently all the tissues of the neck, an *angina Ludovici*, has been observed. Rarely, the glandular inflammation may proceed to suppuration, causing a large and formidable abscess. The affection of the glands and surrounding tissue is more marked in the severe, and especially in the malignant forms.

*Condition of the Urine.*—The urine in diphtheria resembles in its general characters that of other febrile diseases, though the features are less marked than in other affections. It is diminished in quantity, high-coloured, and deposits urates and uric acid. Sometimes, too, it may appear but little changed. But a special feature of the disease is the occurrence of albuminuria, as first pointed out by Dr. Wade of Birmingham, in the *Midland Quarterly Journal of Medicine*, 1857. This appearance is very frequent, and is found, according to statistics, in one-half or even two-thirds of the total number of cases. The amount of albumen may be only slight, or, on the other hand, very great; and it may disappear from day to day, to again return. With it may be found in the urine hyaline or epithelial casts, but only very rarely blood, and the occurrence of albuminuria is in only extremely exceptional cases accompanied by anasarca or followed by any uræmic symptoms. Whether albuminuria is present or not, the daily excretion of urea is normal, or increased. It differs markedly from the albuminuria of scarlet fever, in making its appearance nearly always during the acute course of the disease: very seldom at the time of convalescence. In most cases it appears on the third or fourth day. Sanné, amongst others, has made extended investigations into this complication. He examined, in reference to albuminuria, 224 cases; and found that it appeared on the first day in three, on the second day in ten, on the third and also on the fourth day in thirty, on the fifth day in twenty-two. After the fifteenth day albuminuria occurred only once for the first time. As a general rule, the greater the quantity of albumen in the urine, and the earlier its appearance, the more severe is the case; but the mere presence of albuminuria does not affect the prognosis of the diphtheria. In most cases the albumen disappears during convalescence; though, as the writer can confirm, it may continue in the urine for months in moderate amount, and

unaccompanied by other symptoms, beyond slight weakness and anæmia. On the other hand, the writer has reason to believe that in exceptional cases chronic nephritis may have its origin in an attack of diphtheria.

*Temperature.*—The temperature in diphtheria has no definite course. It rises suddenly early in the disease, and as a rule continues, with morning remissions, until the beginning of convalescence, when it declines gradually. The average temperature at the height of the attack is from 102° to 103°, and very rarely does it exceed 104° F. It may become raised above the average limit by the advent of complications, such as broncho-pneumonia or suppuration in the neck; but, with these exceptions, the height of the temperature is no certain guide to the severity of the general disease or to the extent of the local lesion. In cases of prolonged diphtheria, such as will be noted below, after remissions or even intermissions of the raised temperature, renewed attacks of pyrexia are met with, generally associated with increase of the general symptoms.

*The Pulse and Heart.*—The pulse in diphtheria is throughout of an asthenic character, short, ill-sustained, and of low tension. In the majority of cases it is frequent, above the degree to be expected from the fever. Dr. Sidney Phillips, in the *British Medical Journal*, 1890, i. p. 960, has analysed 100 cases of diphtheria with reference to the states of the circulation which may occur. In three per cent. of his cases the pulse was unduly slow, and these instances were characterised by vomiting and by a fatal issue. He concludes that a very rapid pulse, also a very dangerous symptom, may occur without any other phenomenon, but may be accompanied by vomiting and extreme drowsiness. Irregularity both in the rhythm and the force of the pulse is not uncommon and not unfavourable. The heart-sounds are feeble, and the first sound short. Disturbances of the intervals between the sounds are, in the opinion of the writer, of great importance. A rapid sequence of the second sound upon the first, but especially 'spacing' of the sounds, as heard at the apex of the heart—that is, an equalling of the intervals between the first and second, and the second and succeeding first sound respectively—are of most serious import, and indicate a liability to sudden cardiac failure, the most dangerous complication of diphtheria. These alterations in the sounds of the heart may be detected both at the height of the fever and during convalescence, and are equally important at either time. Acute dilatation of the heart, usually of the right ventricle, sometimes of the left, is not infrequent in diphtheria. It may be accompanied by a systolic murmur, heard at the apex or over the area of the pulmonary artery. But such a murmur is not due to endocarditis. Bou-

chard and Labadie-Lagrave have maintained that endocarditis is a frequent complication of diphtheria, but their opinion has been supported by no other observers. The cardiac dilatation does not occasion dropsy, as it does in most other conditions, but the weakness of the heart's impulse is the cause of the thrombosis of the large veins which sometimes, though rarely, occurs.

*Rash.*—A certain degree of efflorescence on the skin is not uncommon in unmixed cases of diphtheria, and especially in the severe variety. It is usually seen about the neck and the upper part of the chest. It is a diffuse redness, not punctate, and not papular in the slightest degree. This is not a specific rash, and must be carefully distinguished from that of scarlet fever. To the writer, it has appeared to be somewhat darker in colour than the rash of scarlet fever. In the malignant forms purpuric spots of different sizes may be observed, scattered over the whole surface of the body in great numbers.

*COMPLICATIONS AND SEQUELÆ.*—Many of the complications have already been described in the foregoing account of the disorder, and such will be here only enumerated.

1. Extension of the false membrane to the larynx and lower air-passages, to the nose, ear, and eye.

2. Formation of false membrane on external wounds and abrasions.

3. Extensive ulceration, and even gangrene, of the tissues of the throat. Hænoch mentions perforation of the palate as an occasional result.

4. Great inflammation in the neck, and rarely suppuration.

5. Albuminuria.

6. Suppression of urine, and uræmia. This has been described by Oertel and by a few other observers, but its occurrence is contrary to general experience; and, as Dr. Hilton Fagge has remarked, it is probable that scarlet fever has been overlooked in such cases.

7. Dilatation of the heart.

8. Cardiac failure, syncope, and sudden death. This complication is the most dangerous which can occur in diphtheria, and may come on at any period of the disease, and even during convalescence. It is preceded usually by a very frequent, sometimes by a slow, pulse, but its special warning is an alteration in the sounds of the heart, as described above. The cardiac failure may in some cases show itself by a gradual failure of the circulation, but usually by sudden syncope, which is nearly always fatal. The most distressing cases in which this occurs are those where the patient, some time after the decline of pyrexia, is in full convalescence, or even in apparently good health, yet suddenly faints and almost instantly dies. No patient is absolutely safe from this fatal

mishap until at least six weeks after the apparent end of the disease.

9. Hæmorrhages. These may occur, not only from the ulcerated mucous membranes, but also from any mucous surface. They may be so copious as to seriously weaken the patient.

10. Thrombosis of veins.

11. Affections of the bronchi and lungs. General bronchitis may be met with in diphtheria before the larynx is attacked, and may be followed by extensive broncho-pneumonia. Both these complications are, however, more frequent when the air-passages become involved in the special diphtheritic inflammation. Collapse of lung, emphysema, and hæmorrhage into the tissues of the lung are also met with at times. Their special symptoms are added to those of the original complaint, and need not be here detailed.

12. Paralysis. Though paralysis may follow other febrile disorders, yet it is so frequently an association of diphtheria as to form a characteristic feature of the disease. The paralysis, as statistics show, varies in its frequency in different epidemics. Cadet de Gassicourt found that it occurred in 128 cases out of 937. Dr. Gowers states that its frequency has been reported as from 8 to 66 per cent., and that probably one-fourth of the cases which do not die from the primary disease suffer from paralysis. The paralysis as a rule appears about two or three weeks after the decline of the pyrexia, but it may occur during the height of the disease, even as early as the second day (Sanné), and it may be delayed until the fourth or fifth week from the attack. As Landouzy has shown, the older the patient the more liable he is to this sequela. It is more common after slight cases of diphtheria, but its onset is earlier in the severe forms.

The first part to be affected by the paralysis is the soft palate. The patient then speaks with a nasal voice, and is unable to swallow fluids, since the immobile palate no longer closes the entrance to the posterior nares, and so fluids regurgitate through the nose. The palate hangs motionless during respiration and phonation, and is insensitive to touch. Occasionally, and for a time only, one side of the palate remains unaffected, and then the uvula is drawn to one side and the curves of the palatal edges are unequal. In rare cases the inability to swallow extends to solids also, from paralysis of the muscles of the pharynx.

The next most common paralysis is that of the eye. The most obvious effect of this paralysis, and that which most readily attracts the attention of the patient's friends, is an external strabismus from paralysis of the internal rectus muscle. But this is not the most common result. More frequently we meet with a paralysis of the ciliary muscle, usually in both eyes, which has as its effect

a loss of the power of accommodation. Unless the patient has previously been myopic, he is now unable to adjust his eye for near objects, as, for instance, in the act of reading, while vision for distant objects remains unaltered. The pupil, too, is sluggish in contracting to light. Other muscles of the eyeball may suffer, so that various forms of squint are met with, and sometimes ptosis; while, in exceptional cases, one or even both eyes have become absolutely motionless from paralysis of all their muscles. Concentric contraction of the fields of vision has been described.

Other organs of sense suffer but rarely, but Dr. Gowers states that he has met with loss of taste, and loss of smell without loss of taste.

The limbs next suffer from the paralysis, and usually the lower before the upper limbs. The paralysis is generally preceded by tingling feelings, sense of 'pins and needles,' or actual pain, in the parts about to be attacked. Then gradually and slowly the paralysis sets in. It is but rarely complete, but always sufficient to render the muscles practically useless. The muscles are flaccid and soon begin to atrophy. The knee-jerks are lost; but this phenomenon may be met with, as Dr. Buzzard and Dr. Hadden have shown, early in the primary disease; and Dr. Angel Money has found that there may be a previous exaggeration of the knee-jerks. All reflexes are abolished. The affected muscles after a time cannot be stimulated to contraction by the faradic current, though they still retain their reaction to galvanism. Together with the paralysis, and sometimes without it, a certain amount of ataxy is not infrequently seen, most obvious in the legs, though also sometimes found in the arms.

Sensation, in one or all of its varieties, is usually lost to a greater or less extent, but not always in proportion to the loss of muscular power. The distribution of the anæsthesia varies. It may be spread over the whole limb, or appear only in patches. It is usually most marked in the fingers and toes.

These affections of the limbs are usually bilateral, though not necessarily equal on the two sides.

There is scarcely a part of the body which may not be affected by the paralysis, but other parts than those mentioned are comparatively rarely attacked, and only in severe cases. The intercostal muscles may be paralysed, and less frequently the diaphragm, so that respiration becomes difficult or even impossible. The head falls forward, if the patient is in the upright position, from weakness of the neck muscles. The muscles of the larynx are sometimes attacked. The muscles attached to the epiglottis cease to close the orifice of the larynx, and food may then reach the air-passages. More rarely, the muscles of the vocal cords themselves are

weakened or paralysed, and the voice is altered in quality or entirely lost. The bladder and rectum may be affected in severe cases, but generally they are unaffected. Amongst the muscles most rarely attacked are those of the face and tongue.

In the most severe cases it is possible for nearly all the muscles above-mentioned to be paralysed at the same time, so that the patient lies in bed utterly helpless. This condition, however, is extremely rare. More usually, the paralysis recovers in one part while it progresses in another, and as a rule does not recur in a part it has already left. A patient the subject of diphtheritic paralysis is in most danger of heart-failure, for this complication, while liable to set in during the height of the primary disorder, is specially prone to occur during paralysis. The nutrition of the body may fail from the inability to swallow food; and from food entering the larynx pneumonia may arise. In very rare cases death ensues from paralysis of the respiratory muscles. Yet all these dangers are comparatively rare; and usually the paralysis, however severe, after a time gradually and entirely disappears. According to Dr. Gowers, the average duration of the paralysis is from six to eight weeks in cases that recover, but it remains longest in the limbs, and may there last for as long as four or six months.

**TERMINATIONS.**—(1) *Resolution.*—When the attack ends favourably the fever declines by lysis; the membranes soften, separate, and do not reappear; the patient gradually recovers strength, and resumes his ordinary health. The duration of ordinary cases is about ten to fourteen days, but the attack may be prolonged by recrudescences to a much longer period.

(2) *Death.*—Death occurs in diphtheria (a) by gradual asthenia from mal-nutrition; (b) by rapid asthenia from excessive effect of the poison; (c) by cardiac failure; (d) by asphyxia from spread of the membrane to the larynx, or paralysis of the respiratory muscles; or (e) by any of the complications enumerated above.

**ANATOMICAL CHARACTERS.**—*False membrane.*—The naked-eye appearances of the false membrane have already been sufficiently described. When a section of the mucous membrane covered by the exudation is examined under the microscope, the false membrane is seen to consist of either threads or shapeless masses of fibrin, in which are embedded leucocytes, more or less degenerated, red blood-corpuscles, and shrivelled cell-nuclei. In the deeper layers of the false membrane the outlines of epithelial cells can be seen, showing their bodies swollen into a hyaline or granular mass; the nuclei are not visible; and these cells no longer easily take the ordinary stains. No definite line of demarcation between the false membrane

and the true mucous membrane can be seen: the one joins the other gradually and irregularly. The mucous membrane shows swelling and proliferation of its epithelial cells, and is infiltrated with leucocytes. Its blood-vessels are congested, sometimes thrombosed, and hæmorrhages are seen in the tissue. Numerous micro-organisms, of which a fuller account will be given in the next section, are found, not only in the false membrane, but in the tissue of the mucous membrane, its veins and lymphatics. In the later stages, further degenerative changes are found in the false membrane. It is then much more granular, as seen under the microscope; contains numbers of fatty globules and crystals of fatty acids and cholesterin; and swarms with the micro-organisms of putrescence. The false membrane consists in part of an exudation of fibrin elements, but mainly of the products of coagulative necrosis of the epithelial cells. Hence its close connexion with the mucous membrane.

*Glands.*—The swollen glands are, on section, soft, red, and pulpy, resembling splenic tissue. They show the changes of acute lymphadenitis, and contain micrococci. The lymphatics leading to them are also enlarged and inflamed, and contain micro-organisms. Spots of suppuration are occasionally, but rarely, found in the glands.

*Respiratory organs.*—The larynx, trachea, and bronchi may be covered by false membrane. Its characters here are slightly different from those found in the pharynx. It is thinner, somewhat softer, and more easily separated from its attachments. These characters are due to no peculiarity in the membrane itself, but probably to the thinner layer of epithelium from which in this situation it takes its rise. The membrane may be prolonged into the smallest bronchi which can be followed out. More commonly, however, after proceeding down the trachea a little way, it becomes soft, loose, and at last continuous with a thick purulent or mucopurulent exudation, which covers the mucous membrane of the bronchi, but is not attached to it. The lungs may be congested, or show the changes of broncho-pneumonia or of pulmonary apoplexy. Very frequently, too, emphysema or insufflation of the lungs is seen in patches.

The mediastinal glands may be swollen, and resemble in appearance those of the neck.

*Circulatory organs.*—The cavities of the heart, especially those of the right side, contain *ante-mortem* clots firmly adherent to the endocardium. Endocarditis is not found. The muscular tissue of the heart is nearly always softened, and may sometimes show carditis or fatty degeneration. In a few cases, as the writer has found, colonies of micro-organisms may be seen embedded in the muscular tissue. Frequently, and especially if there

has been difficulty of respiration, small punctiform hæmorrhages may be observed beneath the visceral layers of the pericardium and pleuræ. In the malignant form, the blood after death is found to be of a dark chocolate colour, and very fluid.

Pleurisy and pericarditis are only rarely seen.

*Digestive organs.*—The false membrane may be found extending down the whole of the œsophagus, and may affect the mucous membrane of the stomach, and even that of the intestines. The Peyer's patches and the lymphatic nodules of the intestines are swollen, and may sometimes show ulcerations. The liver is congested. The spleen is enlarged, but not excessively so, and on section is found to be softened and pulpy.

*Urinary organs.*—The kidneys, in all cases, show the change of their epithelial cells called cloudy degeneration. When albuminuria has been present during life, the kidneys present the usual appearances of acute parenchymatous nephritis. The organs are swollen, pale in colour, and soft on section. The cortical portion is unduly enlarged, and of a yellowish-white colour. On microscopic examination, the cells of the convoluted tubes, and also those of the glomerular and capsular epithelium are seen to be swollen, and often proliferated. The changes only differ from those of scarlatinal nephritis in being of a less intense degree, less hæmorrhagic, and in being present often in scattered patches rather than diffused over the whole organ. Sanné asserted that in most cases only one kidney was affected, and thus explained the absence of uræmia and dropsical symptoms. This assertion, however, is not confirmed by the observations of the writer or of others.

*Muscles and joints.*—The external muscles, and sometimes those of the heart, may show the waxy degeneration of Zenker. In rare cases the joints may be found inflamed.

*Nervous system.*—Associated with the paralysis, changes have been found in the spinal cord and the peripheric nerves. Degeneration and atrophy of the anterior cornua of the spinal cord have been observed by Drs. Abercrombie, Percy Kidd, Mott, and others, while extensive degeneration of the peripheric nerves was first clearly and fully demonstrated by Déjerine, an observation since confirmed by many others. The degeneration of the nerves is seldom found in the whole of their extent, and may attack the portions near the spinal cord or those more remote. The change corresponds in its distribution and degree with the region and severity of the paralysis. The affected muscles show granular and fatty degeneration.

*PATHOLOGY.*—The older discussions as to whether diphtheria was from the first a general disease with a local manifestation in the throat—comparable, indeed, to enteric

fever—need now be merely mentioned. It is at the present time beyond doubt that the primary local lesion in diphtheria is due to changes at the site of inoculation, and that from this are absorbed the poisonous matters which give rise to the general symptoms. In the false membrane there are found swarms of micro-organisms, most of which, however, are either entrapped by the fibrinous exudation or are the accompaniment of the putrefactive changes going on in the membrane and in its vicinity. Such adventitious organisms are found only in the superficial layers of the false membrane, except in the malignant form, where they may be met with in the deeper tissues, in the thrombosed vessels, and in the lymphatics and glands. In 1883 Klebs discovered a bacillus in the deeper layer of the membrane, which he believed to be the specific organism of the disease. Its relation to the disease was afterwards fully worked out by Löffler in 1884, who showed that its cultivations, when injected into guinea-pigs, produce changes at the site of the inoculation identical with those found in the throat of man, and afterwards the general symptoms of the human disease. Further progress in this direction has been made by Dr. Klein, who, in the *Report of the Medical Officer to the Local Government Board for 1889*, has shown that the bacilli described by Klebs and Löffler are of two different kinds, which he has separated. Both are found in all parts of the diphtheric membrane, but are free from other organisms in the deeper layers. One of these bacilli, styled by Dr. Klein the pseudo-diphtheric bacillus, is not constant in its presence, and can only be cultivated at a raised temperature. The other, asserted by Dr. Klein to be the true bacillus of the disease, grows on gelatine at the ordinary temperature of a room, and has other technical differences which serve for its separation. It grows readily in milk. These bacilli are only found at the site of the local manifestation of the diphtheria and in the neighbouring glands; never, except in cows, in the blood or deeper tissues; so that if they are the cause of the disease, the general symptoms must be due to poisoning by some chemical product of the growth of the bacillus. This poison, or what is believed to be such a poison, has been separated from growths of the bacillus by Roux and Yersin, Fränkel and Brieger, and others; and by means of its inoculation they were able to produce the general symptoms of the disease. The theory, then, which at present best explains the pathology of the disease, is that we have an infection of the throat or of a superficial wound by the diphtheria bacillus, which there takes root, and by the products of its growth infects the whole organism. How its poison produces the general symptoms of the disease we have

at present no theories to explain. Recently, experiments have been made by Behring and Kitasato, Brieger and Fränkel, and Löffler, as to the possibility of weakening the action of the bacillus by various methods, and so conferring immunity against its further action; but, so far, no results have been obtained which are of use in human therapeutics.

The pathology of the cardiac failure so often met with is also obscure. It may be due to actual poisoning of the heart-muscle by the virus of the disease; and this may be the process at work when this accident occurs in the early stages of the fever. The stoppage of heart, though in regard to its symptoms a sudden phenomenon, may be really due to a gradually accumulating weakness. Dr. Sidny Phillips has shown that from cardiac weakness there may be a gradual filling up of the right ventricle by thrombus, accompanied by symptoms scarcely appreciable in the midst of the general febrile disturbance. Sudden death, however, will occur when in the right ventricle and pulmonary artery there is not left sufficient room for the circulation to be carried on. In the later stages, the cardiac failure is probably due to changes in the pneumogastric nerve or its central connexions—changes, however, which as yet have not been demonstrated.

The changes in the nerves have been found to be parenchymatous in nature, except in one case reported by Meyer, where nodular enlargements were seen, due to infiltration of the nerve-sheath. Though the changes are generally described as due to 'parenchymatous neuritis,' there is no proof of their being inflammatory, and the appearances observed are degenerative. In some cases of post-diphtheritic paralysis there is great difficulty in tracing the affection to an antecedent sore-throat, and the question has arisen whether the sequelæ may occur without any specific local lesion. That there may be a non-membranous primary diphtheria has been argued above, and this may be followed by paralysis. A remarkable series of cases illustrating the difficulties in which the physician may be involved, is recorded by Boissarie in the *Gazette Hebdomadaire*, 1881, p. 310, amongst which, during an epidemic, the first symptoms observed were those of paralysis in many cases, in some followed by angina, whereas, in succeeding cases, the disease had its accustomed course.

The albuminuria may be due to several causes. In many cases, of course, the organic changes in the kidney sufficiently account for the symptom. But in other cases, and especially in those where it is an early symptom of the disease and quickly disappears, it would appear to the writer to be due to a disorder of the circulation dependent upon the weakness of the heart's action. This may be combined with organic change, as it is in all cases of Bright's disease, but the essential factor in

the production of the albuminuria is the disorder of circulation.

*Mode of access of the poison to the system.*—Diphtheria is eminently a contagious disorder. A large number of the cases can be traced to actual contact with diphtheritic membrane or the secretions therefrom. Thus, surgeons who, after performing tracheotomy, have sucked out the secretion which blocked the tube, have very frequently contracted the disease, as have also relations who have kissed a patient. The cases alluded to earlier in this article, in which wounds have been inoculated by diphtheritic poison, are also demonstrative of this fact.

But diphtheria is also infectious, as is shown by its spread from one district and from one country to another. It occurs most frequently in epidemics, and yet in the present day it is so endemic in our large towns that they are never free from it. It forms a large item in the regular annual bills of mortality.

Its spread seems to have no connexion with the water-supply. But instances are now numerous in which outbreaks of diphtheria have been associated with a particular supply of milk. Here we may not only have to deal with a mere transference of the poison from man to man, milk being merely the vehicle, as is the case with scarlet fever, but the question also arises whether there may be a disease in cows which is identical in pathology with human diphtheria and capable of producing the latter. Dr. Klein's recent observations are of great importance in this connexion. During epidemics, it had been noted in a few instances that the cows, from which the infected milk had been derived, had had chaps or sores on the teats. Dr. Klein inoculated healthy cows with cultivations of his diphtheria bacillus, and in consequence of this observed not only a local affection at the point of inoculation, and general symptoms too, but also an eruption on the teats and udders. He also succeeded in obtaining cultivations of his diphtheria bacillus from the milk of such a cow, and, further, an outbreak of diphtheria amongst the cats of the Brown Institution was traceable to two cats who had been accidentally fed on the milk of the infected cows. These observations are extremely suggestive, and, if confirmed, will throw great light upon the ætiology of diphtheria.

Medical men have, at different times, attributed outbreaks of diphtheria in man to infection or spread of infection from cats and other domestic animals, but no distinct proof of such a method of attack has hitherto been published. Nevertheless, Dr. Klein has shown that cats spontaneously suffer from a disease which is associated with the presence of his diphtheria bacillus, and also that the same disease is produced in them by inoculation on the cornea with poison derived from human diphtheria. Dr. Klein is ot

opinion that in cats the primary seat of infection, in idiopathic cases, is the lungs.

**ÆTIOLOGY.**—The exciting cause of diphtheria is doubtless an organised poison, and it may be accepted, provisionally at least, that it is the diphtheria bacillus described by Dr. Klein. The means, however, by which this germ obtains access to the body, and is allowed to work its ill effects therein, are of all importance, and constitute the predisposing causes of the disease. These may shortly be discussed as follows:—

1. *Age.*—The statistics of the incidence of diphtheria at various ages have been collated by Dr. Thorne Thorne in his 'Milroy Lectures' for 1891. The greatest number of cases, he remarks, occur between the ages of two and five years, or, more widely stated, between three and twelve years, and this excess may possibly be due to the greater exposure to infection during this period from the aggregation of children in schools.

2. *Sex.*—The disease is more frequent in females, possibly, according to Dr. Thorne, because of the greater danger of infection they undergo in nursing the sick.

3. *Heredity.*—There undoubtedly are some families in whom a more than usual number of cases of diphtheria occur, and this probably is due to an inherited weakness, rendering the members unduly receptive of the poison.

4. *Season.*—Dr. Tatham, when medical officer of health for Salford, published in his reports most careful statistics as to the number of cases occurring in that locality, where diphtheria is especially rife, at various times of the year. Dr. Tatham has kindly furnished the writer with the table which follows, in which it is clearly shown that an increase in the number of cases of diphtheria begins in the third quarter of the year; the number attains its maximum in the fourth quarter, and, while declining in the first quarter, nevertheless is still in excess of the number occurring in the second quarter.

BOROUGH OF SALFORD.

*Reported Attacks of Diphtheria in the several Quarters of the Seven Years 1883-1889. (Dr. Tatham.)*

	First quarters (Jan., Feb., March)	Second quarters (April, May, June)	Third quarters (July, Aug., Sept.)	Fourth quarters (Oct., Nov., Dec.)
Reported cases . . . .	270	233	290	431
Proportion to whole . .	22%	19%	24%	35%
Deaths amongst the above reported cases.	79	62	58	125
Case fatality . . . . .	29%	27%	20%	29%

The table also shows (see percentage of deaths) that in the third quarter of the year

the milder cases occur, and the greatest number of deaths in the fourth and first quarters, the excess in the first quarter being probably due to a legacy of severe cases left from the preceding quarter.

5. *Rank.*—The disease is more prevalent among the poor, probably because overcrowding increases facilities for contagion and infection.

6. *Hygienic conditions.*—There is no doubt that exposure to sewage emanations is a fruitful source of diphtheria. How this comes about is not precisely certain: we do not yet know that the diphtheria-germ grows in sewage, or is given off in sewage-gas. The cases reported by Dr. Hill of perineal diphtheria, already mentioned, tend to show that this might be the mode of infection. Dr. Thorne, however, is of opinion that sewage-gas produces a morbid condition of the fauces which renders the patient more liable to the reception of the diphtheria poison. He makes the very apposite remark that diphtheria has greatly increased of late years, and especially in towns, whilst improvement in hygienic surroundings, and especially in the disposal of sewage, has received much more attention, and in consequence of this, diseases undoubtedly due to sewage infection, such as enteric fever, have greatly diminished. This is a fact which militates against the view that diphtheria is connected with poisoning by sewage, but the statistics of the association between the two are very positive.

In former years, diphtheria was more common in country districts than in towns, or, to speak more accurately, in the sparsely populated than in the densely populated parts. In recent times, as shown by Dr. Longstaff in the *Report of the Medical Officer to the Local Government Board for 1888*, this relation has been reversed, and now diphtheria is the more common in thickly populated districts. The statistics also show that the geological conditions have but little effect on the incidence of the disease. Dr. Thorne, however, asserts that this is only true as regards the structure of the soil itself, in relation to drainage. A clay soil may be rendered immune from diphtheria by the possibility of free drainage. But where moisture is accumulated, and also allowed to remain stagnant, there diphtheria is rife.

7. *Influence of other diseases.*—The way for the diphtheria poison may be prepared by other diseases, such as tonsillitis, rendering the fauces a vulnerable point, or by diseases which weaken the system and so diminish its power of resistance. Probably the puerperal state should be associated with the latter group of conditions.

In most works on diphtheria hitherto published, it has been customary to speak of a primary and of a secondary form of the disease. The former is that ordinarily met with. By the latter is understood the occurrence

of diphtheritic lesions and symptoms after various other diseases, amongst which the principal are scarlet fever and measles. It must be remembered in connexion with this subject, that the mere appearance of a false membrane is not a proof of the existence of diphtheria. The same process, coagulative necrosis, which is the main factor in producing the diphtheritic membrane, also takes place on mucous surfaces as the result of many other intense inflammations. Thus, in scarlet fever, the occurrence of a false membrane on the fauces may be due only to a specially irritative effect of the scarlatinal poison. Nevertheless, cases not infrequently occur—and the writer has had many opportunities of examining such—where, together with positive symptoms of scarlatina, large quantities of diphtheritic membrane have formed in the throat, and have even extended to the larynx, and where also after a time the characteristic paralyses of diphtheria have shown themselves. Such cases are specially prone to occur when epidemics of scarlet fever and diphtheria co-etaneously exist, and there is reasonable ground for believing that they are due to a dual infection by the poison of both diseases. In attacks of 'scarlatinal diphtheria' a special streptococcus has been found by several observers, but the evidence of its identity is still incomplete.

8. *Previous attacks.*—One attack of diphtheria confers no immunity upon its subject. Even during convalescence the patient has been known to incubate the disease afresh, and this process may be repeated more than once.

DIAGNOSIS.—Almost any affection of the throat may be mistaken for diphtheria, but the mistake is only likely to happen in the case of acute follicular tonsillitis, scarlet fever, and syphilitic affections. Their points of difference may be summarised thus:—

1. *Acute follicular tonsillitis.*—Here the amount of fever may be equal to or even greater than that found in diphtheria. Usually the fever begins more suddenly, without prodromata, and is accompanied by more pain in the throat and more disagreeable general symptoms than occur in the early stage of diphtheria. In fact, the less serious disease has in most cases the more severe symptoms. In acute follicular tonsillitis, even from the first, exudation can be seen issuing from the tonsillar crypts, forming many small white points upon the surface of the swollen tonsils, and nowhere else. In diphtheria some hours, or even days, usually elapse before more than one patch of membrane is seen, and this may possibly not be upon the tonsil; or, if there be more than one, a patch situated elsewhere than on the tonsil will decide the diagnosis. The diphtheritic membrane, too, soon becomes thick and leathery, and is not

easily removed, while the exudation of tonsillitis remains soft and creamy and is easily wiped away with a brush. In acute tonsillitis nasal catarrh is not often met with, while in diphtheria of moderate severity it is frequently an early symptom.

2. *Scarlet fever.*—Diphtheria rarely begins with the same suddenness as scarlet fever; and vomiting, too, is not usually an early symptom. The temperature in scarlet fever rapidly rises to a height which is rarely found in diphtheria, and then only in the later stages of an acute attack. After twenty-four hours, too, the scarlet-fever rash appears. This may possibly be mistaken for the erythema rarely seen in diphtheria. But the diphtheria-rash, when present, does not spread over the whole body, being limited to the upper parts of the chest and the neck; it is not punctate; and it is never an early symptom. The special albuminuria of scarlet fever first makes its appearance late in the disease or at the beginning of convalescence; and if albuminuria should occur earlier, it is only slight, and such as may accompany any febrile state. The urine, too, in scarlet fever frequently contains blood together with the albumen. In diphtheria, on the other hand, albuminuria is an early symptom; and in a doubtful case, if the urine contains much albumen after only two or three days of the fever, a diagnosis of diphtheria is the more probable. The urine in diphtheria rarely contains blood. The throat affections of the two diseases may resemble each other very closely, for in scarlet fever, not only may the slough of the mucous membrane simulate the false membrane of malignant diphtheria, but, as already stated, there may occur in scarlet fever a false membrane having the same pathology as that of diphtheria. Yet, in scarlet fever, ulceration is more prominent than in diphtheria; shreds of membrane are not expectorated, and there is no tendency of the throat-affection to spread to the larynx. The writer has already mentioned, as his opinion, that in the cases where laryngeal affection has ensued upon scarlatinal symptoms, there has been a double infection by the two poisons.

3. *Syphilitic affections.*—Syphilis, in the secondary stage, may produce white patches over various parts of the throat, due to coagulative necrosis of the epithelial cells. These may be mistaken for diphtheritic membrane. But they are never accompanied by so much fever as occurs in diphtheria; they are not so raised as diphtheritic patches; and the parts around are not so swollen as in diphtheria. Moreover, the rash of secondary syphilis present on the body-surface at the same time is diagnostic. The ulceration of the throat which may appear in the tertiary stage of syphilis might be mistaken for a diphtheritic condition during an epidemic of diphtheria; but the remembrance of the possibility of its

occurrence should be sufficient to remove the chance of error.

The paralysis of diphtheria has the characters of paralysis due to peripheral neuritis, in the article upon which subject details will be found as to its diagnosis. The special characters distinguishing it as of diphtheritic origin are—the history of the throat trouble, if this be obtainable; the peculiar progress of the paralysis; and the early affection of the palate and eyes, which are rarely touched by other forms of peripheral neuritis. See NEURITIS, PERIPHERAL.

PROGNOSIS.—The prognosis in diphtheria must always be guarded, even in the mild cases, for serious symptoms may at any time break out. The prognosis is the more serious the greater the quantity of membrane to be seen in the throat, the higher the temperature, and the greater the weakness and the severity of the general symptoms. The early signs of heart-failure—namely, an unduly slow or very rapid pulse, and the alterations in the cardiac sounds already detailed—are warnings of impending great peril. The malignant form is nearly always fatal; and consequently the occurrence of much hæmorrhage from the throat or nose, a gangrenous condition of the false membrane and surrounding parts of the throat, or signs of much nasal affection, are of grave significance. The occurrence of albuminuria adds nothing at all to the severity of the original disease, and therefore is *per se* of no consequence in judging the prognosis. But, with this exception, any one of the complications mentioned above adds to the gravity of the attack. This is especially the case with extension of the disease to the larynx, which, if unrelieved by operation, in nearly all cases will kill the patient. After operation, too, a large number of patients die either from the severity of the original attack, or from extension of the membrane down the bronchi and consequent occurrence of diffused pneumonia. In the experience of the writer, those cases of laryngeal diphtheria which have followed on scarlatinal symptoms are specially fatal. Generally speaking, the prognosis is the less favourable the younger the subject.

The prognosis of diphtheritic paralysis is on the whole favourable. Nearly always the paralysis recovers. The unfavourable cases are those in which the muscles of the larynx and those of the trunk are paralysed.

TREATMENT. — 1. *Prophylaxis*. — Diphtheria is undoubtedly both contagious and infectious. The infection lies in the exhalations of the patient, and especially in the portions of false membrane expelled from the throat. For this reason it is necessary that a case of diphtheria should be stringently isolated, and that all false membrane coughed up should be at once received into a vessel containing an antiseptic fluid, such as carbolic acid (1 in 20) or perchloride of mercury (1 in

500). During the course of an epidemic no milk should be partaken of by healthy persons unless boiled shortly before being used. In the light of our present knowledge, doubtful though this may be, it would also be well to banish cats and birds from a household during an outbreak of diphtheria. Löffler has fixed eight days after the apparent disappearance of the disorder, as the period during which a patient should remain in quarantine.

2. *Surroundings of the patient*. — The patient should be kept in a warm, well-ventilated room. If there is any sign of affection of the respiratory passages the air of the room should be kept moist, and this may be effected by allowing the nozzle of a bronchitis-kettle to enter within the covering of a tent-bed. A warning may here be given that, without due care, *cool* and not *hot* moisture may thus be supplied to the patient, and may be extremely harmful by depressing the vital powers and inducing pneumonia. Heed to this point is especially necessary after the performance of tracheotomy.

3. *Diet*. — The extremely weakening effect of diphtheria must always be borne in mind, and every effort made to help the patient to assimilate nutriment during the attack. This, however, is a difficult matter, for not only does the patient frequently loathe food, but if, in spite of this, it be administered in injudicious quantities, vomiting may be set up, which will totally preclude nutriment being given in the ordinary way. In these matters the judgment of the careful physician may be taxed to the utmost, and must decide on the merits of each individual case apart from any general laws. As a rule, milk must be the staple article of diet. It may with advantage be peptonised, and mixed with lime-water or soda-water in the proportion of 2 in 3. It should be given at intervals of two hours, or, if only small quantities can be taken at a time, every hour. With it, twice or thrice a day, the yolk of an egg may be beaten up, or arrowroot boiled in milk may occasionally be substituted. Again, strong beef-tea should at intervals be given, not only as a relief to the milk-diet, but also for its stimulant effects. If the condition of the throat do not debar the patient from swallowing them, beef-jelly, or chicken- or beef-essence, may be given similarly. Where milk disagrees, raw beef-juice will sometimes be found of great value. Children will not infrequently refuse to swallow food, even when the throat-affection does not interfere with its passage. Here two means may be made use of to maintain the nutrition: either the child may be fed by the nasal tube, a disagreeable proceeding which is rarely applicable in diphtheria, or the feeding may be carried on *per rectum* by means of nutrient enemata or suppositories. The latter method must always be resorted to when severe vomiting prevents food being given by the mouth.

4. *Stimulants.*—Alcohol is needed in every case of severe diphtheria, and is best given in the form of champagne or brandy, at intervals of two or three hours, in quantities proportionate to the age of the patient and the severity of the attack. It must always be given freely when any of the signs of cardiac failure are perceived.

5. *Internal medication.*—Of recent years rational therapeutics has been employed with great advantage in the treatment of diphtheria. The disease has long been thought to be due to the action of germs or of the products of their growth, and for this reason many germicide remedies have been proposed. Quinine was used before any such theories were mooted, but is a most valuable remedy. Again, the perchloride and also the periodide of mercury have been employed with great success. The perchloride of mercury,  $\frac{1}{2}$  to  $\frac{1}{4}$  of a grain, with sulphate of quinine 2 grains, administered to an adult patient every third or fourth hour, is an efficacious combination. The periodide of mercury can best be given in the soluble form recently introduced by pharmacists. A still more powerful drug, the cyanide of mercury, has been recommended, but cannot be other than a dangerous remedy. Salicylic acid and its compounds, too, have been used with apparent benefit. Of the older and empiric remedies, perchloride of iron and chlorate of potassium have been the most employed, and are undoubtedly of great value. They may be given in combination together with glycerine, and the iron should be administered freely so long as vomiting does not occur. The general antifebrile remedies may also be given in diphtheria, but acids are preferable to alkaline compounds, since the latter are depressing to the circulation. On no account whatever should antifebrin, antipyrin, or any of the so-called new antipyretics be used in diphtheria.

The number of other drugs which have been employed in the treatment of this disease is very large, but as the writer has no experience of them he can do no more than mention them. Amongst them are peroxide of hydrogen, hyposulphite of sodium, and turpentine, not to say chloral and jaborandi, which in the opinion of the writer are dangerous.

The older physicians frequently administered emetics in diphtheria, with the object of detaching and expelling false membranes from the air-passages and throat. Their results from this treatment were often good. But now that more is known of the heart-failure so common in diphtheria, and of the depressant effects which emetics have upon the circulation, they are used more rarely. If employed at all, only the least depressant, such as sulphate of zinc, ipecacuanha, or apomorphine should be given.

6. *Local remedies.*—The throat is the

locality from which the specific poison of diphtheria is absorbed into the system, but it is very doubtful whether remedies applied thereto are of any effect upon the poison itself. Nevertheless, we have in diphtheria a poisoning by general septic matter, the result of the putrefactive changes going on in the throat, and much good can be done by remedies which arrest this process. No force whatever should be used to remove the membranes, but the throat should be sponged out frequently with a solution of perchloride or mercury (1 in 500), of carbolic acid (1 in 40), or of resorcin (1 in 20). An excellent application, too, is the solution of the perchloride of iron, either alone or mixed with an equal quantity of glycerine. The tincture of iodine applied locally has also been found very beneficial.<sup>1</sup> These applications may be alternated with the use of the carbolic spray. A large number of other applications have been recommended at various times, but those just mentioned will be found the most useful. Many of those which have been used have no other merit than that of being harmless. Some, such as papain, &c., have been recommended because they had the power of dissolving diphtheritic membrane outside the body. They have, however, not been found so efficacious in dissolving the membrane *in situ*; and, if they had been, they would only have been of use when the membrane attacked the air-passages. Their thorough application, in such conditions, is difficult or impossible, and, moreover, in the same circumstances, other treatment is more urgently required.

7. *Treatment of certain complications.*—

(a) *Spread of the membrane to the air-passages.*—In view of the extreme fatality of this complication, operative treatment is at once called for, as soon as it is diagnosed by the signs already detailed, even though it be in an early stage. The opinion of surgeons is as yet divided as to the comparative merits of tracheotomy and Dr. O'Dwyer's intubation of the larynx, but one or other operation must be performed. It is true that even after this the prognosis is still bad, for death from pneumonia is very common; yet the best chance has been afforded to the patient.

(b) *Spread of membrane to the nares.*—When this occurs, antiseptic solutions similar to those used for the throat should be injected frequently into the nares. Care, too, should be taken to quickly wipe away all secretion issuing from the nostrils, in order that excoriations and secondary infection may be avoided.

(c) *Vomiting.*—This is best treated by the sucking of ice, and the administration of carbolic acid, creasote, or bismuth. Ice to the

<sup>1</sup> In a note to the writer, Dr. Adamson, of Rye, reports highly of the *internal* use of tincture of iodine in frequent doses of 3 to 7 minims. It produced no ill effects, and was easily taken.

epigastrium, or a blister over the epigastrium, will frequently be of use; but if a blister be used, care must be employed that the resulting sore be not infected by the diphtheritic membrane. If the vomiting is severe, food must be given by the rectum.

(d) *Heart-failure*.—Here free stimulation must be resorted to, both during the attack and when it is threatened. During the attack remedies are not of much avail, for death is so frequently sudden; but, if possible, nitrite of amyl or nitroglycerine may be administered. The subcutaneous injection of liquor strychninæ hydrochloratis in doses of three minims repeated every half hour (for adults) will sometimes tide the patient through the dangerous period. When the signs of heart-failure are perceived, even if the patient is apparently convalescent, he must be kept in the recumbent posture at absolute rest. General tonic remedies should be given, together with strychnine, iron, and digitalis.

(e) *Paralysis*.—For this complication the most efficacious remedies are good diet, good air, and the administration of iron and strychnine. Massage, when the extremities are affected, is often of value, and the application of a weak faradic current seems sometimes to promote recovery.

ROBERT MAGUIRE.

**DIPHThERITIC**.—Relating to diphtheria. The term is applied to the membrane formed in diphtheria; and it is also associated with certain symptoms occurring in the course of the disease, such as diphtheritic paralysis. See *CROUPOUS*; and *DIPHThERIA*.

**DIPLOE**, Diseases of.—See *SKULL*, Diseases of.

**DIPLOPIA** (*διπλός*, double; and *ὄπτομαι*, I see).—Double vision. See *STRABISMUS*.

**DIPSOMANIA** (*δίψα*, thirst; and *μανία*, madness).—SYNON.: *Oinomania*; Fr. *Manie Ébrieuse*, or *Crapuleuse*; Ger. *Trunksucht*.

**DEFINITION**.—An irritable condition of the nervous system, characterised by a craving, generally periodic, for alcoholic and other stimulants.

**ÆTIOLOGY**.—This peculiar condition may be brought on by a course of intemperate drinking; but it is seldom the result of that cause alone, and is not infrequent in persons who have never been intemperate prior to the development of the morbid craving. The occurrence of this form of insanity, as of other degenerative nervous diseases, may generally be traced in the family history of the patients. But sunstroke, a blow on the head, or other direct injury to the brain may excite it; and it may be symptomatic of epilepsy, or of structural disease of the brain. It may be developed at any period of adult life; but most frequently declares itself during the pubescent and climacteric periods.

**SYMPTOMS**.—An instability of character and indications of peculiar nervous irritability may generally be recognised as having preceded the distinct development of the craving. It is also usual to find such persons as are predisposed to the disorder abnormally sensitive to the influence of stimulants. Sometimes very small quantities of alcohol produce appreciable intoxication. The duration of the periods of craving is variable; but most commonly they last one or two weeks. The remissions continue for periods varying from two to twelve months. During the period of craving the whole moral being is enthralled by the morbid desire; and the regard for truth, decency, or duty is generally altogether lost. Moderate indulgence in a stimulant may bring on the morbid craving; but the desire is frequently developed without any such introduction. Members of the household in which a patient lives can indeed often recognise the indications of a coming attack by a restlessness and depression which precede any such indulgence. During the intervals the patient seems, except when the brain has been weakened by frequent attacks, to recover completely; and he generally displays great confidence in his ability to resist the tendency in future. Repeated attacks always produce a permanent degradation, both intellectual and moral; and if the patient live long enough he lapses into a state of dementia. It sometimes happens that some cerebral lesion, of which the dipsomania had been symptomatic, manifests itself in paralytic or convulsive symptoms; and the appearance of such phenomena is often accompanied by a modification of the craving.

**DIAGNOSIS**.—True dipsomania may easily be, and often is, confounded with mere habitual drunkenness. In dipsomania, however, there is, as a fundamental condition, a pathological condition of the brain which manifests itself irrespective of external circumstances of temptation. In habitual drunkenness the craving consists mainly in a desire to keep up a condition of stimulation to which the brain has become accustomed. The habit is the result merely of compliance with a vicious custom, and there is no such periodicity or independence of external influences in the symptoms as is found in the true disease.

**TREATMENT**.—Prolonged abstinence from stimulants, and adherence to the tonic regimen, are the only measures from which any amelioration can be hoped for. It is seldom possible to restrain the gratification of the craving without compulsory seclusion; and even when this has been successfully enforced for a considerable period, the morbid tendency is seldom eradicated. By the 'Habitual Drunkards Act, 1879,' amended and made perpetual by the 'Inebriates Act, 1888,' a person who is, by reason of habitual intemperance, at times dangerous to himself

or others, or incapable of managing himself or his affairs, may, upon his own written application, attested by two justices, be admitted into a licensed 'Retreat,' and detained for such period, not exceeding twelve months, as he may specify in such application. Several retreats have been licensed under the provisions of this Act for the treatment of dipsomaniacs, with, in many cases, satisfactory results.

JOHN SIBBALD.

**DISCRETE** (*discerno*, I separate).—This adjective is used in reference to certain cutaneous eruptions, in which the spots or pustules are separate from each other; for example, discrete small-pox as distinguished from confluent.

**DISCUTIENTS** (*discutio*, I drive away).

**DEFINITION.**—Local applications which are supposed to remove or disperse the congestion and effusion of inflamed parts, and the swelling of the skin over them.

**ENUMERATION.**—The chief discutient measures or agents are:—Friction; Pressure; Warm moist applications; Counter-irritation by blisters or liniments; Mercury and its preparations; Iodine and its preparations, including the Iodides of Potassium, Lead, and Cadmium.

**USES.**—These remedies are generally applied over enlarged joints, enlarged glands, or cystic tumours. The most powerful amongst them are blisters, which sometimes remove enlargements with great rapidity; next to these come mercury and iodine and their preparations, either alone or in combination. Their action is aided by heat and pressure. The effect of the former is seen in the Indian treatment of goitre, which consists in rubbing iodide of mercury ointment over the tumour, and exposing the patient to the full rays of the sun, or to the warmth of a large fire. The beneficial effects of pressure are observed in the diminution which takes place in enlarged and swollen joints under the application of mercurial ointment or strapping, the friction with the hand in applying the ointment, and the pressure exerted by the strapping greatly increasing the efficacy of the mercurial preparation in removing swelling. See FRICTION.

T. LAUDER BRUNTON.

**DISEASE** (*dēs*, from; and *aise*, ease).  
SYNON.: Fr. *Maladie*; Ger. *Krankheit*.

**DEFINITION.**—Disease may be defined as a deviation from the standard of health in any of the functions or component materials of the body. See PATHOLOGY.

The expression 'a disease' is frequently used with reference to a supposed unit of causation. Thus, it may be applied to some simple phenomenon, for example, neuralgia, when that phenomenon is the sole effect of a cause, such as a carious tooth; or it may include many concurrent or consecutive result-

ant phenomena, such as those of syphilis or typhoid fever.

**GENERAL CONSIDERATIONS.**—It is well known that changes of function and of structure are brought about and influenced by a great variety of agencies. These agencies, some of which act from within, others from without, are recognised as the *causes* of disease. Such changes, whether they be functional, affecting more especially the vital properties of the body without manifest alterations of structure, or structural, clearly affecting its physical properties, constitute what is familiarly known as DISEASE, which is hence said to be *functional* or *structural* respectively. These changes are merely the evidence of an altered or perverted action, which is then in operation or has already occurred, the nature of which is considered under the head of PATHOLOGY. When these deviations from health can be recognised during life they are described as the *symptoms* or *signs* of disease. For example, when a person, after exposure, it may be to wet or to cold, or both, is found to have an increase of temperature, with a quick pulse and perverted secretions, to complain of thirst, and of pain at the joints with effusion in and around them, we say that such person is labouring under disease; and we call it 'rheumatism,' because that name has been assigned to a complexus of deviations from health, such as those then presented by this individual. When typhoid poison has been introduced into the body, it leads in like manner to a number of functional and structural changes, which, taken together, constitute what we call 'typhoid fever.' Or, again, under certain circumstances there appears to be generated in the system, whether as the result of a tendency established before birth (hereditary) or by habits of life (acquired), an agency which, acting morbidly, produces a series of phenomena which we call 'gout.' These several forms of disease may be *classified* in groups, arranged in accordance with the causes which give rise to them, or their nature, their seat, their duration, &c. Rules are laid down for the modes of distinguishing or *diagnosticating* one disease from another; for *prognosticating*, as far as may be, their result; and for their *prevention* and *treatment*.

Thus it comes that the discussion or description of any *particular disease* consists of an account of the causes that give rise to it, or its *Ætiology*; the changes of structure or of function which constitute it, that is, its *ANATOMICAL CHARACTERS* and *PATHOLOGY*; the phenomena attending these changes, otherwise, the *SYMPTOMS* and *SIGNS* of the disease; the facts that serve to distinguish this particular disease from other diseases, that is, its *DIAGNOSIS*; the means of forecasting its progress and termination, which constitute its *PROGNOSIS*; and finally the

measures by which it may be prevented, relieved, or removed, that is, its TREATMENT. Throughout this work the various diseases are, as far as is practicable, discussed upon this uniform plan.

It is well to remember that, as thus understood, DISEASE is an abstract or relative term, and not an entity having a special and independent existence. Physiology has in recent days diffused a clear and penetrating light over many of the processes of life in health, which were previously dark and obscure. Pathology, which is physiology applied to the study of unhealthy function and structure, earnestly follows the footsteps of the sister science. We are therefore not hoping and believing too much when we express our conviction, that the time is not remote when we shall be able to trace those early and subtle changes which constitute disease, as well as the causes which give them origin, and that we shall thus be enabled to define in a more philosophic form and with more practical results what disease really is. In the meantime we must be content to work upon the phenomena before us, to investigate so far as we can the causes of disease, how to recognise its presence and its nature, how to estimate its progress and its duration, and finally how to prevent its occurrence or to cure it when it has occurred. These varied and important points will be found discussed, as above stated, under suitable headings, in the articles immediately following, and in other parts of this work.

RICHARD QUAIN.

**DISEASE, Causes of.**—DEFINITION.—Whatever is capable of damaging the structure of any organ or tissue of the body, or of interfering with its function, may be a cause of disease. This definition implies that such causes are numerous, and that of many of them science is yet ignorant. To give a succinct account of them is therefore difficult, nor is the difficulty diminished by the fact that, in most diseases, we can trace a succession or combination of causes.

**GENERAL CLASSIFICATION.**—The causes of disease have been divided into (1) *Predisposing* or *Remote*, and (2) *Exciting* or *Proximate*. Illustrations will explain what is meant by these terms:—Two individuals are exposed to the contagion of typhus in equal degree; one, wearied by bodily and mental labour, ‘catches’ the disease—that is to say, his condition has *predisposed* him to the exciting cause of the malady; the other, in vigorous health, escapes the contagion—the *exciting* cause of disease. Predisposition in fact *prepares* persons by rendering them more susceptible to the influence of exciting causes of disease. Many persons are *predisposed* to emphysema because of heredity; they contract bronchitis, which, by its attendant cough, easily *excites* the malady.

Such illustrations might be extended to a multitude of diseases. Predisposition may be inherited; or it may be acquired, and be due to various accidental causes. In most cases there is a combination of predisposing causes; in a man, for example, lowered by fatigue, want of food, exposure and debauch, an attack of bronchitis or pneumonia will be readily excited. Though where many factors unite in creating the predisposition to disease, it is not infrequently possible to estimate the part played by each, yet practically it is often difficult to say even how much is due to predisposition.

Moreover it is not always easy to distinguish predisposing from exciting causes. Predisposition carried to excess becomes in some cases an exciting cause of disease, and often there is a combination of both causes. There are certain distinct exciting causes—for example, heat, cold, or injuries of various kinds; but most of these can also claim a predisposing power. The contagia of specific diseases are good examples of direct exciting causes.

In proceeding to discuss the subject of ætiology, no attempt will be made to separate definitely predisposing from exciting causes of disease. The writers will endeavour rather to indicate as far as possible, under the head of each factor of causation, the direction in which it especially acts.

The following causes will be considered: (1) *Race*, (2) *Heredity*, (3) *Sex*, (4) *Age*, (5) *Intermarriage*, (6) *Temperament*, (7) *Climate*, (8) *Town and country*, (9) *Hygienic conditions*, (10) *Occupation*, (11) *Air*, (12) *Previous disease*, (13) *Mental and moral conditions*, (14) *External physical conditions*, (15) *Cold and chill*, (16) *Poisons*, (17) *Diet*, (18) *Epidemic diseases, contagion, malaria, parasites, and growths*.

**1. Race.**—The statements formerly adduced to show the influence of this factor of life in the production of disease, have been of late years subjected to a searching criticism, with the result that many are either rejected as inaccurate, or resolved into other factors, such as climate, dress, and diet.

What is left of them may be thus summarised. ‘Lethargy’ and ‘ainhum’ are diseases peculiar to negroes, who also seem especially liable to small-pox and leprosy. In the same race, on the other hand, hæmorrhoids and calculi are unusually rare; and an almost complete immunity from yellow fever is enjoyed by its members. This immunity is said to be proportioned to the purity of the race, but probably may be lost or diminished by residence in a colder zone (Hirsch). Among Europeans, the manifestations of hysteria and insanity are most marked in the Latin races.

**2. Heredity.**—This is one of the most complex predisposing causes, and is therefore proportionately difficult to estimate. In the first

place it is unquestionable that certain specific diseases, such as small-pox and syphilis, may be directly transmitted from parent to child. The second and more complex problem is to determine whether or to what extent immunity from, or susceptibility to, specific contagia, disordered functions, or diseased tissues, can be transmitted from parent to child. The terms 'temperament' and 'idiosyncrasy' are, as Mr. Hutchinson has pointed out, simply expressions of individual heredity.

A virgin community suffers more severely from an epidemic of small-pox than one in which the disease has long been known. Some kind of transmitted immunity is therefore probable. Some specific diseases run in families. Of these the best example is tubercle, with which have been classed leprosy and even typhoid fever and diphtheria. Here it is probably a predisposition to, not a direct transmission of, the disease in question. There is amongst mankind not only an inheritance of such prominent diseases as phthisis, but of peculiarities in the manner they meet and pass through minor ailments. Thus, in families with a 'nervous history,' we meet with predisposition to headaches of nervous type, irregularities of digestion in the form of diarrhoea and vomiting, and a multitude of conditions which have been ascribed to vasomotor disturbances. The members of some families live long in spite of exposure to almost every exciting source of mischief, and contrast most favourably with others who, as far as one can determine, have all things in their favour. There is no doubt that a tendency to gout, the hæmorrhagic diathesis, pseudo-hypertrophic paralysis, Friedreich's disease, hay fever, enuresis, and other diseases is handed down from generation to generation. Of many minor complaints there is less certainty; but it is exceedingly probable that persons are predisposed to bronchitis and other catarrhs by inheritance. It is acknowledged that epilepsy descends from parent to child, and that, in fact, individuals, because of heredity, are often the subjects of nervous diseases excited by causes which those from a different stock are enabled to throw off readily. The various forms of insanity are striking examples. It is supposed that the inheritance lies in the tissues themselves—that there is a something in the tissue-elements which predisposes to certain diseases in certain families. Too much attention is generally bestowed on direct transmission of diseases from parent to child, and too little on tendencies resulting from the sum of forces acting through the innumerable generations of the past. See ATAVISM; and HEREDITY.

3. **Sex.**—There are great differences in the organs and functions of the sexes, and in consequence a great contrast in their predisposition to disease. The female is more delicately constructed than the male, and

those organs which the two sexes possess in common differ in weight and in 'fineness'; and a general consideration at once indicates that the female is less fitted than the male to resist many exciting causes of disease. This is particularly evident at climacteric periods of life: with the onset of puberty girls suffer far more than males, and especially from a variety of reflex spasmodic disorders, which require but little provocation for their development. All the phenomena classed under the head of hysteria often occur at this period. At the same time, and shortly afterwards, there is a tendency to ulcer of the stomach, to persistent constipation, to peculiar attacks of neuralgia—especially of the intercostal nerves, and to acute rheumatism, lapsing into the subacute or chronic kind. Anæmia and chlorosis are also commonly met with at the period of puberty, and if then neglected they are apt to persist, and predispose the individual still more to disease. Goitre, too, is more prevalent among females than among males.

Even so early as the period of puberty the external circumstances of the sexes differ, and on this depends, in a certain measure, the difference in their predisposition. Education, domestic habits and customs, and, above all, occupation, play an important part, and these are largely influential in the production of the diseases just mentioned. But, underlying these outside influences, there is inherent in the sexes a difference in predisposition; for when they are exposed, as often happens, to the same surroundings, they suffer from widely separated diseases. Males are more subject to epilepsy, tetanus, gout, diabetes, locomotor ataxy, pseudo-hypertrophic paralysis, hæmophilia, vesical diseases, and acute lung-affections, than females. The list shows that occupations which involve hard mental and bodily work and constant exposure explain some of the varieties in predisposition. It is probable that females are more frequently ailing than males, but very often their illnesses are associated with the menstrual functions, and are trifling in degree; and, though more males are born than females, towards the later years of life the average of the sexes becomes more equally balanced, or even reversed, because the mortality of males is greater than that of females. It is sufficient to allude to the fact that pregnancy and lying-in predispose females to diseases from which males are entirely exempt; and that there is a considerable difference in the sexes as regards venereal affections, both as to predisposition and the effects of the diseases.

4. **Age.**—This has a most important influence as a predisposing cause of disease. In Wagner's *Manual of General Pathology* the periods of age are thus subdivided:—

- (1) Nursing age (infancy)—from birth to 7th-10th month.
- (2) Childhood—from 1st to 2nd dentition.

- (3) Boyhood—from 2nd dentition to puberty.  
 (4) Adolescence—from puberty to 20th–25th year.  
 (5) Early manhood—from 25th to 45th year.  
 (6) Later manhood—from 45th to 60th year.  
 (7) Old age—from 60th onwards.

This division is excellent, but in no definition is there more need to look out for exceptions than in that of age. The term *age* is strictly comparative; some individuals are old at forty, others young at sixty. Persons fail with regard to particular organs while young in years; and, on the other hand, others acquire an increased power in the same as years advance, of which the brain affords an apt illustration. The minor organs of the body betray the like peculiarities, and in the early decay of teeth, the changes in the hair and the skin, we meet with indications of old age though the individuals are young in years. But, generally, the predispositions of the young and old are striking by their contrast. The young are exempt from fatty degenerations, which are so common amongst those of advanced life, and, in consequence, many diseases amongst them are, *cæteris paribus*, less deadly; and not only does age, by reason of the changes which naturally occur as life goes on, predispose to disease, but all outward conditions become changed. Children—speaking generally—are apt to suffer from acute catarrhal affections of the mucous tracts, glandular diseases, skin-diseases, tuberculosis of acute type, 'scrofula,' inflammation of endo- and pericardium, and a variety of complaints traceable to improper feeding, bad ventilation, overcrowding, and to hereditary tendencies. From acute tuberculosis the aged are almost entirely exempt, and in them the manifestations of hereditary tendencies are observed less frequently than in the young. The very young and the very old are equally subject to bronchial catarrh, and the mortality from this disease at each extreme of life is exceedingly great. But in the young the predisposition to this affection is almost invariably associated with catarrh of the intestinal tract, and diseases which indicate a general constitutional depression; while in the old a predisposition to bronchial catarrh is caused by a degenerative change in the lungs themselves, or in the air-passages. During infancy the nervous system is rapidly developing, and during this stage general convulsions and convulsive disorders, such as laryngismus stridulus, result from slight forms of reflex irritation. As age advances the tendency to spasm decreases, and convulsions become much less marked. The nervous system furnishes other illustrations of 'epochal' diseases. From five to fifteen chorea is common, but differs from convulsions in the greater frequency with which girls are attacked (three to one). Hysteria and the various sensations and vaso-motor disorders

which characterise the climacteric period are also largely 'epochal,' and almost exclusively confined to the female sex.

Some of the specific diseases, especially measles, scarlatina, and pertussis, are more common amongst children than adults, which is partly explained by the fact that the latter class have passed through the ordeal of those diseases, and are thus proof against them. Rickets also is essentially a disease of infancy and early childhood.

The onset of puberty is a constant source of predisposition to disease, for with it comes a complete transformation in the mental and physical characters, so that the individual, if not very carefully watched, deviates from even the most perfect health into a permanent tendency to disease. The system at this period—especially in the case of females—is frequently unable to bear anything which interrupts or interferes with its activity. The generative organs undergo great changes, and with them the whole moral and physical nature is altered. At this period of life there is a predisposition to both bodily and mental diseases; and though constipation, want of sleep, and excessive work at school, especially among girls, are important factors in the causation of disease at this time, perversions of any organ or faculty may be started, and, once started, they are apt to continue; so that there is established, literally, a permanent predisposition to disease, and this predisposition swells very largely the list of affections which are dealt with under the generic term Hysteria. Lung-affections—excepting pure bronchitis—are more common at and shortly after the time of puberty than in previous years; but, excepting in the instance of phthisis, hereditary tendencies are less manifested than during childhood. Even hereditary epilepsy is, if postponed beyond early years, likely to be postponed to the period of adult life.

As has been said, the degenerations of organs and tissues begin to show with much uncertainty, but after the fortieth year of life we almost invariably meet with one or other of them. Their degree and their consequences vary with the surroundings of the individual—with his habits, temperament, occupation, and like influences. Diseases of the large vessels are especially common at this epoch, such as aneurysms of the aorta and of the large arterial trunks in the extremities. Henceforward all the diseases peculiar to advancing age become common. The results of previous disease are now declared by a decided predisposition to exciting causes which have been hitherto withstood. Old age is a relative term. A man is old and predisposed to trifling excitants because his lungs have lost their elasticity, or his brain its regularity of circulation, or his heart its vigour: in each of these cases, as in a host of others, the predisposition is

strictly one of degeneration—most frequently of the vascular system. Again, inherited diseases do not declare themselves in some cases till the later years of life, and of this carcinoma is a striking example. The old are predisposed to lowering diseases—low pneumonia or bronchitis; and to a variety of nervous affections which the vigorous can resist. In short, they succumb to insignificant exciting causes, because of the general or partial decay of the tissues and textures of the body.

5. **Intermarriage.**—Intermarriage certainly predisposes to disease, but it is not easy to determine how far its predisposing powers extend. Hereditary influences are thereby intensified. If the practice were confined to carefully selected cases, some advantage might ensue. Breeders of first-class animals practise intermarriage, and thereby develop speed, quality, and endurance in the offspring. It is beyond question that this practice of breeders of racehorses is eminently successful for the time, but it by no means follows that the permanent results are good. We are bound to look not only to the immediate, but to the ultimate results of intermarriage—in short, to decide whether intermarriages predispose to disease, to the injury of the community. But no rule, free from exception, can be laid down on this subject, for beyond all doubt many intermarriages have led to both physical, moral, and mental advantages. There is no doubt that malformations are handed down, and that where these are marked in families it is injudicious for persons to intermarry. Where also serious diseases, such as phthisis, insanity, or cancer, have been met with on both sides, it is most advisable that intermarriage should not take place.

6. **Temperament.**—Temperament is only of importance as expressing certain inherited predispositions to disease. It is not in itself either a predisposing or an exciting cause. Many forms of temperament were described by the older writers. According to them, persons of *sanguine* temperament are disposed to congestions of organs, and hæmorrhages, on comparatively small provocation. *Phlegmatic* individuals are the subjects of those diseases which are readily excited by want of mental and bodily energy and activity. A third set belong to the *nervous* temperament; such persons are easily excited and easily depressed, and are prone among other diseases to hysteria or hypochondriasis. Nervous persons suffer quickly from delirium and other brain-symptoms, which aggravate and render dangerous an otherwise hopeful malady. In the acute specific diseases this is particularly manifested; *nervous* people are undoubtedly predisposed to them, and when once attacked are unduly liable to dangerous complications. Practically we meet with 'mixed' temperaments, though one may especially prevail.

On the whole, much confusion might be avoided if these terms were altogether discarded, and the connotation of each, stated in definite language expressing what is actually observed in each case, substituted. It would then be evident upon how slight a basis statements regarding temperament are often grounded. See TEMPERAMENT.

7. **Climate.**—This includes many factors, all of which may predispose to disease. Amongst these, the influence of altitude, moisture, temperature, season, and soil will be considered:—(a) *Altitude.* Dwellers in mountainous regions suffer with excessive frequency from cardiac hypertrophy and from hernia, though the causes of these should be more properly sought under 'occupation' than 'climate.' 'Mountain sickness' is wholly dependent on barometric pressure, and is only rarely met with below a level of 10,000 ft. The prevalence of both malaria and phthisis is almost inversely proportional to the height of the district above the sea-level. Cholera is most prevalent in valleys and plains. (b) *Moisture.* Dry air is generally considered less provocative of bronchitis than moist, and this disease may be regarded as one of moist climate. (c) *Temperature.* In the tropics various endemic fevers prevail which are unknown in this country, for they cease to exist when the temperature sinks below a certain level (about 60° F.), probably because the micro-organisms concerned in their spread are unable to thrive below that temperature. Frost very often cuts short epidemics in our own country in like manner. Particular organs are predisposed to disease by climate—the liver in the East Indies, the lungs and the kidneys in regions where the temperature is capricious. Tropical heat is essential to the existence of yellow fever, malaria, and dengue; and is an important factor in the production of such diseases as cholera, tropical stomatitis, dysentery, and hepatitis. Heat may kill suddenly, as in sun-stroke and heatstroke, or excite cerebral mischief just short of death, especially in confined, ill-ventilated places. Long-continued heat lowers the vital powers, and may excite such slight diseases as eczema of a simple character, or such grave affections as inflammation of the membranes of the brain. Choleraic attacks in this country usually are associated with exposure to immoderate heat. (d) *Season.* The influence of season may be resolved into the influences of temperature, rainfall, and prevalent winds. Animal parasitic diseases are more prevalent in the summer. Yellow fever, malaria, and dengue are rife in the summer and autumn, when the rainfall is heavy. Deaths from lung-diseases are most numerous in the winter, and from nervous diseases in the spring. (e) *Soil.* No better proof of the tendency to disease dependent upon the nature of the soil can be given than that yielded by the many careful

observations which have shown that where a proper system of drainage of soil-water is carried out, the tendency to pulmonary diseases is very greatly diminished. Clay soils are cold, damp, and impermeable, and favour diseases aroused by these combined agencies; sandy and gravelly soils readily drain themselves, are warm and dry, and thus far tend to protect those who live on them against disease. Climates are also modified by trees, rocks, rivers, lakes, and other physical conditions. Detritus carried down by streams and deposited along their banks or at their estuaries, has, like ground vegetation and its decay, an undoubted predisposing influence in the spread of malaria. The upturning of large tracts with imperfect cultivation is also frequently followed by outbreaks of malaria. Particular districts also predispose to certain diseases. Cretinism is most common in close valleys; urinary calculus is endemic in many districts of Great Britain and elsewhere. In some circumscribed areas in Scotland, in Norfolk, and other districts, individuals are especially liable to stone.

Fogs in large cities depress most people; and it is not too much to say that many diseases may be traced to a predisposition which 'bad weather' has started. Alcoholism has been provoked in this way. It is clear, therefore, that under the head of climate there are many combining influences, which affect the moral, mental, and bodily nature of individuals, and through one or all may predispose to disease. In this country, and probably elsewhere, those who dwell on mountains are less liable to disease than the dwellers on plains—marshy plains especially. New comers are more prone to the endemic diseases of a district than the natives. The best instances of this are dysentery and malaria. It should be remembered, however, that it is not climate alone which varies the predisposition amongst persons residing in different regions; for their habits, diet, and social conditions differ far more than the climate in which they dwell, and predisposition to disease should never be ascribed solely to climatic conditions unless accidental influences have been investigated and eliminated.<sup>1</sup> See CLIMATE.

**8. Town and Country.**—The influences of town and country, as predisposing to disease, require separate consideration. With them may be considered dwellings, and a variety of minor sources of predisposition. The mortality of country districts is less than that of towns, but towns differ in this

respect amongst themselves. The health of the largest city in Great Britain, for example, compares favourably with any of the large manufacturing towns and with many rural communities. It is easy to understand that differences must depend on the occupation, food, and habits of the people, and on their external surroundings—air, light, drainage, and like conditions. In larger towns occupations are more lowering than in country districts; while the physical and mental strain is greater, and has fewer interruptions. Late hours, intemperance, and prostitution prevail in towns; but it is by no means certain that, in proportion to the population, these ills are less frequent in the more remote districts. In towns overcrowding checks ventilation, makes drainage difficult, so that subsoils become saturated, clouds the atmosphere with smoke and dust, intermingles the sexes (amongst the lower classes), so that succeeding generations are stunted in their development, and in a variety of other ways predisposes to disease. But in some towns these evils have been obviated by sanitary measures, and as a consequence towns often predispose less than country districts to diseases due to defective drainage and overcrowding. Some startling illustrations of this fact have occurred since sanitary science has been acknowledged to be of national importance. In villages, for instance, reputedly 'model,' epidemics of disease associated with defective drainage and evil domestic arrangements have decimated the inhabitants, and the virulence of these epidemics has been greater than that of those met with in large towns. The latter are nowadays for the most part better drained than country districts; thus milk-epidemics of typhoid fever have been imported into large cities from isolated farms where the drainage alone seemed to be at fault. Amongst children rickets, scrofula, tuberculosis, and choleraic diarrhoea are far more prevalent in town than country; and the more crowded the population the greater the mortality from phthisis. The mortality amongst young children is far greater in towns than in country districts. Adulterations are an evil in large communities, affecting people of all ages. From many of these country districts are free. Unadulterated milk is now as easily obtained in towns as in the country. So while dwellers in the country may have bad drainage and bad houses, they have pure air, as a rule, and every opportunity of breathing it out of doors. Even the alcoholised drinks in many country districts are home-made and harmless when compared with the adulterated raw spirits taken by the lower classes of large towns. On the other hand, however, dysentery is a disease of villages rather than of towns.

**9. Hygienic Conditions.**—One of the most common causes of disease coming under this head is want of cleanliness. To this is

<sup>1</sup> Imperfect drainage leads to what are popularly known as 'damp walls' in dwellings, and thus predisposes to pulmonary diseases by interfering with ventilation—in fact, by preventing the admission of pure air into such dwellings. This truth bears out the teachings we have derived from Dr. Buchanan's investigations at Ely and elsewhere.

due a variety of skin-diseases, such as eczema of the scalp in children, and diseases caused by pediculi. It interferes with the functions of the skin generally, and by it even a common cold is modified. Clothing is a part of the subject. Insufficient, improper, or excessive clothing are most important sources of predisposition, amongst the rich as well as amongst the poor; for, though the climate of this country is so variable, corresponding adaptations of dress are for the most part neglected. Clothing is used to prevent the loss of heat, which occurs in three ways—by radiation, conduction, and evaporation; and a proper adaptation should prevent undue loss of heat, allowing for the escape, instead of ensuring the condensation, of the secretions from the skin, and should be sufficiently loose to permit the movement of the limbs and thorax. Non-conducting, loosely woven, flexible materials are clearly the best adapted for this purpose. Neglect of these requirements is largely instrumental in the causation of disease, particularly in young women of our large towns. A very common cause of predisposition to disease is neglect of proper bedding. Deficient ventilation and overcrowding are prolific sources of mischief. Overcrowding and insufficient ventilation are among the greatest evils of our chief cities and towns; and not only is it in their homes and workshops that populations are overcrowded, but in their places of recreation and of religious worship. Briefly it may be said that overcrowding predisposes to moral, mental, and physical deterioration; to all communicable diseases, including all septic and parasitic conditions, and especially to typhus fever and tubercle; to pulmonary affections; and to a variety of nervous diseases. By lowering the *morale* of populations it increases all other predispositions, and, in fact, passes into an active exciting cause of disease.

**10. Occupation.**—Occupation acts in two ways as a factor in the production of disease. (1) It may expose the worker to certain deleterious influences which are themselves exciting causes of disease. Of this class the best illustration is furnished by workers in poisonous substances, such as antimony, arsenic, copper, brass, lead, mercury, mineral acids, phosphorus, and indiarubber. Turners, knife-grinders, and stone-masons suffer from inflammatory diseases of their air-passages and lungs, induced by the inhalation of fine particles resulting from their work. Corns, chronic inflammatory nodules, and various deformities are produced in those whose occupation involves constant pressure on any one part. Shoemakers and chair-makers illustrate this statement, the latter presenting a characteristic corn and subjacent pleural thickening in the first right interspace.

(2) Occupation has a still wider predisposing influence. The general health may be

lowered, and the resisting power diminished, by the conditions of life in which many occupations are carried on. Among these are (a) the *breathing of vitiated air* due to overcrowding or to deficient ventilation; (b) *deficient exercise*; (c) *exhaustion*, physical or mental, from overwork; (d) the *excessive use of particular parts* of the body; (e) *exposure* to cold and wet, or to excessive changes of temperature; (f) *improper or irregular feeding*; (g) or, on the contrary, absence of adequate employment. These influences are often combined and their effects proportionately increased.

Physical overwork is often conjoined with exposure and improper or irregular food-supply, and the combination has a marked effect. It has so predisposed armies to disease that their ranks have been decimated by fever, pneumonia, and bronchitis, far more than by actual fighting. Not a few medical men have been affected by the contagia of the acute specific diseases, because when exposed to them they were worn out by bodily and mental exertion, and by protracted fasting.

Many other illustrations might be given. Soldiers are most liable to thoracic aneurysm and sunstroke; and young women with no fixed employment, or exhausted from overwork and anxiety, to hysteria. A coachman who uses for many hours his pectoral muscles in driving, suffers chiefly in them when he has an attack of muscular rheumatism.

Many of the diseases mainly dependent on occupation may be avoided or alleviated by careful attention to hygienic precautions.

**11. Air.**—The question of air has already been alluded to, in considering climate, occupation, town and country, overcrowding, &c., and it is scarcely necessary to dwell much more on its ætiological effects. Air influences the predisposition to disease according to its degree of rarefaction, moisture or dryness, warmth or coldness, and the impurities, mechanical or chemical, which may adulterate it. In the article CLIMATE many of these atmospheric conditions are fully dwelt upon, and their tendencies explained. Impurities in the air are exceedingly prevalent; and mechanical substances suspended in it can excite irritable conditions of the air-passages which may pass on to inflammation, and even destruction, of the lungs. Pharyngeal and laryngeal affections are a common consequence of these impurities, and occur largely amongst certain classes whose occupation loads the air with fine particles as already described. There is scarcely a mineral used in the arts which cannot, by inhalation, excite or predispose to disease. The air may also be rendered impure by chemical agencies, and the moment the normal proportion of its elements is disturbed it becomes a source of disease. Excess of carbonic acid is especially an element of

mischief—causing headache, dyspepsia, and nervous depression. The presence of ammonia and of sulphuretted hydrogen is attended by like results. The human economy is, however, so framed that its organs can often very rapidly throw off the evil effects of these gases when breathed in overcrowded rooms, so that no permanent mischief is established. Poisoned air plays a part in the production of tubercle, anæmia, and lowered conditions generally; but it is an incomplete comprehension of the causes of these conditions to set down all to this one. The air, also, may be poisoned by other gases, such as carburetted hydrogen.

The atmosphere is modified by currents—sometimes to the relief, sometimes to the damage of mankind. Winds can remove sources of contagion—they can ‘clear the air.’ But according to many authorities they can also bring contagion into localities. Cholera and other diseases have, it is said, followed aërial currents, that is, have been carried by them. The influence of winds upon health varies with the individual concerned. Some persons are invigorated by warm south-westerly breezes. A north-easterly wind will stimulate the energy of one person, but reduce another to a condition of abject misery. The colder winds generally exert their tonic action to most advantage during the middle periods of life. While easterly winds prevail, acute lung-diseases prevail, particularly in the aged. Eczematous conditions often owe their origin to the same cause.

**12. Previous Disease.**—Previous disease often predisposes to the same or to some other affection, and no clinical history is of value unless it includes an account of former illnesses. In difficult and doubtful cases a true statement of these often gives the clue to diagnosis, and even patients themselves are alive to the value set upon an accurate account of their life-ailments. An attack of croupous pneumonia predisposes to recurrence, especially during the three months succeeding the attack; and it may leave behind a predisposition extending far beyond the original disease. Chorea, acute rheumatism, tonsillitis, malarial fevers, and epilepsy tend to recur, as also do the ordinary convulsions of children; but in all these and many other cases it is difficult to estimate the exact part played by derived predisposition, because in all the primary predisposition may be the main agent in the subsequent attacks. In practical medicine it is distinctly recognised that certain diseases predispose to disease, and in their case recurrence is, very properly, jealously guarded against. Pertussis is supposed to predispose to measles, and *vice versa*. There is distinctly a connexion between chorea, rheumatism, and scarlet fever, and these diseases may follow one another in any order. Again, previous disease

may leave behind pathological conditions which remain in abeyance, until excited by causes which the healthy individual could readily withstand. Pertussis often ends, to all appearances, favourably, but afterwards the patients may suffer from severe lung-affections, upon trifling exposure to exciting influences. Caseated or calcified tubercles in the lungs may after a long period of quiescence excite acute phthisis; hepatic mischief followed by collection of gall-stones in the gall-bladder may cause peritonitis and other diseases. Slight complaints are even more marked in their predisposing powers than serious diseases. On the other hand, previous disease sometimes protects individuals and communities; for example, vaccination can save nations from the most terrible of epidemics—small-pox. In the case of scarlet fever, typhus, pertussis, measles, &c., an almost perfect immunity is acquired by those who have already suffered from them. Of course, as with small-pox, no one denies that second attacks of these diseases do occur, but such attacks are wholly exceptional.

**13. Mental and Moral Conditions.**—Bad news may cause sudden death, or, short of this, may interfere with the functions of particular organs. Sudden mental worry may excite dangerous interference with digestion, or start an abnormal cardiac rhythm. Fright has turned the hair white within a few days or hours in healthy persons. Mental and moral shock may check or increase the flow of urine, and, in fact, may affect all the excreting and secreting organs of the economy. Mental overwork may excite cerebral symptoms of a dangerous and even fatal nature, probably dependent upon a general inflammatory hyperæmia. It may also increase the predisposition to tubercular meningitis. Undue or sudden emotional disturbances can excite serious mischief, just as they can predispose to it. Again, the mind is affected by imitative influences; thus a single hysterical patient may arouse in others symptoms almost identical with her own. The subject of the direct influence of the mental and moral state on disease is, however, too wide to be here dwelt upon.

**14. External Physical Conditions.**—These are very numerous as exciting causes of disease. Violent or over-exertion may cause herniæ, hæmorrhages, as from the vessels of the lungs, cerebral congestions, and even ruptures of the valves of the heart, and in one or all of these cases lead directly to death. Over-exertion with the voice may be followed by pharyngitis or laryngitis. Syncope has occurred in the most healthy from violent exertion in hill-climbing, in boat-racing, walking and running matches, &c., acute dilatation of the ventricles probably occurring. In less marked cases an ‘irritable heart’ may result. Various forms of direct injury are frequent causes of disease.

**15. Cold and Chill.**—Cold carried to excess may prove fatal at once. The influences of severe cold are described under its own heading, and it is only with the diseases excited by cold in the everyday acceptation of these terms that we shall deal here. See **COLD**, Effects of Severe or Extreme.

Cold is the most common cause of disease in temperate climates, especially in the changeable climate of this country. It can excite disease directly, and can affect probably all the organs of the body, causing either disturbed function or organic mischief. Cold, when severe, contracts the vessels; interferes with the circulation, and all vital activity; and in this way may cause death. But it is with moderate degrees of cold we have chiefly to deal. A momentary exposure to a cold draught is as frequent an excitant of disease as general exposure for a long time. A cold draught playing on the cheek may cause facial paralysis, sore-throat, or bronchitis; that is to say, cold applied locally may excite disease in the neighbourhood of its application or in distant organs. It is probable, therefore, that cold may act in several ways: (1) it may interfere with circulation; (2) it may affect the extremities of nerves and excite disease by reflex action; or (3) it may check secretions of the skin, the mucous membranes, or other parts.

We cannot wonder, therefore, that diseases of the throat, larynx, and lungs are frequently excited by cold. Bronchitis and pneumonia are its most common results; and as the young and the old are less capable of enduring cold than adults, its results are, in their case, more frequently fatal. Diarrhœa, renal diseases, congestion of the liver, acute and chronic rheumatism, simple dyspepsia, and a host of other affections, are traceable in many instances only to cold. Predisposition has much to do with the effects of cold: some individuals suffer from one form of disease when exposed to it, others from entirely different affections. In some, 'a common cold' is most evidenced by severe muscular pains and fever, in others by a nasal discharge, in others by headache, and so on. Some persons never suffer from 'cold' without having an attack of herpes labialis; and some are most prone to 'catch cold' whenever the weather suddenly becomes warm. Numerous similar idiosyncrasies might be given. The effects of cold should always be considered in almost all predisposing causes of disease. 'Cold' is a vague term and not thoroughly understood; there is, therefore, all the more reason why, when it comes under consideration in individual cases, its precise effects should be most carefully considered and recorded.

**16. Poisons.**—Poisonous gases are powerful excitants of disease, and so are poisons generally, whether animal, vegetable, or inorganic. They may kill quickly, or excite

a disease of long-continued or even permanent nature.

**17. Diet.**—Food and drink may by their abuse excite disease, and gluttony is as certainly an excitant as drunkenness, though this fact is often lost sight of. Excess of food does not refer simply to the quantity taken, but to its quality—its nature, richness, and the times when it is taken. *Excess of food* overloads the stomach, makes calls upon it which it cannot meet, and dyspepsia is the result. Excess of food, if digested, charges the blood with materials not demanded by the economy, and disease of excretory organs or fatty degenerations may thus be excited. *Want of food*, by diminishing the resisting power, also predisposes to disease, such as pneumonia and bronchitis. When the *proper proportions of the elements* of food are neglected, disease results, as *e.g.* scurvy from deficiency of fresh vegetables. Rickets, according to Dr. Cheadle, is due to want of fat, while gout is often provoked by a largely nitrogenous diet, and diabetes by an excessively saccharine one. *Particular foods* will immediately excite violent gastric catarrhs in some individuals, while others can bear them perfectly well. Clotted lumps of casein or starchy foods may irritate the alimentary canal of an infant, as well as, from their indigestibility, afford no nourishment. Many deaths ascribed to 'consumptive bowels' are due to this cause. Putrid food is an active poison. Certain kinds of *fish* are poisonous in themselves, and so may be shell-fish and crustaceans. The *Bothriocephalus latus* enters the body through the flesh of the pike; and, according to Hutchinson, leprosy is derived from salted fish. Diseased rye produces ergotism, and diseased maize pellagra. Flesh from animals suffering from 'trembles,' is believed to give rise to 'milk sickness'; trichinosis and the common forms of tape-worm result from the ingestion of beef or pork affected with the particular parasite in question; and some vegetable foods laden with salts of lime have been supposed to cause urinary calculi. Water and milk are prolific sources of mischief, through the impurities they so often contain. The drinking-waters of large towns are usually derived from rivers, and filtration is not a sufficient purification; or, if obtained from a purer source, they may become contaminated in transit or storage, so that disease may be excited by their use, especially typhoid fever. Scarlet fever, cholera, and malaria have, according to some, been traced to the same source. Many parasites thus find entrance, *e.g.* the ova of the *Tænia solium* (developing in the cystic stage), *Distoma hæmatobium*, *Ankylostoma duodenale*, *Filaria sanguinis hominis*, and Guinea-worm. Hydatids may be derived from the same source, or conveyed directly to the food from the nose or feces of an infected dog. Diarrhœa, dysentery, Oriental boil, and various forms of

metallic poisoning (lead, copper, arsenic, zinc, and mercury) sometimes owe their presence to the minute quantities taken in drinking-water. Goitre and cretinism have been attributed to inorganic impurity in water. Alcohol is a most extensive source of disease: it causes, when taken in excess, cerebral, gastric, intestinal, hepatic, and renal affections, and can lower the system so far as to predispose to other diseases. *See* ALCOHOLISM; ENTOZOA; and POISONOUS FOOD.

18. **Epidemic Diseases, Contagion, Malaria, Parasites, and Growths** are treated of under separate headings. It is now generally established that the diseases known as the acute specific diseases are mostly direct consequences of some contagium. So among the most common exciting causes of disease we must class the contagia of the several fevers, of syphilis, tubercle, and many others. *See* CONTAGION; PERSONAL HEALTH; and PUBLIC HEALTH.

J. PEARSON IRVINE.  
H. MONTAGUE MURRAY.

**DISEASE, Classification of.**—Various classifications of diseases, or systems of nosology, have been adopted by different writers, but it is beyond the province of this work to discuss these arrangements, neither of which fulfils all that is required, or can be regarded as satisfactory. All that can be done here is to point out the characters upon which the chief divisions of diseases are founded.

The first classification deserving of mention is that into (1) **General** and (2) **Local**. **General** diseases include those in which the whole system is involved from the commencement, and it comprehends as subdivisions: (a) The *acute specific fevers*, and certain other diseases due to the introduction of some special morbid agent into the body from without, or in some instances developed within the system—for example, typhus and typhoid fevers, scarlatina, small-pox, malarial fevers, hydrophobia, syphilis, pyæmia, and septicæmia. (b) The so-called *constitutional, cachectic, diathetic, or blood-diseases*, some of which seem to depend upon the production of deleterious elements within the system, which are capable of recognition, such as rheumatism and gout; while others are independent of any such obvious pathological causes, but are supposed to be severally associated with a peculiar dyscrasia or diathesis—for instance, cancer, tuberculosis, scurvy, rickets. **Local** diseases are those which primarily affect particular organs or tissues, each being liable to its own peculiar lesions. Thus we have diseases of the lungs, heart, stomach, liver, kidneys, brain, and the other organs; of the mucous membranes, serous or fibro-serous membranes, skin, periosteum, bone, and other structures. This division into general and local diseases is useful within proper limits, but it must be

remembered that general maladies are often revealed or accompanied by local lesions, and that complaints which are originally local often more or less speedily set up general disturbance. Moreover, it is still a question whether some maladies are to be regarded as general or local in the first instance. *See* SYMMETRY, IN RELATION TO DISEASE.

Another division of diseases, which applies more particularly to those which are of a local nature, is into (1) **Organic or Structural**, and (2) **Functional**. These terms are self-explanatory, the former implying that there is some organic change in the affected part, which we can discover and demonstrate; the latter indicating that there is mere functional disorder, which is independent of any recognisable lesion. That there are structural changes in many affections which are regarded as functional, is, however, highly probable, though our means of observation are not sufficiently powerful to enable us to detect them. In connexion with each organ, a special classification of its individual complaints under one or other of these primary headings is usually adopted, this subdivision depending upon the affections to which the particular organ is liable. As illustrations of functional disorders may be mentioned disturbed action of certain organs, as of the heart, causing palpitation; derangement of the secretory or excretory functions, as in the case of the stomach, liver, or kidneys; and many nervous disorders. Organic diseases are exemplified by inflammation and its consequences; alterations in growth and development; degenerations; malformations; and new-growths. In this work it has not been deemed advisable to describe the diseases of the several organs according to any definite scientific arrangement, but in some cases an alphabetical order has been adopted, while in others individual writers have had free scope to classify the affections of a particular organ according to their own judgment.

Again, diseases may be classified according to their causation and mode of origin. Thus they are divided into: (1) **Hereditary**, or those which are transmitted either directly from parents to children, or indirectly, as the result of a family taint; and (2) **Acquired**, or those which are developed anew in persons free from hereditary taint. When a morbid condition exists at birth, it is said to be **Congenital**. Other divisions, founded on an ætiological basis, are into: (1) **Contagious or Infectious**, and (2) **Non-contagious**; and into (1) **Specific**, or those diseases which are due to a specific cause, and (2) **Non-specific**.

There are other classifications of diseases, which need only be mentioned here. Thus, according to their intensity and duration, they are said to be: (1) **Acute**; (2) **Sub-acute**; or (3) **Chronic**. Another arrange-

ment, founded on their mode of progress, is into : (1) **Continuous** ; (2) **Periodical**, or affections which come on at more or less definite intervals ; (3) **Paroxysmal**, or those which are characterised by sudden or acute paroxysms ; and (4) **Recurrent**, or diseases which tend to recur. Lastly, according to their mode of distribution amongst communities or in districts, complaints are said to be : (1) **Sporadic** ; (2) **Epidemic** ; (3) **Endemic** ; and (4) **Pandemic**. The meanings of these terms are defined under their several headings, but they are sufficiently familiar as indicating the mode of distribution of the diseases to which they respectively belong.

With regard to the classification of diseases which is likely to be permanently adopted in the future for general use, it is probable that this will be founded on a pathological basis, and that, as our knowledge of morbid conditions and processes becomes more extensive, accurate, and definite, it may become possible to establish a system of nosology which will be both scientific and practically useful.

FREDERICK T. ROBERTS.

**DISEASE, Diagnosis of** ( $\delta\acute{\iota}\alpha$ , apart ; and  $\gamma\omega\acute{\sigma}\kappa\omega$ , I know).—SYNON. : Fr. *Diagnose* ; Ger. *Diagnose*.

**DEFINITION.**—Diagnosis is the art of recognising the presence of disease, and of distinguishing different diseases from each other. The term is also applied to the result obtained.

**GENERAL CONSIDERATIONS.**—The *general principles* only of diagnosis will be here discussed. *Special diagnoses* will be treated of in connexion with the several diseases to which they have reference.

In many respects diagnosis is a subject of great interest and importance. First, in a scientific point of view, it is essential that all knowledge should be accurate. Secondly, accuracy of diagnosis, founded upon a sound pathology, enables us to frame a scientific classification of disease in its diverse forms. Thirdly, it is by an accurate determination of the nature of the disease which may be present in any given case that we are able to anticipate its course, and to employ the right kind of remedies in its treatment. It is imperfection of diagnosis which leads in many instances to an under-estimate of the value of therapeutical agents ; for when the nature of a disease is mistaken we are led to employ improper and unsuitable remedies, the failure of which is then erroneously attributed to the inefficiency of the agents, and not to the unfitness of the treatment employed. If our diagnosis had been correct or complete, the remedy employed would more often have had the desired effect.

In order to arrive at a diagnosis we must study the phenomena or characters of each individual case, and then trace its connexion with those groups of symptoms which have

been previously recognised and described as belonging to special or distinct classes of disease. Assuming that the classification has been already made, we proceed to deal with the means which enable us to identify each individual case, and to connect it with previously classified forms of disease.

**MEANS OF DIAGNOSIS.**—To obtain accuracy in diagnosis we must be prepared with a knowledge of the several forms and the varieties which disease assumes ; we must be familiar also with the functions and structure of the several organs of the body in health. It is by observing and comparing the changes wrought by disease in these functions and structures that we are enabled to discover the presence of, and to determine the nature of, the morbid process going on. In forming, then, a diagnosis in any particular case, the physician must, as far as possible, keep in view the real or the ideal condition of the patient in a state of health. He must endeavour to place him in as natural a position as may be, and as little disturbed by the presence of his attendant, or by external circumstances, as possible. The physician must then obtain a history from the patient himself or from others of the incidence of the disease ; and having done this he must proceed to investigate for himself the condition of the patient.

1. *Previous history of the patient.*—The history implies of course a statement of the age and sex of the patient, as of his home and his employment—each of which may have a special relation to disease. It should also include an inquiry into the antecedent generations of the patient, and how far he may have any proclivity to congenital disease or malformation. This inquiry should have reference to both positive and negative facts. It should extend not only to the previous existence of disease in the family, but also to the absence of particular diseases or types of constitution. The patient's history should include a statement as regards the diseases and injuries from which he may previously have suffered ; the remedies used for them ; and the climatic and other influences to which he has been exposed. Nor must the physician neglect to ascertain the history of any children that the patient may have had, as the nature of disease from which the offspring have suffered in many instances throws light upon the health of the parent.

2. *History of present illness.*—The history of the present illness should include the determination of the date of its commencement ; its probable cause ; its earliest phenomena and its progress as influenced by external circumstances, including treatment.

3. *The present condition of the patient.*—Here we have to deal with two classes of phenomena ; namely (*a*) those feelings or facts of self-consciousness which the patient describes to us—*subjective phenomena* ; and

(b) those signs which we ourselves observe—*objective phenomena*.

(a) *Subjective phenomena*.—The patient describes to us his feelings—as of strength or weakness, of numbness, tingling or pain, of wakefulness or wandering; he can tell of affections of vision, of hearing, of smell, or of taste; of breathlessness, cough, palpitation, or of feelings of sinking or faintness; of difficulty of swallowing, thirst, loss of appetite, nausea or sickness, or various sensations and actions connected with the abdomen; of feelings associated with the genito-urinary organs, such as pain or difficulty in passing water; of cramps, spasms, or other alterations of sensation or motility; or of disturbances of sensibility and activity, &c. Each of these signs of deviation from health will have its own value and significance. The physician must at the same time carefully note how far the condition of the patient is in accordance with his statements, and whether there may not be present some reason or cause for concealment or for exaggeration.

(b) *Objective phenomena*.—In studying the objective phenomena connected with disease, the physician makes use of his special senses, assisted by the several instruments with which modern science has provided him.

First, in matters of *eyesight*, he sees the general aspect and expression of the patient, which will include the colour of the skin (such as may result from the fulness or emptiness of the blood-vessels, from the yellowness caused by jaundice, from the blueness of cyanosis, or from pigmentation, &c.); the presence and character of cutaneous eruptions (especially in the exanthemata); the expression proper, such as that of ease or suffering, and of depression or excitement; the conditions of obesity and plethora, or of wasting and bloodlessness. He will also observe the position of the patient—how he lies, or sits, or stands; how he breathes; the appearance of the eyes, the tongue, &c. Further, the sense of sight will be employed in determining conditions of a local or less general nature. Observation must be made of the size, the shape, and movements of parts, and of their expansion or contraction. With the aid of special instruments, such as the ophthalmoscope, the laryngoscope, the cystoscope, the various specula, sounds, &c., the physician will be able to examine parts of the body of the patient beyond the reach of the unassisted eye. The chest-measurer or the stethometer will render more exact the information already obtained by the eye and hand as to the size and mobility of parts. The use of each of the several instruments above mentioned, as a means of diagnosis, will be found described under the heads of their respective names, or in the article on **PHYSICAL EXAMINATION**.

The sense of *hearing* tells of the character

of the breathing, the voice and speech of the patient, including cough, hoarseness or aphonia, 'aphasia,' &c. But the ear is especially applied to the study by auscultation of the sounds produced in connexion with the heart, the lungs, and other organs. The signs thus elicited will be found fully described elsewhere.

The sense of *touch*, or feeling, will communicate a knowledge of the temperature, of moisture or dryness, of size, shape, elevation or depression, of smoothness or roughness, of the pulse or pulsation, vibration, fremitus, of extent of movement, resistance, softness or hardness, and of fluctuation. The accuracy of the results of these observations by touch may be tested by the use of the thermometer, the calipers, and the tape-measure.

The sense of *smell* aids diagnosis in certain cases. The general odour of the patient may be observed in small-pox, in rheumatism, and some wasting diseases (such as phthisis), and in syphilis; and the odour of particular parts and secretions, as the urine in diabetes, the fæces in jaundice, &c., and in cases of the use of certain drugs, or in poisoning. Information is also afforded by the odour of certain discharges, as in ozæna, leucorrhœa, cancer, &c.

The sense of *taste* is seldom employed in clinical investigation, but the physician may make use of the patient's taste, as in tasting the urine in diabetes.

*Further aids in diagnosis*.—Having thus summarily described the employment of the special senses in diagnosis, and given examples of their use, we may briefly mention some other agencies of more general application. The acuteness of the patient's sense of touch may be determined by the æsthesiometer. The capacity of the lungs may be measured by the spirometer; the strength of muscles by the dynamometer; the contractility of muscles by galvanism; and the force and character of the pulse are determined by the sphygmograph. Constant use is found by the microscope, the test-tube, the spectroscope, the endoscope, and polariscope, which aid in determining the character of the various secretions or morbid products that require to be submitted to investigation. The result of treatment may also be mentioned as an aid to diagnosis, as, for example, when an indurated sore yields to the use of mercury. Again, the knowledge that a person has been in a malarious district enables us to decide on the intermittent nature of certain symptoms that may be present. In some cases it may be necessary to render a patient insensible by anæsthetics, with a view to making a complete examination, or in investigating feigned diseases. The administration of small doses of charcoal has been suggested as a means of determining the presence of a passage through the bowels when more or less obstruction exists.

Such, then, are the means used for taking note of those deviations from health which occur in the several functions and structures of the body, and which constitute what are known as the *symptoms and signs of disease*; these are terms which will be found more specially treated of under the heads, DISEASE, Symptoms and Signs of; and PHYSICAL EXAMINATION.

THE DIFFICULTIES OF DIAGNOSIS.—It need scarcely be said here that the practice of diagnosis is not free from great difficulties. We know how hard it is to obtain in ordinary daily life a reliable account or description of any past or present event. There must be still greater difficulty in obtaining an accurate medical history of a patient's case. He has to tell of facts of which practically he may know much, but scientifically very little. He may be forgetful or ignorant on points about which we most need to be informed. He may be inclined to exaggerate or to suppress facts of material import. Nor are the difficulties less in regard to the *objective phenomena* with which we have to deal. The symptoms of a disease are not always so clear and definite as to mark its nature—that is, to be *pathognomonic*. They are more often slight, undefined, obscure, and to be found with difficulty. The symptoms of one disease may very closely resemble those of another, whilst those of the same disease will vary at different stages, and in different individuals. Again, the symptoms of a disease may be complicated by the co-existence of those of another disease; whilst a symptom sufficiently striking in itself may be common to, and present in, several different diseases. We need only mention, for example, feverishness, pain, cough, breathlessness, and blood-spitting.

These are some of the difficulties which he who has to study the operation of disease in life, has to contend with. He must come prepared for the duty with a knowledge, as we have already said, of the body, its structure and its functions in health, and with a knowledge, too, of those combinations of morbid actions which constitute special forms of disease. For, as regards this latter knowledge, all the observations made would remain as isolated phenomena if they could not in each case be grouped as constituting distinct diseases.

We have thus indicated the difficulties of obtaining accurate knowledge as regards both the subjective and objective phenomena. The difficulties are not less when the exercise of the intellectual and reasoning faculties is called upon to analyse, to compare, and to group these phenomena.

The physician may commence his inquiry by tracing up the history of the case and its several incidents, a method which is called the *synthetical*; or he may commence by ascertaining the present condition of the patient, and going as it were backwards in

his inquiry—a method which is known as the *analytical*. As a general rule, both methods are combined in the practice of diagnosis.

Observers can sometimes arrive at a *direct diagnosis*, aided by the presence of some characteristic symptom or sign of disease. When diseases which are essentially different have symptoms more or less common to both, the physician will have to institute a *comparison* between them, until he finds sufficient evidence, in the presence or in the absence of some distinctive symptom or sign, to satisfy him as to the nature of the disease which is present. By being able thus to trace the absence or the presence of a given symptom, he may be able to *exclude* the possibility of the existence of one or other of the diseases under investigation. These modes of investigation will be found fully illustrated in the diagnosis of the several diseases described throughout this work.

In conclusion, it must be remembered that these investigations, which call for the exercise of the highest mental faculties, should be conducted without prejudice and without haste. We should never be ready to accept as clear that which is obscure, as established that which is open to question; above all, we should remember that, though to err is human, it is our duty to endeavour to ascertain in each and every case, before commencing its treatment, what its real nature is, as far as it may be possible for us to do so. It cannot be too often repeated that the application of a right remedy depends on an accurate diagnosis, and that the prevention and the cure of disease are the aims and ultimate objects of our science.

RICHARD QUAIN.

DISEASE, Duration of.—The duration of a disease signifies the period which elapses between its onset and its termination, in whatever way this may take place. In some instances disease can hardly be said to have any duration, a sudden lesion occurring, which instantaneously, or in a very short time, destroys life; under such circumstances, however, some previous disease has usually existed, though perhaps without giving any clinical evidence of its presence, which determines the occurrence of the sudden event. This may be illustrated by some cases of apoplexy, and of rupture of the heart or of an aneurysm. Most affections, as regards their duration, come under one of the three categories already referred to under the CLASSIFICATION OF DISEASE—namely, *acute*, *subacute*, or *chronic*; but it does not serve any useful purpose to fix upon any definite limit of time as specially expressed by these several terms. See ACUTE; and CHRONIC.

*Acute* diseases are of limited duration, and in many of them this is remarkably uniform on the whole, as may be illustrated by the

acute specific fevers and acute idiopathic pneumonia. Even in such affections, however, there are deviations from the ordinary course, instances occurring in which the duration is longer or shorter than that usually observed, and this fact depends on various circumstances, of which the most obvious are the intensity of the disease in any particular case, the previous condition and surrounding circumstances of the patient, the occurrence of complications, and the treatment adopted. Complaints which are *sub-acute* as regards their duration may be exemplified by many cases of whooping cough and chorea, and by some cases of pleurisy, phthisis, pneumonia, gastric or enteric catarrh, and certain skin affections. A large number of diseases are *chronic* in their duration, and many of these when once established become permanent, whilst others are ultimately capable of being cured. As illustrations may be mentioned organic diseases of the heart, most cases of phthisis, cirrhosis of the liver, chronic Bright's disease, dyspepsia, many skin affections, and also morbid growths in various structures.

Some complaints, as regards their duration, can only belong to one or other of the groups just indicated, but a considerable proportion may in different cases be either acute, sub-acute, or chronic. Again, it must be borne in mind that a disease may be acute or even sudden in its origin, but afterwards may subside into a chronic malady. Certain affections are chronic as regards their entire duration, but are characterised by the occurrence at regular or irregular intervals of acute or even sudden attacks, lasting a more or less definite time, which course of events is exemplified by cases of ague, epilepsy, and asthma.

FREDERICK T. ROBERTS.

**DISEASE, Germs of.**—See GERMS OF DISEASE.

**DISEASE, Prognosis of** (*πρό*, before; and *γνώσκω*, I know).—SYNON.: Fr. *Prognostic*; Ger. *Prognose*.

**DEFINITION.**—Prognosis is the art of forecasting the progress and termination of any given case of disease. The term is also applied to the foreknowledge thus obtained.

**GENERAL CONSIDERATIONS.**—It is a matter of interest and often of great importance to be able to indicate with precision how a case of disease or injury will be likely to advance and terminate. This question must be always present to the physician's mind; and it can rarely be absent from that of the patient and of those who are interested in his well-being. It can thus be easily seen how much depends upon the answer of the physician to the questions constantly proposed to him, How long is this illness likely to last? How is it likely to terminate? If in recovery, will the recovery be complete or partial? If in death, when and how?

**FOUNDATIONS OF PROGNOSIS.**—The knowledge which can give trustworthy answers to such questions as the preceding must be founded upon an accurate diagnosis of the nature of the disease from which the individual is suffering; upon the capability of remedies to control it; and, lastly, upon an estimate of the constitutional and vital powers of the patient.

First, as regards the *nature of the disease*. Some diseases which are mild in their nature run a definite course and end favourably: take, for example, a simple catarrh. Others commence with great intensity, and come to a favourable or unfavourable termination very rapidly: for instance, Asiatic cholera, of which many of the subjects die in less than twenty-four hours from the time of their first becoming manifestly ill. A third group, such as typhus, typhoid fever, and certain of the exanthemata, run a longer and more defined course, seldom terminating in death except after the lapse of many days, nor in recovery except after a period of weeks. Another class of maladies, chronic in character, rarely acute, such as tuberculous diseases of the lungs, render the patient more or less an invalid so long as he lives, and generally end fatally. The like observation will apply to the so-called malignant diseases.

Secondly, the *intensity of the particular attack* affords further grounds for prognosticating the result. Thus, in a fever, great prostration, high temperature, and rapid pulse, indicative of the severity of the disease, must lead to the formation of an unfavourable prognosis; just as great debility and wasting, with disturbance of the nutritive functions generally, would indicate a like result in chronic diseases.

Thirdly, with regard to local diseases or complications, whatever the nature of the disease may be, the *organ affected* must form an important element in prognosis. Thus disease of the brain, or of the heart, or of the lungs, or, in a lesser degree, of other viscera, must, even when not specially severe, be looked upon as affording grounds for anxiety, from a prognostic point of view.

Fourthly, as regards the *constitution, age, and sex of the patient*, it may be safely anticipated that in a patient with a good constitution the prognosis will be more favourable than in a person with a feeble or broken-down constitution. Persons whose vital powers are unimpaired will resist disease, and recover under circumstances which would be fatal to other individuals, in whom, on the one hand, plethoric habits may predispose to acute and rapid changes, or who, on the other hand, by degeneration of tissues or by the existence of chronic disease may be rendered liable to succumb, and that rapidly, to morbid influences which healthier textures could resist and overcome.

Disease is badly borne by the very young

and the very old. In very young children disease rapidly runs its course, favourably or unfavourably. The aged have little power of reaction or of resistance; and disease in them, though less pronounced, more frequently ends unfavourably. In middle life, disease may be expected to assume an acute or sthenic form.

As a rule, sex has little influence on the prognosis of disease, except that usually diseases of equal severity are more amenable to treatment in females than in males. Nervous symptoms are, however, more easily developed in women, exaggerating a condition that might not otherwise be unfavourable. Menstruation, pregnancy, parturition, and lactation have all a certain amount of influence, sometimes favourable and sometimes the reverse, on disease in the female.

Fifthly, with respect to *treatment*: a more or less favourable prognosis may be founded upon the fact that the patient can enjoy all the advantages afforded by rest, diet, change of climate, &c., which may not be available under other circumstances for like cases. It is well known that there are some remedies which have a specific effect upon certain diseases, as quinine upon intermittent fever; mercury in some manifestations of syphilis; iodide of potassium in certain stages of the same disease; and colchicum in gout. In such cases a much more favourable prognosis can, of course, be given than in those for which no such specific remedies are known to exist. Experience, however, tells us that favourable results follow in many other cases in which suitable though not actually specific remedies can be applied.

Taking into consideration, then, the above conditions—the nature, the intensity, and the seat of the disease; the constitution, the resisting power, the age, and the sex of the patient; and the possibility of applying suitable and efficient remedies—we are able, in a large number of cases, to arrive at an accurate conclusion as to what the course and result of a disease will be.

**DIFFICULTIES OF PROGNOSIS.**—Still, to arrive at an accurate prognosis is often most difficult. Disease is not always identical in its character, nor definite in its progress or results. The constitutions of individuals vary, and it is often very difficult to measure their powers of resistance. Remedies, too, vary in their action and their operation; and sometimes we are deceived in the best-founded conclusion as to the results that they can accomplish. There are few physicians who cannot recount errors of prognosis made by themselves or by their colleagues. Many persons now live who were doomed to die; and many persons have died whose death was not anticipated. It is the duty of the physician, when asked for his opinion, to state it honestly, but with great discretion, and in general with as much hope as is

fairly admissible. He must be guarded as to the manner in which his opinion is communicated to the patient, for there are many individuals whose temperament is such that the progress of their disease would be greatly influenced for good or for evil by the expression of a favourable or of an unfavourable opinion. At the same time, the physician must avoid deceit; and if there be risk or danger in communicating an unfavourable prognosis to the patient, he must at least communicate it to some judicious individual amongst the patient's friends. Altogether, too much caution cannot be exercised in stating, in any obscure case, what its progress and result will be. There are many cases in which the medical attendant will be justified in replying that he is a physician, and not a prophet. He cannot always foretell results, his aim and object ever being to mitigate the patient's suffering, to prolong life, and to cure the disease if possible; full often to profess or to do more than this is beyond his art.

RICHARD QUAIN.

### DISEASE, Symptoms and Signs of.

When disease affects any of the functions or structures of the body it produces certain altered actions or changes, which, when observed during life, become evidences of its presence and often of its nature, and which then are called the symptoms and signs of disease.

The terms *symptom* and *sign* are often used synonymously, though the derivations of the words are by no means the same. *Symptom*, according to its derivation (*σύμπτωμα* = a coincidence), means simply a *coincidence*; that is to say, it coincides with the presence of certain phenomena. The term *sign* (from *signum*) is more distinctive, and seems more directly to point to some special or peculiar condition. Recently, however, an attempt has been made to give a more special meaning to these terms. *Symptom*, more especially if it be characterised by the prefix *vital*, is intended to refer to modifications of functions, or to such *subjective* phenomena as we can learn from the patient's account of his feelings. On the other hand, the term *sign*, more markedly with the prefix *physical*, indicates those morbid changes which are *objective*, or may be recognised by the senses of the physician, assisted by other appliances.

It would possibly be well if the meanings of the words—symptoms and signs of disease—as above stated, were to come into general use; but there are many difficulties in the way. For example, if the ear be applied to the chest in the case of incompetence of the aortic valves, we hear a murmur, and we say that there are 'physical signs' of aortic valve imperfection; but the locomotive pulse, and its peculiar beat, would by many be called a 'symptom' of incompetence of the aortic

valves. It is therefore extremely difficult to draw the distinction between the terms 'symptom' and 'sign.'

By whatever name these phenomena may be called, we must rely upon them as the chief means by which we are enabled to form our diagnosis. The more accurate and complete our knowledge of the functions of the body and of its component parts, and the more capable we are of interpreting, with all the completeness possible, the changes produced by disease, the more accurate will be our diagnosis as to its presence and its nature. How these phenomena may be best observed will be found discussed under the articles on DISEASE, Diagnosis of; and PHYSICAL EXAMINATION.

RICHARD QUAIN.

**DISEASE, Terminations of.**—The terminations of a disease must be regarded both from a *pathological* and from a *clinical* point of view.

Each *pathological* process or condition has modes of ending peculiar to itself, but it is beyond the province of this article to discuss these at any length, and one or two illustrations must suffice. Thus inflammation may terminate by resolution; by the formation of different effusions or exudations; or by causing suppuration, softening, induration, ulceration, or gangrene. Fever, if it end favourably, may terminate by crisis, lysis, or a combination of these modes, or in an irregular fashion. An effusion of blood may remain, more or less altered; may become organised; may soften and undergo a puriform change; may form a cyst; or may be altogether absorbed.

The *clinical* terminations of diseases are highly important, and demand more consideration. In the first place, a disease frequently terminates in the *death* of the patient. This event may take place suddenly or very rapidly, from the occurrence of some serious lesion, or of grave functional disorder of an organ essential for carrying on the phenomena which constitute life. In other cases death is the termination of a more or less acute illness, either affecting a person previously in the enjoyment of good health; or, what is not uncommon, being the consummation of a chronic malady, which has existed for a longer or shorter period. In still other instances, death is a slow and chronic process, the patient gradually sinking, several causes and morbid conditions often ultimately contributing to the fatal event. The modes in which death occurs are described elsewhere, and therefore need not be discussed in this article. See DEATH, Modes of.

In the next place, a large proportion of cases of disease end in *complete* and *entire* recovery, the patients being restored to their previous state of health, and no organic mischief remaining. This result may be ex-

pected in most of the ailments or functional disorders which are of such common occurrence, provided proper treatment is carried out. Again, the great majority of cases of acute disease terminate in complete recovery, taking them in the mass, though several affections of this class, when they do not prove fatal, are liable to leave behind more or less serious deterioration of the general health, or even actual organic disease. In this class of cases, when recovery does ensue, it is usually only after a more or less prolonged period of convalescence. Chronic complaints, if they are of a structural nature, cannot in most instances end in complete recovery, although to all appearance the patient may often be quite restored to health. Even in these cases, however, an actual cure may sometimes be effected, and that after a disease has had a prolonged duration. This is illustrated by several chronic skin-affections, syphilis, and chronic inflammation of mucous surfaces. Or it may happen that the patient recovers perfectly, only with the destruction of some structure which is not essential to life, such as a group of lymphatic glands.

Thirdly, *partial* or *incomplete* recovery is a very common mode of termination. This is observed in many cases of acute disease, where either the patient remains permanently in a state of general ill-health, without any actual structural lesion being discoverable; or some positive organic affection has been established, of which phthisis remaining after acute pneumonia, or cardiac disease following acute rheumatism, afford apt illustrations. An attack of an acute malady may also serve to bring out some latent constitutional predisposition; or may leave the patient in such a condition that certain so-called constitutional maladies are readily originated from slight causes. Partial recovery, amounting often to very marked improvement, may take place in many serious diseases of a chronic nature. This is illustrated by numerous cases of pulmonary consumption, in which disease great improvement is often observed, not only as regards the symptoms, but also in the local lesions, so much so that patients not uncommonly regard themselves as cured. Again, there are some complaints in which apparent recovery is brought about, but a tendency to recurrence remains, either without any obvious reason or from slight causes. Such affections are exemplified by ague, asthma, neuralgia, intestinal catarrh, bronchitis, and certain skin-diseases. As instances of incomplete recovery may be also mentioned the cure of some prominent symptom or symptoms, while the disease which originates these phenomena continues unaltered. Thus, it may be possible to get rid of ascites, which the patient regards as the disease from which he suffers, while cirrhosis of the liver, upon which the ascites depends,

is a permanent condition; extensive dropsy and other symptoms associated with cardiac disease may also be got rid of, while the organic mischief still remains. Sudden lesions may terminate in partial recovery. For instance, a case in which a sudden hæmorrhage into the brain has occurred, attended with marked apoplectic and paralytic symptoms, not uncommonly improves remarkably in course of time, the clot being more or less absorbed. Some complaints, which are usually sudden in their onset, may apparently be recovered from completely, but sometimes set up conditions which ultimately lead to permanent disease. Thus the passage of a gall-stone or of a renal calculus may excite such irritation as to cause an inflammatory process to be set up, which may induce permanent mischief, such as closure of the bile-duct or of the ureter in the several instances, and the effects may not be perceptible until a considerable interval has elapsed.

Lastly, it must be remarked that some affections can hardly be said to have any termination. They continue during the life of the individual, perhaps interfering but little or not at all with the health, or at all events not in any way contributing to the death of the patient, when that event does happen. This applies to many of the ailments from which people suffer; as well as to many chronic organic diseases not in themselves serious or giving rise to any important symptoms, and not implicating structures essential to life.

FREDERICK T. ROBERTS.

**DISEASE, Treatment of.**—This term has reference to the means by which disease may be prevented—*prophylactic* or *preventive* treatment; or its effects counteracted when it occurs—*remedial* or *curative* treatment.

1. **Preventive Treatment** will be found discussed under the heads—**CONTAGION; CLIMATE; DISEASE, Causes of; DISINFECTION; MALARIA; PERSONAL HEALTH; PUBLIC HEALTH; VACCINATION, &c.**; as well as in the several articles treating of special diseases. It is therefore unnecessary to say more upon the subject in this place.

2. **Curative Treatment.**—Bearing in mind that disease is a deviation from health in the functions or component materials of the body, it must be remembered that there is in organised bodies a tendency to maintain their healthy function and structure, and in case of disease or injury to resume it. This is especially manifest in the lower types of animals, many of which when mutilated are capable of resuming more or less completely their original form, to the extent even of the restoration of lost parts. In man and the higher animals this power of complete restoration is confined to the elementary cells and least complex structures

of which the body consists; the more complex tissues are not reproduced, nor are lost parts restored. There is, however, in man, as in all organised beings, a tendency to rectify deviations from health, and to restore the organisation to its normal condition. To remove or subdue the causes of disease, and to aid this restorative power in the establishment of healthy function and structure, is for the cure of disease the most philosophical indication that can be adopted. But our knowledge of disease and of remedial agents is not sufficient to enable us always to carry out these principles. As the treatment of disease has been directed sometimes to the one object and sometimes to the other, frequently to neither, it has given origin to a great variety of systems or methods of practice. Thus in the earliest history of the healing art, means the most diverse were used for the relief of suffering. Sometimes the suffering or the disease yielded whilst these means were being employed; and it was concluded, on very insufficient grounds, that these agents had 'cured' the disease. Persons who had felt, as they supposed, the beneficial effects of these particular remedies, communicated them to others as the result of their experience; and thus was established what has been known in Medicine as—

*Empiricism.*—This mode of practice has its advantages and its disadvantages. When aided by accurate knowledge and discrimination it often leads to satisfactory results; and many remedies suggested by experience, and that alone, are now found to be in accord with our more advanced scientific knowledge; take, for example, the use of mercury in syphilis, which, though long used empirically, is now known to act by its control over the nutrition of young cellular growths. So also with respect to quinine and other remedies of now established usefulness. On the other hand, mere empiricism, when vaguely applied, taints and damages to this day the treatment of disease. It is this practice which, for example, suggests opium to quiet a cough or a colic, without reference to the cause of the one or the other, and when an expectorant or a purgative would have been the suitable remedy; and it is this empiricism which does such harm in the hands of amateur practitioners, leading them to recommend for the relief of symptoms, remedies which they supposed had relieved like symptoms in other cases, however different the real nature or causes of these symptoms may have been.

*Rational treatment.*—On the other hand, modern science endeavours to take cognisance of the nature of disease, and also of the specific action of remedies; it seeks to counteract the operation of the one by the influence of the other. This constitutes the rational treatment of disease. To extend this system should be the object of the scientific practitioner. On

the one side, it is his duty to study the nature of disease itself, its causes, and their effects; on the other, to study the action of various agents on the living body in health and in disease; and, if possible, to trace how far the one is capable of combating and subduing the other. This study of scientific therapeutics is of comparatively recent date, and is now pursued with great zeal. The results already arrived at are alike satisfactory and encouraging. As rational treatment becomes more firmly established, scientific medicine will take a more elevated and nobler position. The modes or methods by which the two great principles just alluded to, the foundations as they are of the healing art, have been applied are extremely various, and, although these different methods may be traced to the one or to the other, they have received distinctive names, according as they are marked by some special characteristic. A few of these modes of treatment may be briefly enumerated.

1. *Expectant treatment* is founded on the principle that the restorative power of nature should be allowed entire freedom of action, the practitioner neither assisting nor interfering with its operation.

2. What is called *homœopathic treatment* would by some persons be included under the preceding head. It proposes to treat disease by giving in infinitesimal doses substances that are supposed to be capable of producing a diseased condition like that which they are intended to cure. It may be described in the words of Molière, who wrote long before Hahnemann, the inventor of homœopathy, as *l'art d'amuser le malade pendant que la nature guérit*. There is no doubt that in this and in similar methods of treatment, the imagination plays an active and useful part.

3. *Palliative treatment* consists simply in the adoption of means which are calculated to soothe, and to lessen suffering, and thereby to prolong life when the cure of disease is not possible.

4. *Stimulant treatment* is founded on a doctrine which regards most forms of disease as associated with or dependent on a lowered state of the vital powers, and which teaches that in such cases the free use of stimulants is the practice most to be relied on. See **STIMULANTS**.

5. *Antiphlogistic treatment* is the opposite of the preceding. It recognises in many forms of disease increased nervous excitement and vascular fulness, which are to be remedied by depressing agencies, such as low diet, bleeding, purgation, &c. See **BLOOD**, **Abstraction of**; and **DEPLETION**.

6. *Purgative, diaphoretic*, or otherwise *eliminative* treatment aims at removing by the intestinal mucous membrane, by the skin, or by the secreting glands, respectively, certain morbid matters; and thus allowing the re-

storative power of the system to operate more efficiently. See **PURGATIVES**, &c.

7. The *Water cure*, including baths, acts partly on the principle of elimination, partly by exerting a tonic influence. See **BATHS**; and **HYDRO-THERAPEUTICS**.

8. *Revulsive treatment* acts by producing counter-irritation by means of blisters, setons, issues, and the like. See **COUNTER-IRRITANTS**; **REVULSENTS**, &c.

9. *Antiseptic treatment* aims at destroying the germs of disease, whether on the surface of, or within, the body. See **ANTISEPTIC TREATMENT**; and **DISINFECTION**.

10. *Dietetic treatment* constitutes a greater or less portion of all modes of treatment. It implies a reference to the kind of food which is adapted to the circumstances in which the patient is placed, and which is suitable in the disease from which he is suffering. See **DIET**.

11. The treatment by *climate* operates more or less by removing the patient beyond the region of noxious influences, and placing him in circumstances which promote healthy action of the several functions.

It is well known that under these several and varied modes of treatment disease may yield and patients may get well. Hence it has been said that, as different means are made use of to obtain a single result, the treatment of disease can never be absolutely scientific. Phthisis is pointed to, for example, as a disease which one person seeks to relieve by cod-liver oil, another by climate, a third by tonics, a fourth by sedatives, a fifth by attention to the digestive organs, and a sixth by counter-irritation. We need scarcely say that the disease bearing the name of phthisis is an aggregate of phenomena or conditions, the relief of any one of which may lead to the amelioration of the others. Thus the general health might be improved by climate, and with it all the other symptoms. Cod-liver oil, with remedies calculated to improve the digestion, may lead to healthy nutrition, and thus to a mitigation of all the symptoms. The like remark applies to the other agencies mentioned. The treatment of disease must not, then, be condemned as unscientific because it cannot remedy a variety of morbid states by a single agent, but would aim, on still strictly scientific principles, by different agencies to overcome disease, the effects of which are manifested in different forms.

In conclusion, it may be repeated that the end and aim of the practitioner should be, if possible, firstly, to discover the cause or causes on which the disease depends, and to remove or counteract them if practicable; and, secondly, to endeavour, by every available means, to restore to health the functions of the body, and with that object to guide and assist Nature, but never to thwart her operations.

RICHARD QUAIN.

**DISEASES, Types and Varieties of.**—In the case of many diseases more or less distinct *varieties* are recognised, which in some instances constitute well-defined *types*. It is important to understand the precise significance of these terms in different cases.

In the first place the varieties of a particular affection may be founded upon diversities observed in its *clinical history*. Thus, according to the intensity of the symptoms and their duration, a large number of complaints are, as has already been pointed out, divided into acute, sub-acute, and chronic varieties. Again, many diseases, while presenting in the majority of cases a certain group of symptoms, upon which their general clinical description is founded, exhibit striking differences in the exact nature of the phenomena observed, as well as in their gravity, when the mass of cases is taken into account, and on these differences *varieties* or *types* are founded. This is well-exemplified by some of the acute specific fevers, such as typhoid fever, scarlatina, measles, and small-pox. Of these affections several varieties are described, dependent upon the severity of the symptoms, the nature of those which are most prominent, or the characters of the eruption.

In the next place, the classification of a disease into varieties may be founded upon a *pathological* basis. For instance, pulmonary phthisis may, in the writer's opinion, arise from different morbid processes, and many attempts have been made to arrange the cases of this disease into corresponding groups. Illustrations of these pathological varieties are also found in the different forms of cancer; in the varieties of pneumonia, of laryngitis, and of fatty disease of the heart; and in the classification of serous inflammations according to their morbid products, such as fibrinous, serous, purulent, &c. Again, such a pathological condition as dropsy or fever may be divided into varieties. Thus dropsy is arranged according to its situation and distribution, as anasarca, ascites, &c.; or according to its pathological cause, whether cardiac, pulmonary, hepatic, &c. Fever is recognised as having several important types, founded upon its intensity, its course, and the exact nature of the phenomena accompanying the pyrexial state.

Another division of a disease into varieties is *ætiological*, the cases being grouped according to their causation, either the immediate pathological or the more remote exciting causes being employed as the basis of division. Thus we have the different forms of meningitis (simple, tubercular, rheumatic, &c.); the ætiological varieties of pleurisy or peritonitis (idiopathic, traumatic, perforative, tubercular, secondary, &c.); those of joint-inflammation (simple, rheumatic, gouty, serofulous, &c.); or those of intestinal obstruction. Ætiological varieties are often at

the same time characterised by differences in the pathological results and products.

Lastly, it must be mentioned that sometimes a certain *group of symptoms* is summed up for convenience under some single term, which symptoms really depend upon very different morbid conditions and causes; and therefore it often becomes necessary to classify affections thus named into varieties. Dyspepsia, neuralgia, apoplexy, epilepsy, and paralysis will afford illustrations of such an arrangement.

FREDERICK T. ROBERTS.

**DISINFECTION.**—SYNON.: Fr. *Désinfection*; Ger. *Desinficieren*.

**DEFINITION.**—Disinfection, in the proper sense of the term, means any process by which the contagium of a given disease may be destroyed or be rendered inert.

Disinfectants, however, are used in practice for several objects, and in consequence the term has often been vaguely applied to the use of heat or chemical means for preventing the generation or for the destruction of noxious agents, whether products of specific disease or not. In this vague and erroneous sense disinfectants have been confounded with *deodorants*, which merely cover or destroy offensive odours without affecting the contagia; and with *antiseptics*, which 'are fatal to the growth and multiplication of microzymes.' Following the line indicated by the late Dr. Baxter in his valuable report on an experimental study of certain disinfectants, it is necessary to distinguish—

1. The true meaning of the word—that of acting on the specific poisons of communicable diseases in such a way as prevents their spreading.

2. That of acting upon organic substances in such a way as renders them less liable to undergo molecular change and decomposition, whether spontaneously or under the influence of catalytic agents, as in the case of emulsin upon amygdalin; or under the influence of living organisms, such as are connected with fermentation and putrefaction. Examples of this kind of action are seen in salting meat, and in preserving small animals in weak solutions of carbolic acid.

3. That of preventing or arresting decomposition by killing the torulæ associated with fermentation in slightly acid media, or the bacteroid organisms associated with putrefaction in neutral or alkaline media. Properly speaking, this is the action of an antiseptic, but the relative power of disinfectants has been largely estimated by their efficacy in this respect—partly, no doubt, because antiseptic power is desirable in a disinfectant, but chiefly because it is so difficult to submit disinfectants to their proper test by experimenting upon contagium. Recent experiments upon inhibition of growth of pathogenic bacteria show that mercuric chloride,

allyl alcohol, oil of mustard, and certain other ethereal oils prevent the growth of anthrax bacilli in extreme dilution. Quinine, two parts per thousand, prevents the development of anthrax bacilli or that of the spirochæta of relapsing fever.

4. That of the destruction of the noxious products of the metabolism of dead organic matter, however brought about. These products consist chiefly of gases or vapours, many of which, such as sulphuretted hydrogen, ammonia, and sulphide of ammonium, are easily destroyed by appropriate agents, even when used in a very dilute state; and success in this respect is no proof of the value of a disinfectant in its true sense, though the power of destroying such emanations is possessed by almost all disinfectants of practical utility.

GENERAL REMARKS.—It has been proved that the contagia of several diseases must consist of minute solid particles, for they are neither soluble, nor diffusible, nor volatile; they can be seen with the microscope, and can be cultivated out of the body; and we may infer that all other contagia are particulate likewise. A characteristic of contagium, due to its particulate nature, is that dilution lessens the chance of infection, but has little effect upon the case if the disease be taken. There is either no effect at all, or a full specific effect. Contagium particles are apt to exist as clouds in air, water, or milk, instead of being equally distributed throughout; and this bears upon practical disinfection. The particulate and non-gaseous form of contagium floating in the atmosphere prevents it from being absorbed by any liquid or solid disinfectant which does not wash or come into intimate contact with every portion of the air, and as this is impracticable, infected air can only be purified by gaseous disinfectants, such as sulphurous acid or chlorine. Disinfectants of this kind, to be effective, must be present in such a quantity as is incompatible with the existence of human beings. From this it follows that saucers of disinfecting fluids, or irritating vapours and gases in the sick-room, are merely a useless annoyance to the patient, except in so far as they may be desired as deodorants. The best method for dealing with infected air is to replace it by ventilation, especially by means of *ventilating* open fire-places. The proper use of volatile disinfectants is the purification of walls, ceilings, and inaccessible places; and for this purpose, if possible, enough should be used to saturate the atmosphere, remembering that the virulent particles are most likely protected by being buried in a bit of epithelium or surrounded by an albuminous envelope.<sup>1</sup>

<sup>1</sup> Though disinfectant or antiseptic gases of such strength as can be tolerated in the sick-room are utterly inept as regards useful effect upon contagium, it is just possible that they may be of service in

The nature of the medium in which contagious particles are suspended has the most important bearing upon the selection of a disinfectant. The presence of albumen is found to protect septic germs to a considerable extent against the action of permanganate of potassium and chlorine, but has little or no influence upon the action of sulphurous acid and carbolic acid. A striking instance of this was noticed by Schill and Fischer, who found that fresh tubercular sputum containing bacilli and spores was not disinfected in twenty-four hours by an equal volume of solution of mercuric chloride 1 in 500, though an equal volume of 5 per cent. carbolic acid solution accomplished the disinfection in that time. This possibly explains in part the contradictory results obtained from experiments with disinfectants, and also shows the necessity of thoroughly mixing disinfectants with liquids or substances to be disinfected.

Some pathogenic organisms, such as the bacilli of anthrax, are very easily killed, while others, such as the spores of the same bacilli, are extremely resistant. It is possible that in many cases the contagium may be of a species or at a stage when it can be readily destroyed by a feeble disinfectant, but in the absence of knowledge prudence requires the opposite assumption.

A cardinal principle in disinfection is that it should be carried out at the source, or as near the source, of the contagion as possible. Inunction with lard, with carbolised oil (1 to 40), or with glycerine, to clog epithelial scales, and regular washing and change of clothing, will do much to protect the purity of the air against contagium proceeding from the skin in such diseases as scarlet fever and small-pox. One part of ethereal solution of peroxide of hydrogen to eight parts of lard is an excellent application for the skin in typhus. Discharges from the mouth, nose, and bowels, as well as the urine, should be received in vessels containing disinfecting solutions to cover them and give protection to the air; and then larger quantities, or more concentrated solutions, as the case may be, should be thoroughly incorporated with the discharges before they are removed from the original vessels.

Of all agencies for preventing the spread of communicable disease, cleanliness is one of the most important. Facts have been adduced pointing to the conclusion that filth, when undergoing change of a fermentative or putrefactive nature, is in a condition the most conducive to the extension of infectious

destroying or rendering incapable of change the organic matters evolved from the skin and lungs, which are always very noxious, and may be especially so in disease. These organic matters are necessarily more or less re-breathed unless the patient be placed in a current of air. If ventilation sufficient to prevent all odour cannot be provided, then some gas, such as chlorine or ozone, that will destroy the cause of the odour, is certainly desirable.

disease. This is probably explained by the rapid multiplication of any specific disease-organisms contained in the filth. Pending the removal of accumulations of dirt, and for the protection of workmen, fermentation may be delayed by the application of crude carbolic acid, or solution of mercuric chloride.

The ultimate fate of contagium is to be destroyed by putrefaction, and this appears invariably to destroy its specific infective power. In certain cases where real disinfection is impracticable, as in dealing with the accumulation of manure and litter from a number of animals suffering from cattle-plague, the natural processes may be hastened by stacking the material so that it shall 'heat,' or may even be destroyed by spontaneous combustion. Certainly putrefaction should not be delayed by small additions of disinfectants, which cannot accomplish the destruction of all noxious matter present.

**SPECIAL DISINFECTANTS.—1. Heat.**—Heat, dry or moist, is perhaps the best disinfectant we possess. High temperature and length of exposure are, to a certain extent, mutually compensatory, but it appears that a temperature below 140° F. (60° C.) will not disinfect vaccine even with long exposure. Tyndall points out that some germs seem to be in a dormant condition, in which they resist the action of heat unless applied very long or intermittently, so as to start their vitality into growth, when they are easily killed. Disinfection by dry heat (which is, however, best for the destruction of insect vermin) is now being superseded by steam. Dry heat penetrates so slowly into fabrics that they are injured before a disinfecting temperature is attained throughout. The presence of moisture assists the action of hot air, as it likewise does that of gaseous SO<sub>2</sub> and Cl. Koch found that 1½ hour's exposure within test-tubes in an oven containing air saturated with moisture at a temperature from 230° F. to 245° F. destroyed sporeless micro-organisms and spores of fungi, but did not affect spores of the bacilli of potato, hay, and anthrax, nor the spores of garden earth. These were all destroyed by three hours' exposure to 284° F. A current of steam at 212° F. destroyed spores of earth, anthrax, and tubercle-bacilli in less than fifteen minutes, when they were freely exposed. Steam penetrates clothing quickly, but may take some time to raise the temperature of sheltered articles, especially if wet. Modern steam-disinfectors are made with a jacket space to which live steam may be admitted, so that the internal chamber may be used as a simple hot-air chamber where steam is not admitted directly to it. With a proper apparatus the articles come out dry. Steam instantly ruins leather, and fixes permanently any stains of blood or discharges. Stained clothing should be previously soaked in cold water and the water afterwards disinfected by mercuric chloride.

It has been abundantly proved that ordinary boiling and washing will completely disinfect the linen of a fever hospital. The water should attain the full temperature of 212° F.

Nearly all the chemical agents used in disinfection are mutually incompatible, and therefore only one should be used at a time.

**2. Carbolic Acid.**—A solution of this substance of the strength of 5 per cent., or 1 in 20, is the only one fit for use in disinfection. For steeping fine clothing a solution should be made from crystals. The solution generally useful is that obtained by making up one gallon of crude 80 per cent. acid to sixteen gallons with water. The results of the experiments of Baxter and others prove that 'no virulent liquid can be considered disinfected by carbolic acid unless it contain at least 2 per cent. by weight of the pure acid.' More recent experiments show that in some cases this proportion of carbolic acid is not nearly enough. Anthrax spores required to soak over twenty-four hours in 5 per cent. carbolic solution to be destroyed, though blood containing anthrax bacilli was almost instantly sterilised by a 1 per cent. solution. With our present knowledge it would be safer to employ stronger acid, or in any case to use a large quantity of 5 per cent. Combinations of carbolic acid are greatly inferior to the pure acid, and its solutions in oil and alcohol are found to be absolutely inert. Thymol and salicylic acid are affected in the same way by solution in oil. Judging from the light of experiments, carbolic acid vapour is quite useless. Very small quantities of the liquid acid mixed with organic fluids enable them to remain fresh and resist decay for a long time. So little as one-fifth per cent. preserves milk. It is obvious then that small quantities of this disinfectant, instead of destroying contagium, may actually preserve its activity, when otherwise it would have succumbed to the action of natural agencies. This danger may accompany the limited use of any disinfectant that has a 'pickling' or preservative action in small quantity. Owing partly to the volatility of carbolic acid, which removes it in time, and partly to the peculiarity of its action, another danger attends its use in anything short of full strength and large quantity when applied to kill contagium. The acid may, for a time, deprive the contagium of its infective power without permanently abolishing it, and the virulent properties may be regained whenever the acid has evaporated. This has been proved experimentally by Dr. Dougall, of Glasgow, who found that vaccine mixed with carbolic acid (1 in 50) *regained* its infective power after 10 days' exposure to the air.

For removing odour from the hands after working in the dissecting-room, a 1 per cent. solution of carbolic acid is superior in efficacy

to permanganate of potassium, even when strong enough to stain the skin, and is also preferable to chlorinated lime.

**3. Sulphur Dioxide.**—The aqueous solution of this substance contains sulphurous acid. It destroys sulphuretted hydrogen thus,  $\text{SO}_2 + 2\text{H}_2\text{S} = 2\text{H}_2\text{O} + \text{S}_3$ , and combines with ammonia. For aerial disinfection the best plan is simply to burn sulphur in very large quantities. Even in presence of abundant moisture, Koch pronounces  $\text{SO}_2$  useless for practical disinfection. By volume, 10·5 per cent. of the gas in a room failed to affect spores of anthrax, &c., in twenty-four hours, whether wet or dry. About 3 per cent. in forty-eight hours had not destroyed the vitality of micrococcus prodigiosus, pink yeast, or bacteria of blue pus. In an ordinary room a gaseous disinfectant rapidly disappears. On the other hand, Cyrus Edson tries, from the statistics of the Health Department, N.Y., to prove the efficacy of  $\text{SO}_2$  fumigation (3 lbs. sulphur to 1,000 cubic feet) in small-pox, scarlet fever, and to a less extent in measles and diphtheria-infected houses. The statistics are not convincing to the present writer. Sulphur dioxide destroys the activity of dry vaccine on points very rapidly, and even when much diluted stops the amoeboid movements of living cells, kills vibrios, and acts deleteriously on vegetation. Sulphur dioxide preserves meat and other substances, when in closed vessels, for very long periods. It bleaches vegetable colours, attacks iron, and is absorbed by cloth and leather—facts to be remembered in practical disinfection. 1 lb. of sulphur, when burned, produces 11·7 cubic feet of sulphur dioxide gas.

**4. Chlorine.**—Chlorine is most easily obtained from 'chloride of lime' or bleaching powder, by adding hydrochloric or sulphuric acid. Exact proportions cannot be stated, as the value of the bleaching powders varies; but rather more acid than of the equal parts of bleaching powder and strong hydrochloric acid may be taken. The acid should be diluted before use. For deodorising water-closets, some crystals of potassium chlorate may be thrown into a wide-mouthed bottle containing dilute hydrochloric acid. Euchlorine comes off gradually, and is both more effective and more agreeable than chlorine. The most marked character of chlorine is its strong affinity for hydrogen, which enables it to break up compounds containing that body, and to set free in a nascent or active state the oxygen combined with hydrogen in water. It is, therefore, one of the most universally applicable and powerful deodorisers in existence. When merely used as a deodoriser, enough euchlorine may be expelled from moist 'chloride of lime' by the carbonic acid of the air for most purposes. In air saturated with moisture 0·3 per cent. chlorine kills all minor organisms that are freely exposed; but any cover, such as a piece of

blotting-paper, prevents the result. Less moisture demands 1 per cent. chlorine.

**5. Permanganate of Potassium.**—This substance is non-poisonous, and is a good deodorant, especially for the emanations from organic bodies. It is, moreover, free from odour, and its aqueous solution shows, by loss of colour, when it is exhausted. It is a very suitable deodorant for the sick-room, as, when dissolved in water and a large surface of the solution exposed to the air, it will absorb gases to some extent. Contagium, being non-gaseous, is not affected, unless in contact with the solution. Permanganate of potassium is a true disinfectant, oxidising and destroying contagia as well as putrid matters; but the quantity required and the price render its use almost impossible, for enough permanganate has to be used to destroy the medium or vehicle bearing contagium as well as the contagium itself. The solution should contain 5 per cent. permanganate after all chemical action has subsided. Condy's fluid is a solution of this substance in water. When the virulent liquid or matters are small in quantity, permanganate solution forms a capital recipient, and may stand by the bedside as a deodorant till required as a disinfectant. Permanganate has no effect in restraining the appearance of bacteria, or preventing the onset of putrefaction.

**6. Acids.**—The mineral acids and glacial acetic acid have all disinfecting power when used in sufficient quantity for a long time, but there are serious difficulties in the way of their use. HCl, 2 per cent. in solution, required ten days to kill anthrax spores.

**7. Nitrous Acid.**—Nitrous acid can be easily disengaged as a gas by putting bits of copper into nitric acid, or pouring nitric acid upon sawdust or starch. It is the best deodorant for the deadhouse, and, without doubt, it is a disinfectant, but it is too dangerous for ordinary use, as it may easily be breathed in quantity sufficient to cause fatal bronchitis.

**8. Chlorinated Lime.**—Bleaching-powder, commonly known as 'chloride of lime,' gives off chlorine easily, and this probably explains its disinfecting power. It is very cheap and manageable, and hence of much importance.

**9. Quicklime.**—Liberius Kitasato and Pfuhl, confirmed by Drs. Richard and Chantemesse, state that fresh excreta of cholera, enteric fever, and dysentery are sterilised, and the bacilli destroyed, so that the stools are disinfected, by 2 per cent. of a 20 per cent. fresh milk of lime in a few hours. The experiments detailed are not conclusive to the writer as to real disinfection in all cases. Quicklime is so cheap and abundant, that it could be used in any quantity. It is soluble in water in the proportion of only 1 in about 700.

10. **Mercuric Chloride.**—*Perchloride of Mercury, Corrosive Sublimate, Malleus Metallorum*, the most powerful chemical disinfectant known, dissolves in 16 parts of cold water, is extremely poisonous, and corrodes metals. With albuminous matters it forms insoluble precipitates, which are prevented by addition of common salt, HCl, or tartaric acid. Precipitates must be prevented where disinfection has to be repeated, as they might be dangerous. The solution with tartaric acid is not stable, and should be freshly made. A spray of 1 in 1,000, or washing with 1 in 5,000, killed the most resistant organisms in ten minutes. Enough should be used to leave 1 in 5,000 after any HgCl<sub>2</sub> is precipitated by sulphur or albumen. After disinfection is finished, copious washing with water removes the remaining mercury salt. For utensils, clothing, &c., 1 in 2,000, and for excreta an equal bulk of 1 in 1,000 solution, are recommended.

In regard to the agents hitherto considered, we have more or less of the sure light of direct experiment upon contagium; but the claims of the following and a legion of other substances asserted to be 'powerful disinfectants, of which it is impossible to speak too highly,' rest entirely upon chemical theories, or the opinions of physicians, or upon their power of coagulating albumen, or of delaying or preventing putrefaction and fermentation, or of deodorising. It will be scarcely necessary to do more than enumerate the best, as follows:—

11. **Metallic Salts.**—Metallic salts include—(a) *Bichromate of potassium*. (b) *Sulphate of copper*. (c) *Chloride of zinc*. A 5 per cent. solution had no effect on anthrax spores in a month, and 1 per cent. failed to kill micrococcus prodigiosus in forty-eight hours. (d) *Chloride of aluminium*. (e) *Ferric chloride*, which, if strong, liberates offensive fumes from animal matters, but is a fair antiseptic and preservative. (f) *Ferrous sulphate*. (g) *The waste chlorides*, from the manufacture of chlorine, contain MnCl<sub>2</sub>, Fe<sub>2</sub>Cl<sub>6</sub>, and free HCl, which cost next to nothing, and might be used for larger masses of filth or drains.

12. **Ozone.**—This body, got by half-immersing a stick of phosphorus in tepid water, or by mixing gradually 3 parts of strong sulphuric acid and 2 parts permanganate of potassium, oxidises organic matter, and so destroys odours. Terebene and cupralum, a preparation containing terebene, are good deodorants, and give rise to ozone.

13. **Charcoal.**—Charcoal condenses gases within its pores, where combustible gases are destroyed by the condensed oxygen. Contagium, unless in water, does not enter the pores, for, being particulate, it is not absorbed from the air as gases are.

**PRACTICAL DISINFECTION.**—In conclusion, a few remarks may be offered as to the modes of carrying out disinfection under

circumstances in which it is commonly required.

1. **Clothing and Bedding.**—In dealing with the ragged and worthless articles of the poor, local authorities will generally find it most satisfactory to both parties to burn them and replace them with new. By such discreet generosity danger is averted, and goodwill created, which helps in getting early information and carrying out measures, and so, by shortening epidemics, saves expense. If not burned, clothing may be steamed as described under *Heat*, or well boiled with soda. Before coming to the washhouse they may be steeped in 5 per cent. carbolic solution, or perchloride of mercury (1 in 2,000), or chlorinated lime (2 oz. to the gallon).

2. **Rooms.**—The essential process for disinfecting rooms is thorough cleansing with soft soap and hot water, which may contain 5 per cent. carbolic acid, but the carbolic solution is not so easily handled. The walls and ceiling should be brushed, and wall-paper removed. Furniture, if iron—a bedstead for example—is to be taken down and washed with carbolic solution, and removed from the room, or smeared with vaseline if to be fumigated. Textile fabrics should be steamed or boiled, or spread out in the room for fumigation; but this is not so effective, and colours are bleached. Should it be considered desirable to fumigate, the room should be steamed for some hours. The chimney, doors, and windows are to be closed, and crevices covered with paper pasted on. Then 15 lbs. chlorinated lime per 1,000 cubic feet, made into cream with water in shallow dishes, has 22 lbs. HCl added from vessels which tilt over as the operator escapes. The door is to be shut until next day, when the windows and doors are all to be opened, and kept open for twenty-four hours. In white-washed rooms the walls should be scraped, and then washed with hot lime in addition to the fumigation.

3. **Drains, Water-closets, &c.**—Proper drains remove sewage so swiftly and completely, that little or no sewage-gas is formed if ventilation is given. For bad drains carbolic acid, chloride of zinc, or waste chlorides from the manufacture of chlorine, are fair palliatives. The excreta from cases of infectious diseases require a very large quantity of disinfectant, which should be applied in a concentrated form before they are thrown into the water-closet or house-pipes. When a reliable amount of disinfectant is in these cases sent down the pipes, it is apt to corrode them unless it has been allowed to expend its energy on the excreta alone in the first place. If small quantities of disinfectants are poured down water-closets, it is better to mix them with the after-flush water which fills traps and basins, so that the little energy available may be devoted to the destruction of any slime adhering, or portions of organic matter retained. Permanganate

of potassium is the most pleasant agent for this purpose, though expensive. When there are no water-closets, the excreta, in cases of cholera and typhoid fever, should be received in a vessel containing half a pint or more of a 1-in-20 solution of commercial hydrochloric or sulphuric acid, and then put, along with some chlorinated lime, into a covered stoneware vessel in the back yard. After a few hours the contents of this vessel may be thrown into the cesspool or upon the midden.

**4. Dead Bodies.**—Dead bodies, if putrid or bearing contagium, should be wrapped in sheets wet with 1-in-20 carbolic solution, or 1-in-1,000 perchloride of mercury; or, if coffined, sawdust saturated with one of these solutions should be packed around them.

It is necessary clearly to keep in view the object desired when selecting disinfectants, deodorants, or antiseptics; whether it be destruction of contagium, merely 'pickling' and preserving, arresting putrefaction and fermentation, or deodorisation. From all that has been said it is evident that the different 'disinfecting' nostrums, applied as their inventors direct, can have little effect upon contagium, but may have more or less power in the other directions indicated.

JAMES A. RUSSELL.

**DISLOCATION OF ORGANS** (*dis*, apart; and *locus*, a place).—See ORGANS, Displacement of.

**DISPLACEMENT OF ORGANS.**  
See ORGANS, Displacement of.

**DISSECTION WOUNDS.** — See POST-MORTEM WOUNDS.

**DISTOMA.**—See ENTOZOA.

**DISTOMA RINGERI** vel **PULMONALE.**—Under this name the writer originally described in the *Medical Times and Gazette* (July 8, 1882) a species of fluke, the mature form of which inhabits the human lung, where it was first found in Formosa, by Dr. B. S. Ringer, in 1879. The ova of the parasite have frequently been found by Professor Baelz, of Tokio, by the writer, and by others, and by them are associated with a peculiar form of recurrent hæmoptysis, to which the term *endemic hæmoptysis* has been applied.

**SYMPTOMS AND PATHOLOGY.**—The symptoms of disease associated with the presence of the distoma Ringeri are a slight cough; the expectoration of a characteristic rusty-brown, viscid mucus; and at times hæmoptysis, slight or copious. The hæmorrhage occurs at irregular intervals during many years. The expectoration of rusty bronchial mucus is persistent, and in this the ova are readily discovered with the microscope. These are pale brown bodies ( $\frac{3}{500}$  in.  $\times$   $\frac{1}{500}$  in.), ovoid, double-outlined, operculated at the broad end, and containing protoplasmic globules having very active molecular movements. If the

sputum is occasionally shaken up in fresh water, there is developed in most of the ova, in the course of six weeks to two months, an active ciliated embryo which in time escapes by forcing back the operculum. It may be concluded from this that drinking-water, or a fresh-water animal acting as intermediary host, is the medium by which the disease passes from one human subject to another. See ENTOZOA.

This disease has hitherto been found only in Japan, Corea, and Formosa, but its distribution is probably much more extended.

The mature parasite measures from 8 to 10 mm. by 5 to 6 mm. It resides in cavities and burrows connected with the smaller bronchi, into which it discharges its ova. As many as twenty of these parasites have been found in one case.

**TREATMENT.**—Inhalations of sulphurous acid, sprays of turpentine, as well as koussou, quassia, and santonin, have been administered without much apparent benefit.

PATRICK MANSON.

**DIURESIS** ( $\delta\acute{\iota}\alpha$ , through; and  $\omicron\upsilon\rho\acute{\epsilon}\omega$ , I pass water).—A free excretion of urine, whether naturally or artificially induced. See DIURETICS.

**DIURETICS** ( $\delta\acute{\iota}\alpha$ , through; and  $\omicron\upsilon\rho\acute{\epsilon}\omega$ , I pass water).

**DEFINITION.**—Remedies which increase the secretion of urine.

**ENUMERATION.**—The following comprise the most important diuretics: Water; Milk; salts of Potassium, Sodium, and Lithium; Calomel; Alcohol, Nitrous Ether, solution of Ethyl Nitrite; Turpentine, Juniper, Copaiba; Cantharides; Digitalis, Strophanthus, Squill; Caffeine, Theobromine, and its compound with Salicylic Acid and Sodium termed 'Diuretin'; Tobacco; Scoparium, and Sparteine. The action of diuretics is often aided by brisk purgation, depletion, counter-irritation over the loins, and sometimes by the use of mercury.

**ACTION.**—The secretion of urine appears to consist partly of filtration of fluid through the glomeruli of the kidney, and partly of secretion by the cells of the urinary tubules. The amount of urine excreted depends on the rapidity with which the blood flows through the glomeruli, and this again depends on the height of the systemic blood-pressure and the dilatation or contraction of the renal arteries. The secretion is increased by anything which raises the blood-pressure throughout the system generally, or in the renal arteries locally. The systemic blood-pressure may be raised by cold to the surface, digitalis, squill, and tobacco. Digitalis, and possibly other drugs, have also a local action on the renal arteries, which are more readily affected by some drugs than other arteries in the body. Nitrous ether and solution of ethyl nitrite probably act as diuretics by dilating the

renal arteries. The exact mode of action of the other diuretics is not determined; but common salt, nitrate of potassium, urates, and urea increase the flow of urine even although the pressure in the vessels of the kidney is very low. It is therefore probable that they stimulate secretion by acting on the nerves or cells in the kidney itself; and probably caffeine acts in a similar manner.

USES.—Diuretics are employed to increase the flow of urine, and thus remove water or excrementitious products like urea from the body. They are used in cases of general dropsy, or of accumulation of fluid in the peritoneum or pleura. In febrile conditions they are given to aid in the elimination of waste matter. They are also employed in order to render the urine more watery, and thus prevent the deposition of solids from it, and the formation of calculi in the kidney or bladder, or to redissolve such concretions when they are already formed. Digitalis and squill are most useful in dropsy dependent on heart-disease; the other remedies are more effective in dropsy dependent on disease of the kidneys or liver. The action of digitalis and squill is greatly assisted by the addition of a little blue pill; and when the kidneys are much congested or pressed upon from without by accumulation of fluid in the abdominal cavity, diuretics sometimes fail to act until the congestion has been relieved by depletion from the loins or the use of a brisk purgative, and the pressure removed by paracentesis. See DROPSY.

T. LAUDER BRUNTON.

**DIZZINESS.**—See VERTIGO.

**DOCHMIUS** (δόχμιος, twisted).—A genus of nematode worms established by Dujardin. See ENTOZOA.

**DORDRECHT**, in Cape Colony.—See AFRICA, SOUTH.

**DOTHIENENTERITIS** (δοθίην, a pustule; and *ἔντερον*, the intestine).—A synonym for a form of enteritis, accompanied by an enlargement of the follicles, which causes them to resemble pustules. See INTESTINE, Diseases of.

**DOUCHE** (Fr.).—DEFINITION.—A jet of water propelled against some part of the body through a *doccia* or pipe. The size of the jet of water, the degree of its impetus, and its temperature can all be regulated. A douche differs from simple affusion in its application being more local, and the force with which it is applied being greater.

APPLICATION AND ACTION.—Douches of cold and of hot water, of vapour, and occasionally of gas, are employed; but those by far the most commonly used, except where there are hot natural waters, are of cold water.

The immediate effect produced by a cold douche on those who are unaccustomed to it is a feeling of shock, spasmodic shortness of

breathing, palpitation of the heart, and sometimes pain in the back of the head. Locally the first effect of a douche is to deaden the sensibility of the part to which it is applied; but if the douche be powerful enough, reaction of the part comes on in about forty seconds. This continues for a time; but if the douche be kept up for three or four minutes, the pulse falls seven or eight beats, the deadening of sensibility returns, and the temperature of the part is greatly lowered; when the douche is withdrawn, reaction again takes place. This alternate sedative and stimulating effect, producing emptiness and turgescence of the vessels, quickens the action of the capillaries of the part, and thus favours the transmutation of tissue. The mechanical effect of the force with which the douche is applied must not be overlooked. If great, it produces the highest amount of stimulation, which may almost amount to inflammation.

Different portions of the body have different degrees of tolerance of the douche. Thus the extremities and the head bear it better than the chest, and the chest somewhat better than the abdomen; and the posterior aspect of the body bears it much better than the anterior. Patients soon get accustomed to the cold. Warm douches produce less shock, and are more easily borne, but they are, comparatively speaking, little employed in private houses. An alternation of hot and cold douches, known somehow by the name of *Scotch*, is a valuable remedy; in it the hot water rapidly restores the irritability of the part deadened by the cold water, and there is a maximum of action and reaction of the part obtained. Under particular circumstances it may be expedient to use a jet of steam, but this, of course, must be done with caution; and a jet of carbonic acid is sometimes propelled against the eye or ear, or the neck of the uterus. So-called *ascending douches* are used for the rectum or the vagina. Douches for the eye and the ear have been used of late years. In a certain sense what the English call *pumping* is a variety of the douche, and the *shower-bath* is in reality merely a multiplication of fine douches. The action of douches is more or less general according to the portion of the body to which they are applied. Thus the application of a douche to the head has the most general action, and that to the spine the next. In either case it is impossible to limit it very strictly, and there is a certain amount of affusion besides the direct douche. A douche, again, applied to one of the extremities may easily be localised; and a douche may be applied only to one part or to several parts of the body in succession.

Douches merely require a pipe with nozzles of various sizes in connexion with a cistern at a certain elevation, or with a pumping machine; they can easily be improvised. Shower-baths can be procured with equal facility. A vapour-douche can be got by

attaching a pipe to a vessel of boiling water. In the case of fine douches used for the eye, the water is propelled with sufficient force by the action of a caoutchouc bag worked by the hand. Carbonic acid is practically little used, and only where there is an abundant natural supply of the gas.

Perhaps 50° F. may be considered the average temperature of a cold douche, and from four minutes to a quarter of an hour its average duration. The course of douching will probably extend at least over a fortnight. As to the actual temperature of the water, the sensation it produces in the patient depends most on his condition. Thus water of 45° F. may feel ice-cold to one who has just quitted a hot bath. In like manner a douche of slightly-heated water may appear quite warm when applied to a part cooled by a cold douche. The temperature of a douche should vary according to the condition of the patient.

USES.—As a general rule we may say that douches are only applicable in cases of chronic disease; that cold douches are most useful in constitutional diseases; and that warm douches, and the alternation of hot and cold, are most suitable in local affections. The use of hot and cold douches alternately has been much and successfully employed of late in France, for the treatment of certain nervous diseases.

The cold douche, when it is employed gradually and with judgment, is found serviceable in chlorotic and hysterical conditions, in hysterical paralysis, and in over-sensibility of the skin, with tendency to catch cold; and of late years it has formed a part of the special treatment of phthisis in elevated places. As cold affusion on the head is very serviceable in infantile convulsions, so the application of a douche of cold water to the head is a calmative and hypnotic in maniacal cases. It is, perhaps, not so much used in this way as formerly, because it has, like the shower-bath, come to be considered a sort of punishment to troublesome lunatics. Still it is a valuable agent. Hydropathic practitioners have found douches useful revulsives in congestion of the liver and of the uterus.

Locally douches have been used, but with moderate benefit only, in some cases of skin-affections and of chronic ulcers. Their principal local application, however, is in cases of old sprains, in chronic rheumatism or gouty thickenings of joints, in lumbago, in some neuralgias, and in paralysis when it is not too recent. The *Scotch* is far the most effective for these purposes, and there seems to be some evidence of its having been efficacious in threatened *tabes dorsalis*—certainly more efficacious than any other remedy. Douches might be used more extensively in private houses; still, as assistance is always required by the patient, public baths have advantages for their application.

JOHN MACPHERSON.

**DRACUNCULUS** (*dracunculus*, a little dragon).—See ENTOZOA.

**DRAINAGE**.—See PUBLIC HEALTH.

**DRAKENSBERG MOUNTAINS**, in Natal.—See AFRICA, SOUTH.

**DRASTICS** (δρᾶω, I act).—DEFINITION. Violent purgatives.

ENUMERATION.—The drastics most frequently employed are:—Hellebore, Podophyllin, Gamboge, Elaterin, Scammony, Jalap, and Croton Oil.

FOR ACTION AND USES OF DRASTICS, see PURGATIVES. T. LAUDER BRUNTON.

**DRIBURG**, in Westphalia.—Strong Chalybeate Waters. See MINERAL WATERS.

**DROITWICH**, in Worcestershire. Common Salt Waters. See MINERAL WATERS.

**DROPSY** (ὕδρωψ: from ὕδωρ, water; and δψ, aspect, appearance).—SYNON.: Fr. *Hydropisie*; Ger. *Wassersucht*.

DEFINITION.—Accumulation of serous fluid in the subcutaneous cellular tissue, or in a serous cavity.

Dropsy is known by various other names, according to the portion of the body affected. When confined to the subcutaneous cellular tissue it is termed *oedema* or *anasarca*; to the peritoneal cavity, *ascites*. The term is often limited to these two forms of the disease; and exudations similar to that of ascites in other cavities are termed *hydropericardium*, *hydrocephalus*, *hydrocele*, *hydrops oculi*, *hydrops articuli*, and *hydrothorax* or *pleural effusion*, according as they are contained in the pericardium, arachnoid, tunica vaginalis, eye, joint, and pleural cavity respectively.

**PATHOLOGY**.—The accumulation of fluid in the tissues, or in a serous cavity, depends upon more fluid exuding from the blood-vessels than can be taken up by the absorbents. This condition may depend on an increase in the rapidity of exudation above the normal, or on a diminution in the absorption, or on both together. Increased exudation may be due to alteration (1) in the calibre of the arteries or capillaries; (2) in the condition of their walls; (3) in the condition of the blood; and (4) possibly also in the condition of the tissues. Diminished absorption is chiefly due to obstruction of the absorbents, although alteration in the tissues may also play a part. Obstruction may be (1) local or (2) general. So long as no obstruction to absorption occurs, it rarely happens that more fluid can exude from the blood-vessels than the absorbents can again take up. Absorption is partly carried on by the veins, and partly by the lymphatics; principally, however, by the veins. When venous obstruction takes place, fluid is apt to accumulate in that part of the body from which the blood ought to return by the obstructed vessel. But it does not always so

accumulate; for it may happen that the lymphatics are able to absorb all the fluid which exudes from the capillaries, and to return it into the general circulation. Thus it has been found by Ranvier that ligature of the vena cava in a dog does not usually produce œdema of the lower extremities, but if one sciatic nerve be divided in such an animal, the corresponding leg at once becomes œdematous. The reason of this is that, so long as the nerve is intact, the lymphatics can absorb all the fluid which exudes from the capillaries; but when the nerve is divided the arteries dilate, more fluid is poured out than the lymphatics can absorb, it accumulates in the tissues, and œdema ensues. This œdema is not due to paralysis of the limb, but to paralysis of the vessels. For if the sympathetic fibres through which the vaso-motor nerves pass to the sciatic nerve are divided before they join the motor fibres of that nerve in the sacral plexus, the power of movement remains unimpaired, but œdema occurs just as if the whole nerve had been divided. If, on the other hand, the motor strands of the sacral plexus are cut before they are joined by the sympathetic fibres, the limb is as completely paralysed as if the sciatic nerve had been cut, but no œdema takes place. Any obstruction to the venous flow will operate in the same way as ligature of a vein, though to a less extent, the effect varying according to the amount of obstruction. Thus regurgitation of blood through the tricuspid valve tends to produce general anasarca, and obstruction to the portal vein by cirrhosis of the liver tends to cause accumulation of fluid in the abdominal cavity.

It has been mentioned how great an influence dilatation of the arteries from vaso-motor paralysis has upon the *production* of œdema in cases where the veins are obstructed. Arterial dilatation may also produce a local œdema, even when no such obstruction is present, as, for instance, in the tissues around an inflamed part. It has been shown, however, by Winniwarter, that the walls of vessels in an inflamed part are more permeable, and allow fluids to pass through them more easily than healthy vessels will do. It is probably in consequence of this that we find that a slight stimulus, such as scratching the skin, which ordinarily produces in a healthy person only slight dilatation of the capillaries, and consequently redness of the part scratched, will produce an effusion from the vessels, and local swelling of the part at the point scratched, in persons suffering from urticaria. The same thing takes place when the skin is scratched in the neighbourhood of a part stung by a wasp.

But this alteration in the vessels is not the only cause of the œdema, which may occur without any obstruction to the circulation. An alteration in the composition of the blood appears to allow it to permeate more easily

into the tissues, and to produce œdema, even when there is no obstruction of the veins. In cases of anæmia we find œdema, occurring at the ankles, although there is no obstruction to the venous circulation other than that caused by the weight of the column of blood itself. In these cases, however, we have dilatation of the vessels, as is shown by the form of the sphygmographic tracing, and an altered composition of the blood is evidenced by the anæmic look of the patient. In albuminuria the altered composition of the blood appears to be the chief factor in the production of œdema, as the pulse in such cases may be hard, evidencing arterial contraction, and not relaxation.

**ETIOLOGY.**—General dropsy affecting the subcutaneous tissue, the peritoneal cavity, and the internal serous cavities and organs generally, is usually the result of albuminuria, and most frequently of that form which depends on acute nephritis, or on fatty degeneration of the kidney. In cirrhotic disease of the kidney the loss of albumen in the urine is much less, and the alteration in the composition of the blood consequently is not so great as in the first-mentioned form. The arterial tension also is greater than usual, instead of being less. In the amyloid form the œdema is generally moderate.

The next most common cause of dropsy is tricuspid regurgitation, obstructing the venous circulation throughout the body. This regurgitation generally depends on dilatation of the right ventricle consequent upon obstruction to the flow of blood through the lungs, either from chronic bronchitis and emphysema, or mitral obstruction and regurgitation. Dropsy from cardiac disease generally appears first in the feet if the patient has been for some time in an upright position, while dropsy with albuminuria is often first remarked by a puffiness of the eyelids. In the former it appears where the greatest obstruction to re-absorption takes place, and in the latter case in those parts where looseness of the cellular tissue most readily allows of exudation.

Local dropsies have, as a rule, local causes. Even the swelling of the feet in anæmic young women, although dependent on a general cause, namely, dilatation of the vessels, and altered composition of the blood, is determined locally by the greater obstruction to the venous circulation which the pressure of the long column of blood in the veins between the feet and the heart presents. In general dropsy also, those parts which are most dependent are apt to become most swollen. It is not, however, always so, as in certain cases the dependent parts have been noticed to be less dropsical than the others. This curious phenomenon seems to be due to some vaso-motor nervous influence on the vessels of the dependent part. The local œdema of a brawny character, often noticed

around inflamed parts, is partially due to swelling of the tissues themselves, and partially to effusion of fluid between them. This effusion, as has already been mentioned, appears to be caused both by the dilatation of the vessels observed in inflamed parts, and by the greater readiness with which fluids pass through them.

**Dropsy in Serous Cavities.**—The serous cavities of the body—the arachnoid, pleura, pericardium, peritoneum, &c.—are now known to be large lymph-sacs, in communication with the general lymphatic system of the body. The fluid which exudes into them from the blood-vessels is, in the peritoneum and pleura, removed, at least in part, by a pumping action in the movements of respiration. The central tendon of the diaphragm contains spaces, the walls of which are alternately drawn apart and pushed together during its ascent and descent. Their separation draws up lymph from the abdominal cavity, and their compression forces it onwards through the lymphatic vessels. The same thing occurs in the costal pleura, during the respiratory expansion and contraction of the chest. The accumulation of fluid in serous cavities may be due, like its accumulation in the cellular tissues, either to diminished absorption or increased exudation. The diminished absorption occurs here in consequence of pressure upon veins, and possibly also from interference with the pumping action just described. Accumulation of fluid in the ventricles of the brain, or in the sub-arachnoid cavity, is chiefly due to compression of the veins of Galen. In the peritoneum it may be due to obstruction of the portal vein by cirrhosis of the liver or by the pressure of tumours; and it may occur to a greater or less extent in all cavities of the body from general obstruction of the venous circulation, by disease of the heart or lungs, in the same way as anasarca. It may also occur in these cavities from alteration in the blood, as in Bright's disease. Active dropsy may occur in a serous cavity from inflammation, and here the exudation of fluid is much more rapid than in passive dropsy, the vessels of the inflamed part being dilated and more pervious than usual.

**TREATMENT.**—The first thing to be considered in the treatment of dropsy is the removal of its cause, if this be at all possible. Where it is due to obstruction of a vein we must hinder, as much as possible, the accumulation of fluid in the vein, by preventing the part from remaining in a dependent position, while at the same time we try to aid the absorption of fluid by the lymphatics by gentle upward friction. Where it is due to obstruction of the circulation in the lungs, we must diminish, as far as possible, all obstruction to the pulmonary circulation by inhalations, emetics, and expectorants, pushed if necessary so far as to cause nausea or even vomiting.

Where the obstruction is due to dilatation or valvular disease of the heart, we must lessen the work the heart has to do, and insist in bad cases on absolute rest in bed or on a couch, in which the patient's position can be altered, but which he should not leave. An absolute milk diet, like that used in typhoid fever, is often of the greatest possible service, if it can be borne. At the same time we should aid the heart to contract more powerfully by the use of cardiac stimulants, such as alcohol and digitalis. Digitalis probably has a threefold action in cardiac dropsy, by strengthening the heart, by contracting the vessels, and by stimulating the kidneys. It strengthens at the same time that it slows the cardiac pulsations, and by making the heart contract more powerfully it keeps up the onward current of the blood more efficiently, and at the same time lessens the dilatation which tends to render the valves incompetent. Besides its effect on the heart, digitalis has also an action on the vessels, causing the arterioles to contract, and probably reducing the dropsy in this way. For the contraction of the arterioles produced by digitalis is exactly the converse of the condition which occurs after division of the vaso-motor nerves, and which, as we have seen, produces dropsy whenever any obstruction of the circulation exists. It is not known at present whether digitalis also causes increased absorption, but it seems highly probable that it does so, because we know that it stimulates the vaso-motor centre, and stimulation of this part of the nervous system has been shown by Goltz to increase greatly the rapidity of absorption from the lymph-sac of the frog. In addition to this action on the heart and vessels generally, digitalis possesses a specific action upon the vessels of the kidney. It is a powerful diuretic, and by thus lessening the amount of water in the blood it will tend to increase the absorption of serous fluid either from the cellular tissue or serous cavities. When digitalis alone does not succeed, the addition of squill and of a small quantity of blue pill frequently increases its efficacy. Digitalis succeeds best in dropsy caused by valvular disease or dilatation of the heart. Sometimes strophanthus, convallaria, or adonis vernalis may succeed when digitalis fails; but in general digitalis is best. It is not so useful in dropsy arising from renal disease, and here other diuretics are preferable. One of the best is spirit of juniper, given either as a mixture or in the form of Hollands gin. Spirit of nitrous ether, nitre, bitartrate of potassium, and broom are useful in all forms of dropsy. Caffeine is sometimes very useful. Theobromine, the active principle of cocoa, seems also to be useful, and a compound of it with salicylic acid and soda has been recommended under the name of 'diuretin.' Copaiba occasionally succeeds where other diuretics fail. It seems to be most successful in dropsy due to

cirrhosis of the liver. Hydragogue cathartics, such as compound jalap powder, elaterin, &c., which cause copious watery secretion from the intestines, supplement the action of diuretics, and relieve or remove dropsy by removing water from the body, as well as altering its nutrition. Calomel seems to have an almost specific diuretic action, and is useful either alone or in combination with other diuretics or purgatives. In some cases of Bright's disease considerable relief has been obtained by the profuse sweating induced by vapour baths, hot-air baths, jaborandi, or pilocarpine. When the dropsy does not yield to other remedies, the fluid must be removed by paracentesis in the case of serous cavities, and by small superficial incisions or punctures, or by the insertion of very fine trocars with drainage-tubes attached, in the case of the limbs.

T. LAUDER BRUNTON.

**DROWNING, Death by.**—The term 'drowning' is employed, in an extended sense, to signify death from submersion in a liquid medium, and in a more restricted sense to signify death in consequence of obstruction of respiration so caused. Now, though death must necessarily ensue from asphyxia, when the air-passages are submerged, apart from any other complication, asphyxia is not always the mode of death in those who were alive at the moment of submersion. For death may result from mechanical injuries, concussion, shock, syncope, or apoplexy in the very act, or at the moment, of falling into the water. Devergie estimates that 12·5 per cent. of deaths occur from one or other of these causes. In the remaining 87·5 per cent. the phenomena of asphyxia pure and simple are present only in 25 per cent., while in 62·5 per cent. these are more or less modified by the causes above-mentioned, to which must be added the benumbing influence of cold.

When death is not sudden from shock, &c., the ultimate result is the same in the swimmer or non-swimmer, if there be no escape or rescue. All efforts to keep above water fail, vain clutchings are made at whatever comes within reach, water is drawn into the lungs and more or less swallowed, all struggles finally cease, and the body sinks.

The indications of such instinctive efforts form the most important evidence of submersion during life. Drowning is not necessarily to be inferred in the case of a dead body removed from the water; for the body may have been thrown in after death from other causes—asphyxia among the rest.

**THE EVIDENCE OF DEATH FROM DROWNING.** This is cumulative, for we can scarcely say that there is any one indication invariably present which can be looked upon as due to drowning and to nothing else. But one or other, or more, of the following appearances are generally found.

*External.*—The face is either pale, or slightly livid, and frequently bloated if the body has lain some hours in the water. Water flows from the mouth when the body is turned face downwards. Foam at the mouth and nostrils is common; and the tongue is swollen and congested, closely applied to the teeth, or even clenched between them. The skin is pale, or marked here and there by livid discolorations, and the muscles of the hair-bulbs are rigidly contracted, causing the appearance of goose-skin, or *cutis anserina*.

The penis, according to Casper, is retracted, but the more common condition is one of semi-erection.

Indications of struggling are sometimes seen in excoriations of the hands, mud and sand under the nails, or even weeds, straws, or other small objects tightly clenched in the hands.

*Internal.*—The trachea, bronchi, and smaller air-tubes are congested and filled with a mucous froth, more or less tinged with blood. The lungs themselves are congested, œdematous, and pit on pressure. Pressure on them causes froth to exude into the smaller bronchial tubes, and on section a sanguinolent froth and water escape. Indications may be seen of sand, mud, or small weeds drawn deep into the air-passages along with the water in which submersion took place. Water is often found in the pleural cavity.

The stomach occasionally contains water. If this has any special character by which it can be identified with that in which submersion occurred, and not likely to have been drunk to quench thirst, it excludes the theory of its having been swallowed before submersion, and is a strong presumption, if it cannot be said to be a certainty, that it was swallowed during the death-agony, and did not find its way into the stomach after death. The same may be said of water in the lungs.

The right side of the heart and venous system in general frequently present the appearances characteristic of asphyxia; and the brain is often congested. See ASPHYXIA.

Without relying on any one sign as conclusive, we may say that a body which exhibits goose-skin, semi-erection of the penis, excoriations, &c., of the hands, froth at the mouth, water in the lungs and stomach, and congestion of the right heart and venous system, certainly died from drowning. In the presence of some and in the absence of other indications, a careful weighing of all the facts is necessary; but in most cases a satisfactory conclusion can be arrived at.

Complete submersion is usually sufficient to cause death within two minutes; but cases have been recorded of resuscitation after a much longer period. Many of these can be attributed to the exaggerated estimation of time by anxious on-lookers; but there are

other well-authenticated instances, which may be explained by the supervention of syncope and temporary cessation of the respiratory process. That which renders resuscitation after submersion less likely than after a corresponding period of mere suffocation is the entry of water into the lungs by aspiration.

**TREATMENT.**—The treatment of the drowned consists in the persistent use of artificial respiration (see **ARTIFICIAL RESPIRATION**; and **RESUSCITATION**) so long as any signs of life remain, together with the application of means to counteract the great abstraction of body-heat which occurs even when the aerial temperature is comparatively high. Before commencing the movements of artificial respiration, the mouth and nostrils should be freed from water and froth, by holding the head somewhat low, face downwards, for a few seconds. Artificial respiration should then be immediately proceeded with. At the same time the wet clothes should be removed, and the body wrapped up in warm clothes obtained from bystanders, pending the arrival of warm blankets, hot bottles or bricks, &c., from the nearest house. Assiduous friction of the extremities should also be kept up. These directions—artificial respiration excepted—naturally presuppose assistance. If this be not at hand, the operator must rely mainly on artificial respiration. In the performance of artificial respiration by Sylvester's method, especial care must be taken against pressing on the stomach, for as it so frequently contains water, this may be forced up the œsophagus, and drawn into the lungs by the next inspiratory movement.

When spontaneous respiratory movements commence, attention should be directed to maintain life by the application of warmth externally, assiduous friction of the limbs upwards, and the administration of brandy and water, wine, or coffee. Lung-complications should be watched for and counteracted.

D. FERRIER.

**DROWSINESS.**—Inclination to sleep. See **SLEEP**, Disorders of.

**DRUG-ERUPTIONS.**—**SYNON.**: *Dermatitis medicamentosa*.

Many medicines produce at times various eruptions or discolorations of the skin, owing to idiosyncrasy, irritability of the gastrointestinal mucous membrane, or defective power of elimination on the part of the patient; or, on the side of the drug, from the largeness of the dose or its long-continued administration.

These toxic lesions may be divided into three classes:—(1) Those inflammatory eruptions which are *special to the drug*, such as the agminated pustular eruptions produced by bromine or iodine compounds (see **BROMISM**; and **IODISM**). (2) Various inflammatory eruptions *not distinctive in*

themselves, their nature being only recognisable from the surrounding circumstances. (3) *Discolorations* of the skin, such as are produced by arsenic and nitrate of silver. See **PIGMENTARY DISEASES**.

The great bulk of medicinal eruptions are either erythematous or urticarial, but the kind of eruption produced by the same drug often varies according to the individual who takes it. With the exception of the special eruption of iodides and bromides, the lesions are probably due to irritation of the gastrointestinal mucous membrane acting reflexly on the vascular nerves of the skin; for in many instances the dose has been too small, and the eruption has followed too quickly on the ingestion of the drug, for the rash to be due to the absorption and circulation of the substance itself. With the exception of the special bromide and iodide eruptions, the diagnosis cannot be made from the aspect of the rash. The following lists do not pretend to be quite complete, but represent all the chief eruptions and the drugs which produce them:—

#### List of Drugs and of the Rashes which they Produce.

*Antipyrine (Phenazone).*—Erythema—diffuse, patchy, papular, or morbilliform; urticaria; miliaria.

*Arsenic.*—Erythema—diffuse and erysipelas-like, papular; acuminated or morbilliform; urticaria, vesicles, pustules, furuncles, herpes zoster, gangrene, pigmentation.

*Belladonna.*—Erythema—diffuse, scarlatiniform, or patchy.

*Benzoate of sodium.*—Erythema—patchy.

*Borax.*—Psoriasis, or a scaly eruption like it.

*Boric acid.*—Erythema, followed by vesicles.

*Bromine compounds.*—Erythema—diffuse, patchy, papular or morbilliform, 'nodosum'-like; urticaria, bullæ, pustules—discrete or agminated, furuncles, ecchyma (indirectly), eczema.

*Calomel injection.*—Erythema, scarlatiniform in one case.

*Cannabis indica.*—Vesicles.

*Chloralamide.*—Erythema.

*Chloral hydrate.*—Erythema—diffuse, papular, and scarlatiniform; urticaria, vesicles, bullæ, pustules, purpura, shedding of nails.

*Chlorate of potassium.*—Erythema, papular; discoloration, bluish or icteric.

*Chloroform.*—Purpura.

*Chrysarobin.*—Erythema—diffuse, with œdema, eczema, once pityriasis rubra.

*Cod-liver oil.*—Vesicular, pustular.

*Copaiba.*—Erythema—diffuse, patchy, or scarlatiniform; military papules, petechiæ, vesicles, bullæ, urticaria.

*Cubebs.*—Erythema—papular, military, or morbilliform.

*Digitalis.*—Erythema—scarlatiniform or papular.

*Iodine and its compounds.*—Erythema—diffuse, papular, morbilliform, or nodosum-like; urticaria, discrete pustules, vesicles, purpura, gangrene.

*Iodoform.*—Erythema—diffuse or scarlatiniform; papules, vesicles, bullæ, eczema, purpura.

*Mercury.*—Erythema—diffuse, like erysipelas.

*Morphine and opium.*—Erythema—scarlatiniform, papular, morbilliform; vesicles and bullæ; never any rash from hypodermic injection.

*Phosphorus.*—Bullæ, purpura, icterus.

*Quinine.*—Erythema—diffuse or patchy, scarlatiniform; papules, acuminate or morbilliform, urticaria, vesicles, bullæ, furuncles.

*Salicylic acid and its compounds.*—Erythema—diffuse; urticaria, vesicles, pustules, purpura.

*Salol.*—Urticaria.

*Santonin.*—Urticaria.

*Strychnine.*—Erythema—scarlatiniform.

*Tar.*—Erythema; locally a form of acne.

*Terebene.*—Erythema—papular.

*Turpentine.*—Erythema—diffuse, papular, acuminate, or morbilliform; vesicles.

#### List of Eruptions and of the Drugs which produce them.

*Diffuse or patchy erythema.*—Antipyrine, arsenic, belladonna, benzoate of sodium, boric acid, bromides, chloralamide, chloral hydrate, chrysarobin, copaiba, iodides, salicylic acid, stramonium, tar.

*Scarlatiniform erythema.*—Belladonna, chloral hydrate, copaiba, iodoform, quinine, strychnine, bromide of nickel.

*Papular or morbilliform erythema.*—Antipyrine, arsenic, bromides, chloral hydrate, cubebs, morphine, quinine, terebene, turpentine.

*Nodosum-like erythema.*—Bromides and iodides.

*Urticaria.*—Antipyrine, arsenic, bromides, copaiba, iodides, morphine, quinine, resin, salicylic acid, salol, santonin.

*Vesicles.*—Cannabis indica, chloral hydrate, cod-liver oil, copaiba, iodides, morphine, quinine, salicylic acid, and turpentine.

*Bullæ.*—Bromides, cannabis indica, chloral hydrate, copaiba, iodides, morphine, phosphoric acid, quinine.

*Pustules.*—Arsenic, bromides (confluent), chloral hydrate, iodides (isolated), salicylic acid.

*Purpura.*—Chloral hydrate, chloroform inhalation, iodides, quinine, salicylic acid.

*Pityriasis rubra (?)*.—Bichromate of potassium.

*Psoriasis (?)*.—Borax, bichromate of potassium.

*Eczema.*—Bicarbonate of potassium, bromides, chrysarobin, iodoform.

*Gangrene.*—Arsenic, ergot, iodides, quinine.

*Persistent desquamation.*—Quinine.

*Abscess.*—Quinine.

*Furuncles.*—Arsenic, bromides, quinine.

*Keratosis palmaris.*—Arsenic.

*Pigmentation.*—Arsenic, nitrate of silver, picric acid.

*Herpes Zoster.*—Arsenic.

H. RADCLIFFE CROCKER.

**DRY.**—SYNON.: Fr. *Sec*; Ger. *Trocken.*—A term applied to certain morbid conditions, to express the entire or comparative absence of fluid exudation or secretion, which is often present in such conditions; for example, Dry Gangrene, Dry Cavity, Dry Catarrh, and Dry Pleurisy. The word is also associated with certain auscultatory signs, which convey the impression of want of moisture; for example, Dry Rhonchus and Dry Crackle. See PHYSICAL EXAMINATION.

**DUCHENNE'S PARALYSIS.**—See PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

**DUCTUS ARTERIOSUS, Patency of.**—See HEART, Malformations of.

**DUMBNESS.**—DEFINITION.—The condition of an individual considered incapable of articulating sounds.

Dumbness may arise from a variety of causes, and its prognosis and treatment vary accordingly.

1. **Dumbness due to deafness.**—The most frequent cause of so-called dumbness is congenital or early acquired complete deafness, or defective power of hearing, so that the patient is unable to acquire in an ordinary way the knowledge of articulate sounds. It is important to bear in mind that complete deafness is not essential to this peculiarity. Dumbness is frequently met with in children and others where the only cause is *defective* power of hearing.

**TREATMENT.**—Whether the outcome of complete or partial deafness, the treatment must be based on the belief that the articulating power is latent, and may be developed by teaching the patient to imitate the process of speaking in others, and by a methodical training in lip-language. It is most important that sign-language should not be cultivated at the same time, and that if a language of signs, whether by the hand or gesture, has been previously taught, it should be thoroughly and at once discarded. With patients who have full intellectual power, and who are trained as indicated above, it will be found that for them dumbness is a misnomer, articulate sounds, although with a defective modulation, being readily acquired. Individuals coming under this category are erroneously called deaf-mutes.

2. **Dumbness from central lesion of the hypoglossal nerve.**—This may arise from cerebral hæmorrhage, tumours, or embolism, and the prognosis is most unfavour-

able. It is usually associated with other paralytic conditions, but is of all the most persistent.

**3. Dumbness from peripheral lesion of the hypoglossal nerve.**—This is much less frequent as a cause of dumbness than central lesions; nevertheless cases are met with where hydatid or other tumours result in dumbness through pressure on the nerve itself.

**4. Dumbness from lead-poisoning.**—Among the paralyzes arising from the slow effects of imbibition of lead is paralysis of the tongue, with consequent loss of articulating power. This defect is usually associated with grave impairment of other parts.

**TREATMENT.**—The treatment of dumbness due to lead-poisoning will be best effected by elimination of the poison by the administration of iodide of potassium, and the judicious employment of galvanism to the nervous system.

**5. Dumbness from congenital defects of the tongue or of the palate.**—Various congenital local lesions are met with giving rise to dumbness.

**TREATMENT.**—Many of these cases are remediable by surgical or surgico-dental help, followed by methodical teaching.

**6. Dumbness from emotional lesions.** Dumbness occasionally arises from great emotional disturbance, such as great anger or sudden fright. Moreover, it is often met with, without such marked cause, in individuals, especially of the female sex, having a highly developed emotional life.

**TREATMENT.**—Cases of this kind are usually successfully treated by faradisation about the muscles of the neck; the patient at the same time being encouraged to call his articulating power into action, and in proportion to his success the faradic current being discontinued. The moral treatment here indicated may be greatly assisted by promoting the general health, and placing the patient under the best possible circumstances as to hygiene and moral discipline.

**7. Dumbness from intellectual disorders.**—This is by far the most common cause of true dumbness. It may arise from idiocy or imbecility of a congenital nature; from that which has been acquired early in life; or from dementia as the outcome of acute or chronic brain-disease in middle or advanced life. Among the congenitally feeble-minded, dumbness is a very frequent phenomenon. This arises from the association therewith of deformed mouths and highly-arched palates; from defective power of co-ordination of the muscles of the tongue; and from an inability to transform ideas into word-signs. In the most profound cases there is such an absence of ideas that language of any kind is not required.

**TREATMENT.**—The treatment of this kind of dumbness is one which requires great tact,

and energy; and the success will be commensurate with these aids.

The physical health of the individual should be carefully attended to, so as to induce, by judicious food, frequent bathing, and warm temperature, the highest amount of nervous energy. The power of co-ordination should be sedulously cultivated by methodical exercises, especially of the hands, leading up to well-devised tongue-gymnastics. He should then be taught monosyllabic sounds, by being shown the object represented by the sound, while he imitates the sound when watching the teacher's lips. Having thus acquired the power of producing the word-sign by imitation purely, he is next taught to repeat it from memory when shown the object only. After nouns have thus been taught, the names of qualities and adverbial expressions should be added, and in this manner articulate speech built up.

Where the feeble-mindedness has been acquired after birth, the dumbness resulting therefrom should be treated in a somewhat similar manner to that having a congenital origin, but the prognosis is not so good.

Still more unfavourable is the forecast of dumbness when the result of dementia.

J. LANGDON DOWN.

**DUODENUM, Diseases of.**—These may be considered under the heads of—(1) *Functional disorder*; and (2) *Organic diseases*.

**1. Functional disorder.**—Disorder of the duodenum is said to produce a form of dyspepsia, characterised by pain in the epigastrium and right hypochondrium two or three hours after meals, vomiting, and the distant effects of ordinary dyspepsia—vertigo, headache, drowsiness, burning sensation in the soles of the feet and palms of the hands, &c. Imperfect chymification, which attends ordinary dyspepsia, may induce these symptoms by generating products which irritate the duodenum.

**TREATMENT.**—The treatment is mainly that of disordered digestion. The bowels and the diet are to be carefully regulated; and such remedies as alkalis, bismuth, oxide of manganese, prussic acid or hydrochloric acid exhibited, according to the special requirements of each case.

**2. Organic diseases.**—These are (a) *inflammation*; (b) *ulceration*; and (c) *new-growths*.

(a) **Inflammation.**—**SYNON.**: Duodenitis; **Fr.** *Duodénite*.—*Acute* inflammation of the duodenum is usually of a mild catarrhal character. It either forms part of an enteritis, or is an extension downwards of a similar affection of the stomach. The ordinary exciting causes are exposure to cold, and irritating ingesta or acrid bile. Usually the symptoms of slight gastric catarrh—a loaded tongue, anorexia, nausea, vomiting of tenacious mucus—are followed by jaundice. When the

duodenum is invaded there is said to be fulness and tenderness of the right hypochondrium. As a rule, however, new symptoms are not set up unless the bile-ducts become obstructed by mucus from extension of the catarrh into them, when jaundice supervenes without pain. The attack usually lasts a week or a fortnight, and, with suitable hygienic and dietetic treatment, it passes safely off. It is, however, sometimes followed by jaundice unusually prolonged, and, even when the bile-ducts are free, by prostration and wasting out of proportion to the mildness of the disease in itself. While catarrhal duodenitis does not afford characteristic symptoms, its presence is, however, usually suggested by painless jaundice following exposure to cold, catarrh of the stomach, and enteritis.

**TREATMENT.**—This consists in rest in bed, warmth, liquid diet, counter-irritation by blisters, mustard or hot poultices, and effervescent salines.

*Chronic inflammation*, resulting in thickening of the mucous and submucous tissue, and even adhesion of the duodenum to adjacent organs, is usually associated with chronic ulceration, or with cancer of the duodenum, pancreas, liver, or other structures. Contraction of the duodenum produces symptoms of obstruction similar to those arising from stricture of the pylorus.

(b) **Ulceration.**—*Perforating ulcer*, similar to that of the stomach, is said to be more frequent in men than in women (about 10 to 1), and rarely to occur during childhood; while it sometimes follows severe and extensive burns and scalds.<sup>1</sup> Fatal ulceration has not infrequently been reported in previously healthy subjects. It may then possibly arise from an accidental abrasion of the duodenal mucous membrane (by some indigestible substance), becoming subsequently subjected to the deleterious action of the gastric juice.<sup>2</sup> The ulcer, usually single and found in the upper horizontal portion, when recent has clean-cut edges free from swelling. The wall of the duodenum may be perforated, either without previous adhesion, being followed by

the signs of general peritonitis; or with adhesion to adjacent parts, such as the liver, the gall-bladder, the pancreas, the colon, the hepatic artery, or the posterior wall of the abdomen—into which ulceration extends to a variable extent. Cicatrization may take place with recovery, or it may induce stricture of the duodenum or obliteration of the bile-duct. As a rule the symptoms greatly resemble those of perforating ulcer of the stomach; more frequently, however, the disease is latent, and induces very obscure dyspeptic symptoms prior to fatal perforation. Jaundice is not more common than in the similar affection of the stomach. In burns and scalds perforation seldom occurs before the tenth day. The treatment should be the same as that laid down for gastric ulcer. See STOMACH, Diseases of.

The duodenum may be ulcerated by the action of a gall-stone passing into it directly from the gall-bladder.

(c) **New-Growths.**—The most important of these is scirrhus cancer, which may involve the walls of the duodenum, usually by extension. It tends to produce obstruction, or it may set up chronic duodenitis, or block up the bile-duct and thus give rise to jaundice.

GEORGE OLIVER.

**DURA MATER, Diseases of.**—See MENINGES, Diseases of.

**DURATION OF DISEASE.**—See DISEASE, Duration of.

**DYNAMOMETER** (*δύναμις*, power; and *μέτρον*, a measure).

**DESCRIPTION.**—The dynamometer is an instrument originally invented by M. Duchenne, of Boulogne, for measuring and accurately recording the strength of the hand-grasp, and also for measuring the traction power capable of being exerted by other groups of muscles. The result is shown by an index, which traverses a semicircular dial bearing a scale graduated so as to enable the observer to record the number of kilogrammes which the applied pressure or traction represents. In practice this instrument has been principally employed for estimating the absolute or comparative force of the hand-grasp; and, in view of this restriction, Duchenne's instrument is needlessly complex. It has, moreover, the absolute disadvantage of being a little too broad, and of requiring too much strength on the part of the patient to move the index over the lower figures of the dial. A patient with a small amount of motor power, especially if the hand is small, is often unable to set the index of this dynamometer in motion. A cheaper, simpler, and narrower instrument has therefore been devised by English makers, the index of which can be moved by the application of a much smaller amount of power. This consists of a simple elliptical ring of steel, to

<sup>1</sup> In explanation of the connexion between severe burns and duodenitis ending in perforative ulceration, Dr. William Hunter infers from his experiments with the subcutaneous injection of toluylendiamin (which he found to set up inflammation of the duodenum) that there may be derived from the burnt tissues and excreted through the bile irritant substances which excite ulcerative inflammation. Some recent observers (Fayrer, Bowly, and Perry), however, have failed to discover *post-mortem* evidence of the alleged causal relation between burns and duodenal ulceration; while others (Wilks and Macarthy) have shown the rarity of it. Doubtless the association is much less frequent than has hitherto been held; for example, Raymond Johnson found in the records of University College Hospital only two cases of ulceration out of ninety-three cases of burns (*Trans. Path. Soc. Lond.*, 1890).

<sup>2</sup> Myers, *Trans. Path. Soc. Lond.*, 1890.

the inner and anterior face of which is attached a brass semicircular dial graduated with two rows of figures representing pounds instead of kilogrammes. The compression of the steel ring, by lessening its shorter diameter, moves a metal bar projecting from and sliding in a groove behind the dial, and this by rack-work communicates its movement to the index.

USES.—The dynamometer is a useful instrument, inasmuch as it enables us accurately to ascertain the relative compressing powers of the two hands in cases of incipient or actually developed hemiplegia, and also to learn in a positive and definite manner, from time to time, the amount of improvement or the reverse which may have taken place. Since the power of the muscles of the forearm and hand, like that of other groups of muscles, varies a good deal with the general state of health of the patient, the dynamometer is also capable of yielding valuable information concerning the strength of the patient, even where we have to do with general debility from any cause rather than with a case of paralysis. Any instrument which, in the place of fleeting and more or less vague impressions made upon the mind of the practitioner at the time, enables him to make a more accurate record in figures in his note-book, is a clear gain to practical medicine—more especially when its use involves no appreciable loss of time.

H. CHARLTON BASTIAN.

**DYSÆSTHESIA** (*δυσ-*, with difficulty; and *αἰσθάνομαι*, I feel).—A term applied to impairment of any of the senses, but especially to that of touch. See SENSATION, Disorders of.

**DYSCRASIA** (*δυσ-*, with difficulty, or badly; and *κράσις*, a mixture).—A morbid condition of blood.

This term signifies more than a disposition to disease; it implies the presence of some general disease exerting its pernicious effects upon the blood. Hectic fever, septicæmia, and metastatic inflammations are diseased conditions referable to dyscrasia. A person sickening for a fever is the subject of a specific dyscrasia. See BLOOD-DISEASE.

R. DOUGLAS POWELL.

**DYSENTERY** (*δυσ-*, with difficulty; and *έντερον*, an intestine).—SYNON.: Fr. *Dysenterie*; Ger. *Dysenterie*.

DEFINITION.—A specific febrile disease, accompanied by considerable nervous prostration, and characterised by inflammation of the solitary and tubular glands of the large intestine; sometimes ending in resolution, but frequently terminating in ulceration, occasionally in more or less sloughing or gangrene; always, in the acute stage, accompanied by tormina and tenesmus, the latter being most marked when the disease is situ-

ated in the rectum or lower end of the sigmoid flexure; stools at first more or less fæculent, later on yielding dysenteric products without much if any fæculence, such as blood, mucus, slime, and gelatinoid exudation, or—as in the sloughing or gangrenous forms—like the washings of meat, and possessing a putrid or gangrenous odour, and so-called epithelial, ash-coloured, black, gangrenous, pus-infiltrated or tubular sloughs, chiefly consisting of tough, imperfectly organised exudation.

ÆTIOLOGY AND PATHOLOGY.—In almost all, if not in all, situations where malarious fevers abound, as in the vicinity of the swamps and sluggish rivers of tropical and sub-tropical countries, dysentery prevails in proportion to the intensity and frequency of these fevers. Where, on the other hand, intermittents and remittents have been extinguished by improved drainage and the conversion of marsh into cultivated land, it becomes equally unknown. In or near the tropics, Great Britain, Canada, the United States, and in many other parts of the world, its diminution has proceeded *pari passu* with the decrement of malarious fevers. There would, therefore, seem to be some intimate connexion between the causation of dysentery and intermittent and remittent fevers. Not that the immediate causes of malarial fevers and dysentery are identical: all that is meant is that under certain conditions they concur.

Paroxysmal fevers interfere materially with the nutrition and functions of the digestive organs, and with the proper nourishment and constitution of the blood. Violent congestion of the abdominal viscera is one of the special conditions of the different forms of ague. Both the liver and spleen are liable to temporary and repeated engorgement, and so in fact are all the organs which minister to gastric and intestinal digestion. Even where malarious poisoning may never have resulted in any of the various forms of periodic fever, it may impair the power of the organic nerve-centres and the muscular tone of the blood-vessels, thus disturbing the balance of the portal circulation, and leading to more or less permanent repletion or congestion. As the congestion is most embarrassing during digestion, interfering with the appetite, and the capacity for digesting and assimilating food, its repeated and prolonged existence must deteriorate the quality and modify the quantity of such important secretions as the gastric juice, the bile, the pancreatic juice, and those furnished by the follicles of Lieberkühn, and by Brunner's and the solitary glands. Crude alimentary principles are thus assimilated from the intestinal tract. The liver is especially liable to functional derangement from the stagnation and slowing of the portal circulation, and thus it happens that in dysentery hepatic impairment is almost an invariable accompaniment. Such being some of the abnormal conditions produced by the operation

of malaria in its active or latent form upon the chylipoietic viscera, it is not surprising that, under the prolonged strain, certain portions of the alimentary mucous membrane should break down. Why the solitary glands of the large intestine should be the special seat of dysentery, whilst the corresponding glands in the small intestine should, with few exceptions, escape, it is, in the present state of our knowledge, impossible to say. The most that can be hazarded is, that the elementary structures of these glands take on morbid action through the operation upon them of a *materies morbi* derived directly from their blood-supply. That this poison exists in the blood may be inferred from the fact that constitutional disturbance invariably precedes and accompanies the earlier stages of acute dysentery.

Unwholesome drinking-water is a fertile proximate and exciting cause of dysenteric disease. Bad and unwholesome food of whatever description, by providing aliment incapable of being perfectly digested, may act in producing the disease. In like manner impure air may, by preventing the proper aëration and depuration of the blood, and by promoting the retention in it of inassimilable material, light up dysenteric inflammation of the solitary glands. The transit of acrid and vitiated bile and other secretions poured into the digestive canal, often operates as an exciting cause, and frequently determines a morbid action which might be otherwise righted by resolution, to advance to ulceration, sloughing, or gangrene. It is thus that many a simple but neglected case, scarcely at first distinguishable from diarrhœa—unless indeed every evacuation is most carefully washed and scrutinised—ends in destruction of large masses of the mucous membrane, and death, from the conjoint effect of exhaustion and shock from the separation of the sloughs. Indigestible articles of diet, which cannot be reduced by the juices of the digestive tract to a condition admitting of ready absorption, may act as local irritants and exciting causes. Not only is this so in the earliest visible stage or that of active congestion, but it becomes much more susceptible of demonstration in the exudative and ulcerative phases of the disease, by the repeated investigation of the subjective and objective indications. Thus the aggravation of the tormina and tenesmus in adults and children is traceable to indigestion in diet, or to the passage of undigested morsels of food, recognisable in the stools. Sudden vicissitudes of temperature from a high to a low range, or exposure to damp and cold combined, especially when the vital powers are physiologically depressed, by checking the excretory action of the skin and diminishing the cutaneous circulation, augment the portal congestion and excite dysenteric disease. This is probably the reason why, in a

large proportion of cases, the onset of the disease is ushered in towards midnight or the early morning. The operation of epidemic influences in the causation of dysentery, signifies only that it is most prevalent at those seasons when malarious fevers are most abundant. As there is an intimate connexion between the existence of malaria and the prevalence of dysentery, it is not difficult to understand why, both as regards type and seasonal frequency, dysentery should bear a striking relation to the severity and seasonal prevalence of malarious fevers.

*Is dysentery a contagious or communicable disease?*—Whilst many of the older physicians held that dysentery might be spread by contagion from person to person, it may be affirmed that the experience of most modern practitioners is altogether opposed to this view. There is no clear and unimpeachable evidence to demonstrate that it is propagated in the same way as typhus or small-pox. Dysentery may possibly be communicable, like typhoid fever, through air, water, or food—liquid or solid—charged with material derived from the undisinfected and putrefying products of the disorder. Be this, however, as it may, the complete disinfection or destruction of the alvine evacuations should always be regarded as a sanitary measure of supreme importance.<sup>1</sup>

**ANATOMICAL CHARACTERS AND PATHOLOGY.**—The dysenteric process generally consists of a specific inflammation of the solitary glands (Parkes, Baly, and others). The *first* visible change is congestion, the vessels surrounding and penetrating the capsules being turgid and engorged with blood. The *second* change is augmentation of their contents from the accumulation of albuminous exudation, and enlargement 'from the size of a millet-seed

<sup>1</sup> It is more than probable that there are several varieties of dysentery, each of which is attributable to its own specific cause. Different micro-organisms have from time to time been described in this connexion; but, as there seems to be no consensus of opinion on the subject among observers, it is premature to conclude that dysentery is brought about by the direct action on the glands of the colon of any known micro-organism. The only parasite which has established anything like a claim to be considered specific to dysentery is the *Amœba coli*, discovered by Lösch in 1875. This amœba has of late years been frequently found in dysenteric discharges in places so widely separated as Russia, Central Europe, Egypt, the United States of America, and India. Moreover, it has been found, on more than one occasion, in the pus and walls of liver-abscess complicating dysentery. This association is not without significance; but, as the amœba is certainly not present in every case of dysentery or dysenteric abscess of the liver, we are as yet justified in regarding it mainly as an epiphenomenon grafted on certain cases of colitis and liver-abscess, and not as the specific cause of these diseases. The *Amœba coli*, which when alive displays the characteristic amœboid movements, varies in size from 0.02 to 0.035 mm., is coarsely granular, nucleated, and possesses one or more non-contractile vacuoles. See ENTOMOZOA.—EDITOR.

to a small shot' (Baly). The *third* change, provided the inflammation advances, is rupture of some of the capillaries in the interior of these little vascular glands, extravasation of blood, with the area of the ordinary dark point on the free aspect increased. The *fourth* stage is now marked by atrophy and molecular disintegration of the free aspect of the capsular wall, and escape of its morbid gelatinoid blood-tinged contents into the canal of the intestine. This is the rule; but, in very exceptional cases, the capsule may burst through the attached portion, lighting up inflammation in the neighbouring connective tissue and muscular coat. In a large number of instances the morbid process may stop short, under proper treatment, at any of the first three stages, and repair is then effected by resolution. In many cases the morbid action is cut short after the completion of the fourth stage, without further extension of the disease. The adjoining follicles of Lieberkühn do not, in these cases, necessarily participate, to any great extent, in the diseased process. Under these conditions, when the whole of the exudation has been expelled, the glands regain their tone and functions, and recovery—rapid and complete—ensues. It is not often possible to illustrate these conditions in the *post-mortem* room; because, when death supervenes from dysentery alone, the ravages committed upon every structure of the mucous membrane are so extensive as to destroy the earlier physical phases of the disease. In some cases, however, which have died from intercurrent affections, the writer has been able to demonstrate the earliest stages successfully to his students, at a period prior to the implication of Lieberkühn's follicles, of which the mucous membrane is in great part composed, and to exhibit to them the gelatinoid exudation, termed by others 'gelatinous mucus,' free from or tinged with blood taken from enlarged and diseased solitary glands (*Indian Annals of Medical Science*, p. 190, No. xxiii., 1868).

When, owing to neglect, to constitutional defect in spite of the most careful therapeutic and hygienic management, or to intensity or quantity of the specific poison, the disease is not cured by resolution, the disintegrating or ulcerative process is developed. The whole of the solitary glands engaged perish. The ulceration involves the neighbouring tubular glands, leading to ulcers varying from the size of a mustard-seed to that of a florin or more, in depth generally extending to the submucous connective tissue, and not infrequently laying bare the circular lamina of the muscular coat, sometimes involving the longitudinal layer and perforating it as well as the peritoneal coat, thus admitting of the extravasation of the contents of the bowel into the peritoneal cavity, and lighting up peritonitis, which, if general, is invariably

fatal, but which, if local and confined to the close vicinity of the perforating ulcer, is not necessarily so.

The ulcers vary in appearance, size, and shape. They may be mere abrasions without much loss of structure; *minute*, though penetrating rather deeply into the submucous connective tissue; *irregular*, *serpentine*, or *rodent*, with here and there portions of the surrounding mucous membrane undermined and patulous; *transverse*, embracing partially, or completely, the entire circular outline of the mucous membrane; *circular* or *oval*, with regular and even margins; or *tubercular*, involving the whole substance of the mucous membrane, looking as if they had been punched out of it. These ulcers, as generally observed in the *post-mortem* room, are free from sloughs, and present a pale ashy appearance. Sometimes they are of a vermilion or purple colour, from active or passive congestion. They are often covered with flakes of tenacious lymph or exudation, and this may sometimes be seen spread over the neighbouring mucous membrane. The floors of these ulcers are usually formed by inflamed and thickened submucous areolar tissue; but sometimes this has all been destroyed, and then they are constituted of the muscular coat, thickened and infiltrated by inflammatory products; and when the muscular structure has itself yielded to the ulcerative process, they are made up of congested and swollen peritoneum, which, as already stated, occasionally becomes perforated.

When the ulceration proceeds solely by molecular disintegration massive sloughs are not observed. But when, as not uncommonly happens in asthenic, malaria-stricken, tubercular and worn-out constitutions, tissue-death occurs *en masse*, at an early period of the attack, sloughing of the mucous membrane is to be seen, together with portions of the muscular coats, or gangrene. These sloughs, or gangrenous portions of tissue, may be limited in extent. Some or all of them may be successfully detached during life, and can be identified as they are examined from time to time in the stools. In the *post-mortem* room they may be found partly detached and lying loose, mixed with the fluid contents of the bowel, or attached more or less firmly; sometimes compact, nodular, echymosed, grey or olive-coloured, green or yellow and pus-infiltrated, black, flaky, shreddy, shaggy, flocculent like pieces of teased cotton-wool, or ragged and stringy. In the truly gangrenous dysentery, the mucous and muscular coats are enormously thickened, and large portions are found gangrenous, varying in colour from a pale olive to purple or black. These appearances and conditions may be restricted to the cæcum and ascending colon, or to the sigmoid flexure, but sometimes they are co-extensive with the internal structure of the large intestine from the ileo-colic valve to the anus.

When the ileo-colic valve becomes destroyed, invagination of the lower end of the ileum into the cæcum sometimes happens, causing intestinal obstruction. Occasionally many inches of the gangrenous mucous membrane, with or without the muscular coat, are found either hanging loose in the lower part of the gut, or in process of being detruded from the anus.

In ordinary acute dysentery, advancing to ulceration or sloughing, repair is doubtless accomplished, as a general rule, by granulation and cicatrization. This process can be readily observed in dysenteric lesions of the mucous membrane of the lower end of the rectum, and in healing of the surgical ulcer within the verge of the anus. The reason why repair is frequently accomplished so slowly is because, owing to the irritation caused by the exalted vermicular contraction of the gut and the passage of flatus, fæces, and other products, it is impossible to command the physiological rest necessary for speedy and substantial granulation and cicatrization. Unless the destruction of tissue is very great, the contraction due to cicatrization does not occasion much future inconvenience. But if it embraces a large portion or the whole circumference of the mucous membrane, the subsequent contraction may produce dangerous narrowing of the calibre of the gut, or stricture of the sigmoid flexure or rectum. The thickening and contraction, especially in the attenuated victims of chronic dysentery, can be identified by physical examination. These constrictions are frequently the mechanical cause of constipation and fecal accumulations.

There is no valid reason for believing that the lost tissue is ever actually reproduced in true dysenteric ulceration. When the ulcers have been small, the contraction following repair is sufficient to bring the follicles of Lieberkühn on all sides into close juxtaposition. And this it is which has given rise to the impression among some pathologists, that the lost tissues have been renewed by a process of development and growth. But whenever the ulcers have been too large to admit of obliteration, microscopical examination shows that they have been bridged over by cicatricial tissue, devoid of solitary and tubular glands, and sparingly supplied with blood-vessels and absorbents.

In addition to the above anatomical characters, the mesenteric glands are generally found to be enlarged; and as an accompaniment or sequel, organic disease of the liver, or abscess, is not infrequently discovered to complicate the disease.

**SYMPTOMS.**—Every attack of acute dysentery is preceded by disordered digestion and constitutional disturbance, indicated by loss or capriciousness of appetite and furred tongue, constipation alone or alternated with looseness, dryness of skin, occasional chilliness

and general malaise, with slight rise of the evening temperature. These signs may be viewed as contemporaneous with the progress of the morbid action going on in the solitary glands. As the disease advances, there is more marked chilliness, succeeded by distinct feverishness. If the bowels have been confined, they now act spontaneously—expelling, at one or more acts of defæcation, almost the whole of the contents of the large intestine. If they have been loose, with or without aperient medicines, the fæculence is not so great in quantity. But in either case, beyond a little mucus, there is not as yet any discoverable dysenteric product in the stools. Prior to this conservative evacuation of the bowels, the febrile excitement sometimes runs high; there is thirst, bad taste in the mouth, flatulency, a variable amount of nervous and muscular debility, griping, an accelerated and irritable pulse, restlessness, disturbed sleep or actual insomnia. During, and immediately after, each evacuation there is tenesmus or painful straining—most intense in those cases where the disease is situated in the descending colon, sigmoid flexure, and rectum. The stools are offensive, but there is nothing at this stage pathognomonic in their odour. If, as frequently happens in private practice, the patient comes under treatment at this period, a small dose of castor oil guarded by laudanum, or a full dose of ipecacuanha, with absolute rest in bed and bland liquid nourishment, is sufficient, in a certain proportion of cases, to put a stop to the morbid action, and to promote cure by resolution in from twenty-four to forty-eight hours.

When, however, the disease persists, the symptoms continue in an aggravated form. The tormina and tenesmus become intensified; the desire to go to stool is more frequent, and to remain on the stool or bed-pan more irresistible and enduring, especially if the disease be concentrated in the sigmoid flexure or rectum. In rectal dysentery, there is dysuria, frequent micturition, and sometimes retention, from spasm due to reflex action, necessitating catheterism. The consumption of solid food—even of the most digestible kind—provokes and aggravates the tormina. The griping and tenesmus are now so intensified in degree, and increased in frequency, that each recurrence of them produces much depression and exhaustion, and a pinched and anxious expression of the countenance, with augmented frequency and weakness of the pulse. There is abdominal tenderness. During the acme of the tormina, the patient experiences difficulty in localising this tenderness. He will then declare that he feels agonising pain over the greater part of the abdomen, with or without the application of pressure. But in the absence of the tormina, careful palpation will enable the practitioner to localise it in those portions of the intestine

above the rectum which are affected by dysenteric inflammation. At this stage the tumefaction of the walls of the gut is seldom great enough to be distinguished through the abdominal parietes. The scanty stools are now characteristic, consisting of mucoid exudation tinged with blood, or bloody mucus or slime from the inflamed tubular glands, with isolated portions of gelatinoid exudation, more or less coloured with blood from inflamed and ruptured solitary glands, and with little or no feculence. These conditions are contemporaneous with the rupture of the affected solitary glands and a highly inflamed state of the adjacent follicles of Lieberkühn, as well as of the subjacent and intervening connective tissue. The muscular tissue, though not yet necessarily inflamed, is nevertheless hyperæsthetic. It is this hyperæsthesia which has more to do with the production of the agonising tormina than the diseased glandular organs implanted in and forming a constituent portion of the mucous membrane. These are the symptoms presented in a goodly number of cases met with in private practice, and in a smaller proportion admitted into hospital. The disease is said to have lasted from two to six or eight days. In uncomplicated cases, occurring in tolerably good constitutions, they generally yield, without extension of the mischief, to rest, bland liquid food, and full doses of ipecacuanha.

In cases which have been neglected or aggravated by indiscretions in diet and drink, or by diathetic or other defect of constitution, the dysenteric process passes on to ulceration. If the patient have not been brought under proper therapeutic and hygienic management, the tormina, tenesmus, local tenderness, and hardening of the superimposed abdominal muscles are augmented. The calls to stool are more frequent and painful. The urine is scanty and high-coloured, and is surcharged with lithates and biliary pigment. The stools mainly consist of the foreign products already described, but in greater quantity; are now possessed of a peculiar sickly smell; and yield portions of exudation in masses of greater or smaller size, simulating sloughs, but which, on microscopical examination, are seldom found to afford positive evidence of dead tissue-elements.

Unless the disease bend to treatment, it may terminate in sloughing. This may be either restricted or extensive. Death of portions of the mucous membrane is always accompanied by vital depression or well-defined muscular and nervous prostration, cardiac enervation, and an accelerated and feeble pulse. In some cases tissue-death *en masse* happens at an earlier period, even before the rupture of the solitary glands; and the sloughs can be discovered in the stools on from the eighth to the thirteenth day. The acme of vital prostration is manifested during the detachment of the sloughs, when the exhaustion is often

much increased by hæmorrhage. Improvement in the strength, volume, and slowing of the pulse, and in the expression of the countenance, the comparative relief from tormina and tenesmus, cleaning of the tongue, and the substitution of feculence for dysenteric products in the stools, signify, in these cases, the probable cessation or turning-point of the disease. The converse implies that the sloughing is extending, and a succession of sloughs of various kinds, with or without muscular structure, continues to be passed, which, in the process of separation, is accompanied by much bleeding, especially in patients poisoned by malaria or afflicted with the scorbutic or hæmorrhagic diathesis. The abdominal tenderness and hardening of the parietal muscles are marked during the separation of the sloughs. The affected portions of the colon can be felt to be swollen, doughy, and 'puddingy' (Chevers). The skin, in unfavourable cases, becomes clammy, features and eyes shrunken, body emaciated, appetite in abeyance, thirst great and distressing, tongue dry and brown, pulse feeble and running; the stools extremely offensive, bloody, slimy or watery, with varieties of sloughs and exudation. The sensibility becomes so blunted that the stools are passed without much pain—often involuntarily; and the patient eventually dies from sheer exhaustion from the extension of the sloughing, or the end may be accelerated by perforation of the bowel in one or more places, leading to extravasation of some of the contents of the intestine into the peritoneum, and general peritonitis.

Sometimes, from the commencement of an attack, or during the course of acute dysentery, rapid sloughing forms the chief pathological condition, passing into gangrene of large patches of mucous membrane, attended by increasing prostration and soon merging into collapse. As this extends until, in many instances, it involves almost the whole of the mucous membrane, submucous tissue, and muscular structure, it is characterised by intensified collapse. The tormina and the tenesmus, at first excruciating, suddenly disappear, to the delusive relief of the sufferer. Until the gangrene has spread to a great extent, the stools contain a large quantity of slime, blood, peculiar-looking exudation, and much gelatinoid exudation; but as the living mucous membrane becomes diminished, these products also decrease, and, in their room, we notice a watery product of a dark purple or black colour, resembling the washings of meat, giving off an incomparably offensive and gangrenous odour. To the naked eye, and on washing, these stools seem to be absolutely devoid of feculence. They yield a granular sediment of a black colour—gangrenous *débris* blackened by the combination of sulphur with the iron of disorganised cruorin. The abdomen, at first doughy, becomes tympanitic and free from pain on pressure,

and the surface cold and clammy; the countenance resembles the haggard and sunken appearance presented during the algid stage of cholera; the tongue is dry, brown, and fissured; the pulse is thready and rapid; food and medicines are rejected; the dejecta are passed involuntarily; perception is blunted; delirium supervenes; and the patient at last succumbs, worn out and exhausted, on from the sixth to the thirteenth day, according to the range and severity of the disease, and the constitutional power which he possesses.

**Chronic Dysentery.**—This is sometimes the result of acute dysentery, in spite of the most appropriate management, in persons poisoned by malaria and weakened by fatty or waxy degeneration of the spleen or liver, or both, and in strumous or scorbutic constitutions. The dyscrasia may be so pronounced that the material exuded is incapable of healing up the ulcers by granulation and cicatrization. The ulcers are repeatedly disturbed by peristalsis and the passage of flatus, fæces, undigested morsels of solid food, and the acrid unutilised secretions of the liver, stomach, and pancreas. Thus they are liable to become irritable from renewed congestion and inflammation. The muscular coat participates in the excitement, and becomes infiltrated with exudation, which eventually becomes organised and leads to thickening. The floors and sides of the ulcers are constituted of unhealthy structure. The difficulty thus experienced in effecting repair is augmented. In other cases the intestine becomes atrophied, attenuated, and transparent. The stools are made up of serous exudation, slime, blood, and sometimes of puriform material, with fæculence generally unformed. Almost every stool will be found on washing to contain dysenteric products. Very frequently, especially in cases which have been carefully dieted for a time, neither blood nor mucus can be detected in the stools, which appear to be of a purely diarrhoeic nature. The appetite is uncertain; the tongue often clean, shining, and devoid of epithelium; the pulse weak and irritable; and the abdominal tenderness easily localised. Thickening can often be detected. Tormina are always present; and unless the disease be confined to the cæcum or ascending and transverse colon, there is tenesmus. Multiple abscesses of the liver frequently supervene and carry off the patient; or after months or years of suffering he may perish from inanition and exhaustion, or from intercurrent disease.

**COMPLICATIONS.**—Acute dysentery is frequently complicated by the various forms of malarious fever, typhoid fever, the tubercular or the hæmorrhagic diathesis, purpura, scurvy, hepatic and splenic enlargement, malarious cachexia, or abscess of the liver; and in children by dentition. In every case of dysentery the strictest attention should be paid to existing complications. In patients

inhabiting marshy districts, and those following a seafaring life, the gums should be carefully scrutinised, and the cutaneous surface examined, with a view to ascertain the freedom or otherwise from purpuric or scorbutic taint. The history of the patient should be gone into, in order to make out the probable diathetic proclivities—acquired or hereditary.

**SEQUELÆ.**—That abscess of the liver, single or multiple, frequently follows acute and chronic dysentery, is indisputable; but whether as a result of the general condition existing, or of local pyæmic poisoning or embolism originating in the veins within the area of the ulcers, is still an open question. Dysentery sometimes terminates in permanent thickening of the parietes of the gut with eventual contraction or stricture, causing constipation, fecal accumulation or obstruction. When the seat of stricture can be reached, as in the lower part of the rectum, much relief can be afforded by simple incision and subsequent dilatation by means of bougies. Fissure of the anus, or ulcer within the verge of the anus, is a common sequel. Once diagnosed by examination of the stools and by means of the speculum, it admits of speedy relief by incision and after-surgical management.

**DIAGNOSIS.**—Dysentery can be diagnosed from diarrhœa by the abdominal tenderness, tormina, tenesmus, and the existence of dysenteric products in the stools. Dr. Edward Goodeve, late Professor of Medicine in the Calcutta Medical College, was the first to carry out the practice of washing the stools in dysentery and diarrhœa. The stools are first examined as they lie in the stoolpan. Water is then added in considerable quantity. After a short interval, to allow the dysenteric products to sink to the bottom, the supernatant fluid is gradually poured off. The washing is repeated until the foreign products remain clean and destitute of much smell. When these products are putrescent, or perhaps in all cases, it is convenient to wash the stools with a solution of carbolic acid or other colourless disinfectant. Dysentery is diagnosed from *fissure or ulcer of the rectum* by the fluid or loose character of the stools, with dysenteric products, and by the absence of ulcer, as determined by examination by means of the anal speculum.

**PROGNOSIS.**—*Favourable.*—The following are to be regarded as favourable features in the prognosis of any given case: The ordinary uncomplicated form of acute dysentery; early subsidence of the constitutional disturbance; a steady, firm, and strong pulse, with diminishing frequency and increasing power; moderate abdominal tenderness; absence of tympanites; a placid and normal expression of countenance; absence of sloughs or putrescent matters in the stools; early subsidence of tormina and tenesmus, with the

appearance of *fæculence* and the contemporaneous decrease of dysenteric products; return of appetite and power of digesting and assimilating food; and the absence of inflammatory or suppurative mischief in the liver.

*Unfavourable.*—Unfavourable features, on the contrary, are—The persistence of an elevated temperature; quick pulse, with increasing feebleness; sudden freedom from tormina, tenesmus, and abdominal tenderness, with great vital depression or collapse; doughy thickening of the colon, with dulness on percussion where there should be resonance; sudden increase of abdominal tenderness, with hiccough, nausea, vomiting, and great nervous prostration, tympanites, and peritonitic pain with constant hardness and tension of the abdominal muscles; increase of putrescent and gangrenous products in the stools, like the washings of decomposing flesh; excessive hæmorrhage from the bowel; bleeding from chapped and fissured lips, gums, and mouth; harsh, dry, black or glazed tongue; delirium; picking at the bed-clothes; and scantiness or suppression of urine.

*TREATMENT.*—Should an aperient be required in the congestive, exudative, or ulcerative stages of acute dysentery, or in sudden relapses supervening upon chronic forms of the disease, the readiest, simplest, and most painless is a tepid water enema of from two to four pints. After the operation of the enema, or immediately the patient presents himself suffering from any of these dysenteric conditions, in those cases where no preliminary aperient is indicated, a turpentine epithem or mustard plaster should be applied to the epigastrium for twenty minutes. At the same time, from twenty grains to a drachm of ipecacuanha, suspended in two drachms of syrup of orange-peel and four drachms of water, or in half an ounce of infusion of chamomile, with ten grains of carbonate of sodium or bismuth, or simply made up into conveniently sized pills, should be administered. The recumbent posture, with the head lower than usual, should be enforced. Liquids should be resisted as much as possible for an hour or two. Food should not be given for at least three hours before, and six hours after, the administration of the drug. Movement of every kind must be avoided; and the saliva should not be swallowed, but wiped away with a cloth. Thirst may be quenched by sucking pieces of ice, or, when this cannot be procured, by cold water in teaspoonsful at a time. Nausea will probably occur; perhaps, in some cases, retching and vomiting. When vomiting does occur it seldom happens before the lapse of an hour after the exhibition of the drug, and the ejected matter usually consists of small quantities of gastric secretion. Should the ipecacuanha be rejected, the dose should be repeated

as soon as the stomach has been tranquillised. It will be found beneficial to time the large doses, so as to allow of one being given night and morning, as long as their use is considered necessary. The signal for the relinquishment of these doses is freedom from tormina and tenesmus, with the occurrence of refreshing sleep, *fæculent*, bilious, or ipecacuanha stools, and restoration of the primary processes of assimilation. If no great amount of disorganisation of the mucous membrane have taken place, these favourable changes are frequently noticed after the administration of the first or second dose; and even if undoubted ulceration have set in, they are generally discerned on the second or third day, or earlier. In either case the drug should be abandoned, as the disappearance of the tormina and tenesmus, and the absence of mucus, blood, and slime from the stools, indicate the cessation of dysenteric inflammation, and that the affected portions of the bowel have been placed in the most favourable condition to undergo cure by 'resolution,' if the case have not proceeded to ulceration, or by 'granulation and cicatrisation,' if ulceration or even sloughing have already taken place. Chalk-mixture with hyoscyamus and astringents is now quite sufficient to complete the cure. In some cases ferruginous and bitter tonics are demanded, to give tone to the digestive organs, and to improve the condition of the blood. Counter-irritation, by means of turpentine epithems and mustard plasters to the abdomen, or fomentations, is a valuable adjunct in the management of the disease.

The diet should consist of chicken-broth, beef-tea, essences of chicken, mutton, or beef; sago, arrowroot, or tapioca; and small quantities of good port wine or brandy. During the active period of the disease all food should be given in a liquid form. The disturbing effect of the ipecacuanha given as above directed is only temporary. Abundance of time is, therefore, available between the large doses for the digestion and assimilation of liquid food. At the outset of dysentery, and as long as the tongue is coated, food should be avoided as much as possible; and when taken it should consist of very weak chicken-tea or barley-water. As soon as the tongue is clean a purely milk diet is the best. As the stools become more *fæculent* and consistent, solid food in the shape of tender chicken, lamb, and mutton, with biscuit and bread, light sago, rice, or tapioca pudding should be allowed. Potatoes and other vegetables should be avoided until the tone of the digestive system has been fully re-established. When the dysentery is complicated with a purpuric or scorbutic condition of the blood, the administration of the juice of the grape, orange, pomegranate or lime, and bael sherbet, are essentially necessary as dietetic rather than therapeutic agents.

Opium by the mouth is seldom required. When swallowed it 'locks up' the secretions of the liver, pancreas, and alimentary mucous membrane, rather favouring than reducing the inflammation of the solitary and tubular glands. These bad effects counterbalance the benefits derived from the sleep, diminution of peristaltic action, and temporary decrease of tormina and tenesmus consequent on narcotism. This explains why the real character of the disease is often completely masked by opium, and why apparent amendment is taking place whilst destructive ulceration and sloughing of the mucous membrane is rapidly extending. As ipecacuanha speedily brings about all the good without any of the evil effects of opium, this narcotic is not only superfluous but injurious in any form, excepting as an enema or suppository to relieve tenesmus, particularly in sigmoidal or rectal dysentery. There is less objection to uniting the ipecacuanha with such remedies as are acknowledged to possess the power of lessening the irritability of the stomach, and of increasing its tolerance of the drug, without interfering with the functional activity of those organs whose secretions we are endeavouring to promote with a view to rectify the disturbed balance of the portal circulation. On the contrary, medicines of this order may be beneficially associated with ipecacuanha—such as carbonate of sodium, bismuth, chloroform, camphor, and hyoseyamus.

When dysentery occurs in pregnant women, large doses of ipecacuanha are not contra-indicated; because, if the disease be allowed to proceed (which is more likely to happen under the old than the ipecacuanha treatment) abortion or premature labour is almost certain to follow; and when such a complication supervenes, in the later months of gestation, the mortality almost surpasses that of any other disease. When the dysenteric inflammation is summarily put a stop to by the ipecacuanha, abortion or premature labour is prevented. Under the opiate method of management, premature labour is not averted, but, in the majority of cases, occurs at the acme of the disease, when the sloughs are being thrown off; and the patient succumbs to the conjoint shock to the system. In dysentery complicated with pregnancy opiate enemata to relieve irritation in the rectum are more essential and permissible than under other circumstances.

In the acute dysentery of children ipecacuanha is invaluable. For a child of six months a grain, and for a child of one year two grains, should be given, with an equal quantity of carbonate of sodium, night and morning, until the tormina, tenesmus, and slimy and bloody stools are replaced by relief from pain and by feculent evacuations. It will not often be necessary to continue the drug beyond two or three days at a time. But it should be remembered that the disease

adheres with greater tenacity to children than to adults; and although we observe that ipecacuanha has an immediately beneficial effect in diminishing the blood, mucus, slime, and frequent stools, still we find that dysenteric or slimy motions with undigested food continue to pass. In that case the ipecacuanha, combined with chalk, bismuth, carbonate of sodium, or aromatic powder, should be repeated, once or twice a day, for a certain period, till healthy evacuations are restored. The gums must be lanced when necessary; turpentine liniment or stupes may be applied to the abdomen; weak chicken-broth or arrowroot should be temporarily substituted for milk; and, above all, food must be given in small quantities at a time, and at regularly stated periods. From the age of one year the dose is regulated by adding one grain for each additional year of age up to eighteen, when the doses indicated for adults should be employed.

In cases where evident malarious taint pervades the system and complicates acute dysentery, sulphate of quinine is indispensably necessary. A scruple of the antiperiodic will be most speedily absorbed if dissolved in water acidulated with sulphuric acid, and the exhibition of this may precede by an hour the first dose of ipecacuanha. Ten-grain doses should be given midway between the large doses of ipecacuanha, or during abatement of febrile excitement, until the feverish symptoms have been subdued. Quinine here is quite as important as ipecacuanha, for, until it has successfully checked the disturbing influence which malarious poisoning exercises upon the capillaries of the portal and general circulatory systems, the good effects which ipecacuanha produces are only temporary and incomplete. The mildest febrile exacerbations of a miasmatic origin re-excite dysenteric action, and thus undo the good effected by the action of the ipecacuanha. Hence the urgent necessity for removing without delay every vestige of masked or active malarious fever complicating dysentery. No drug enables us to accomplish this object so safely and so quickly as the sulphate of quinine in large doses.

When ipecacuanha fails to preserve the life of the patient, its failure may be generally attributed to—(1) coexistence of abscess of the liver; (2) unchecked malarious poisoning; (3) permanent enlargement of spleen or liver, or both; (4) irretrievable constitutional cachexia; (5) Addison's disease of the suprarenal capsules; (6) morbus Brightii; (7) phthisis or tuberculosis; (8) strumous disease of the mesenteric glands; (9) peritonitis, with or without perforation of the gut; or (10) the existence of extensive sloughing or gangrene.

The *advantages* of the 'ipecacuanha treatment' (for the revival of which the profession in India and England is indebted to Mr. Scott Docker, of the 2nd battalion of the 7th Royal Fusiliers, stationed at the

Mauritius.—*Lancet* of July 31 and August 14, 1858) in the congestive, exudative, and ulcerative stages of almost every form and type of acute dysentery, as well as in the acute attacks supervening upon chronic dysentery, may be briefly stated. They consist in (1) its simplicity, (2) its safety, (3) its certainty compared with any other method, (4) the promptitude with which the inflammation is stopped, (5) the rapidity with which repair takes place—(a) by resolution or (b) by granulation and cicatrisation, (6) conservation of the constitutional powers, (7) abbreviation of the period required for convalescence, (8) decrease in the frequency of chronic dysentery, (9) decrease in the frequency of abscess of the liver, (10) diminution of mortality to cases treated—all of which are accomplished, (a) without local or general blood-letting, (b) without salivation, (c) without calomel and irritating purgatives, and (d) without opium by the mouth.

Ipecacuanha in large doses may be said to fulfil many important indications. It produces: (1) all the benefits that have been ascribed to blood-letting without robbing the system of one drop of blood, (2) all the advantages of mercurial and other purgatives without their irritating action, (3) all the good results of antimonials and sudorifics without any of their uncertainty, (4) all the euthanasia ascribed to opium without masking, if not aggravating, the disease whilst the mischief is silently accumulating within. Thus, we possess in ipecacuanha a non-spoliative antiphlogistic, a certain cholagogue and unirritating purgative, a powerful sudorific, and a harmless sedative to the heart and the muscular fibres of the intestines.

The *objections* which have been urged against large doses of ipecacuanha in dysentery are, first, its 'depressing influence' kept up by nausea and vomiting; and, secondly, that it is liable to set up 'uncontrollable vomiting.'

First, the depressing power, nausea, and vomiting have all been over-estimated. Nausea is only a temporary and evanescent effect. Vomiting is an exceptional occurrence; and even when it does supervene, it seldom lasts long. As much nourishment, therefore, as may be required to support the strength can be given in the intervals between the large doses of ipecacuanha. But what contributes more to the conservation of the patient's stamina and to the prevention of depression or asthenia, is the speedy cessation of the dysenteric process accomplished by the drug, followed by refreshing sleep and the power of digesting and assimilating nourishing food. Such remarkable results as these soon reconcile any patient suffering from dysentery to an otherwise disagreeable remedy.

Secondly, when uncontrollable sickness and vomiting succeed the employment of this drug in the manner already recom-

mended, the existence of one or other of the serious conditions previously enumerated may be more than suspected. In the absence of these complications, unmanageable vomiting is seldom if ever witnessed. Hence, in a preponderating majority of the cases of dysentery met with this objection is quite untenable. The truth is that every physician who has used ipecacuanha in heroic doses soon learns that depression of the vital powers from it is not to be feared, and is surprised at the small amount of vomiting that follows its administration, and at the unexpected ease with which the stomach tolerates its presence.

When dysentery becomes *chronic* no time should be lost in counselling removal from a malarious to a non-malarious and mild climate. A sea voyage—provided easily digestible food can be secured—is often attended by the happiest results. To men so afflicted 'the salt ration,' as remarked by Dr. Maclean, 'is simply destruction.' The clothing should be warm, and flannels always worn round the abdomen; Dr. Maclean also recommends 'the use of a water belt over the abdomen for some hours daily. This acts as a fomentation, and the steady uniform pressure it maintains seems to favour the absorption of the fibrin effused between the intestinal coats. If there be much uneasiness about the fundament, a water compress over the anus affords more relief than opiate enemata.' The food should be chiefly concentrated soups, milk and lime-water, and sago, cornflour, arrowroot, &c., egg-flip with port, sherry, or brandy; or, if solid food can be digested, the tenderest chicken, lamb, or mutton, with bread and biscuit, may be allowed. Beyond airing in a carriage or chair, no exercise should be attempted. The position should generally be recumbent or semi-recumbent. The erect position excites peristaltic action, and thus disturbs the physiological rest required to facilitate the repair of the ulcers. Antiscorbutic juices should be given where there is the least taint of scurvy or purpura. Frequent blistering does much good; and all forms of counter-irritation are beneficial. In all obstinate cases a diet consisting of milk only ought to be tried; and also enemata of a solution of nitrate of silver ( $\frac{1}{2}$  grain to 1 fl. oz. of distilled water), two or three pints being passed up whilst the patient is in the knee-shoulder position and the pelvis well raised.

Gallic acid, acetate of lead, sulphate of copper, and nitrate of silver, are reputed to act beneficially. Dr. Maclean's favourite remedy, 'particularly in men returning from tropical regions, anæmic from loss of blood and the depraving influence of malaria, is the solution of the perntrate of iron. Under this remedy the whole system often rallies wonderfully, the condition of the blood improves, colour returns to the blanched cheek, the

stools become more natural and less frequent, the appetite improves, and digestion is more perfectly performed. The citrate of iron and quinine may after a time be substituted.' As nearly all chronic cases are underlain by a malarious taint, quinine should form an important element in the therapeutic management, and the greatest care should be taken to secure for the residence of the patient a climate at once mild and temperate and free from suspicion of malaria. Bathing during convalescence is an efficient and welcome auxiliary. Tepid or warm baths, medicated with Tidman's sea-salt or with nitro-muriatic acid, act in stimulating the secreting function of the skin. But it will often happen that, in spite of the most careful dietetic, hygienic, and therapeutic management, no substantial progress towards the repair of the ulcers is made, and the patient eventually dies, worn out from suffering and the asthenia consequent literally on inanition.

JOSEPH EWART.

**DYSIDROSIS** (*δυσ-*, with difficulty; and *ιδρῶς*, sweat).—See **SUDORIPAROUS GLANDS**, Disorders of.

**DYSMENORRHOEA** (*δυσ-*, with difficulty; *μήν*, a month; and *ῥέω*, I flow).—Difficult and painful menstruation. See **MENSTRUATION**, Disorders of.

**DYSOREXIA** (*δυσ-*, with difficulty; and *ῥοξις*, the appetite).—An obsolete term for impaired or depraved appetite. See **APPETITE**.

**DYSPEPSIA** (*δυσ-*, with difficulty; and *πέπτω*, I concoct).—A synonym for indigestion. See **DIGESTION**, Disorders of.

**DYSPHAGIA** (*δυσ-*, with difficulty; and *φάγω*, I eat).—Difficulty in swallowing. See **DEGLUTITION**, Disorders of.

**DYSPHONIA** (*δυσ-*, with difficulty; and *φωνή*, the voice).—Difficulty in producing vocal sounds, so that the voice is more or less enfeebled. See **VOICE**, Disorders of.

**DYSPNOEA** (*δυσ-*, with difficulty; and *πνέω*, I breathe).—Difficulty of breathing. See **RESPIRATION**, Disorders of.

**DYSURIA** (*δυσ-*, with difficulty; and *ὀρέω*, I pass water).—Difficult or painful micturition. See **MICTURITION**, Disorders of.

## E

**EAR, Diseases of.**—SYNON.: Fr. *Maladies de l'Oreille*; Ger. *Ohrenkrankheiten*.

The natural division of the ear into external, middle, and internal, suggests a rational as well as a convenient classification of the disorders to which the auditory apparatus is liable.

**I. EXTERNAL EAR.**—In examination of the external meatus and tympanic membrane, bright diffused daylight, or, when this is not obtainable, light from a bull's-eye lamp lit with gas or electric light, is the best for illumination, and the light should be reflected from a concave perforated mirror of eight-inch focus down a tubular speculum. In any operative proceedings the mirror should be worn on the forehead, as in examining the throat, but otherwise should be held in the hand. As great variations in the calibre of the auditory meatus are met with, it is necessary to be provided with specula of several sizes, the most convenient form being that known as Gruber's.

Of the affections of the external ear the most important are the following:—

**1. Eczema.**—Although the acute form of eczema generally affects the auricle and external auditory meatus, it is far more common to meet with the chronic variety. Elderly females are especially subject to eczema of the ear, and it is to its long con-

tinuance that the remarkable narrowing of the external meatus throughout its whole extent, met with occasionally in the subjects of this complaint, is generally attributable. Such narrowing will often amount to almost complete closure, and it is in these instances that eczema becomes the cause of greatly impaired hearing; for when this condition is arrived at, the passage down to the tympanic membrane is at times so small as only to admit of a very small probe. It is for this reason that, although no special methods of treatment are called for, beyond what is necessary when parts other than the ear are affected with eczema, it is of the greatest importance to keep the meatus sedulously free from secretion, and this occasionally is not a very easy matter.

**2. Changes in cartilage.**—Another condition, in which the external passage becomes subject to partial closure, is shrinking of the cartilaginous part of the meatus. This, again, is a complaint of old age, and is attributable to no known cause. It is readily relieved by the patient wearing a piece of silver tube, to keep the passage patent.

**3. Bony Growths.**—Bony growths in the external auditory canal are of two kinds, true exostoses and hyperostoses.

(1) *True exostoses* arise from the junction of the osseous and cartilaginous portions of

the canal, owe their origin to acute inflammation, and are somewhat rapid in their growth. They present very little difficulty in their removal, inasmuch as they are attached by a pedicle, and are not composed of very dense tissue.

(2) *Hyperostoses* arise from the osseous part of the canal. These growths are of ivory hardness, and have a large base. They are occasionally single and confined to one ear, but generally are multiple, and more usually than not affect the meatus of both ears simultaneously. The form which they habitually assume is that of three growths, the apices of which meet, or nearly so, in the axis of the canal. Thus, seeing that a very small opening is quite enough to permit of the passage of sonorous vibrations to the tympanum, their presence remains undiscovered until a minute piece of cerumen completely closes an already nearly closed canal, and so by the deafness thus produced leads to their detection. Their growth is very slow, as they are observed to remain without increase for many years, and although without doubt they are at times congenital, it would seem that their existence is often called into being by a local irritation, first, because in a large number of cases the individuals so suffering have been for long periods addicted to diving; secondly, they are frequently preceded by a discharge passing for years through a perforated membrane and over the canal.

**TREATMENT.**—The occasions when these growths should be removed ought to be strictly limited to two conditions: 1st, when they interfere with the escape of purulent discharge, and so place the patient in danger of cerebral inflammation; 2nd, when by absolutely closing the canal they induce great deafness. In the large proportion of cases the occasional removal of cerumen which blocks up the small opening that remains is all that is necessary, and this should be done by the use of a small hook rather than by syringing, as the passage of water behind the growths is to be avoided. When, however, it is imperative to remove bony growths for either of the above-named reasons, the best method consists in drilling them away with a dental engine, to which may be attached drills of various shapes and sizes. This operation should be performed under reflected light, and ether must be given. Owing to the extreme hardness of the growths, this process occupies considerable time, but with due care it is not a dangerous proceeding. When the object in view is simply the relief of deafness, it is sufficient to drill away the apices of the growths, but when it is done for the purpose of providing a complete escape to purulent matter it is desirable to remove the chief part of the enlargement.

**4. Inflammation.**—The external auditory meatus is subject to inflammation, diffused or circumscribed, the latter occurring

in the form of small abscesses or boils. Both affections are attended by acute pain, and in each the general health of the patient has been out of order for some time previous to the local trouble.

**TREATMENT.**—Treatment in the direction of improving the general health, and local bleeding by means of leeches applied in front of the tragus, will often rapidly relieve the diffused form of inflammation; but when it has continued for a long period (as it not infrequently does), in addition to the soft tissues the periosteum becomes affected. The passage then throughout its whole extent becomes so swelled as to nearly close the external opening, and pain is constant. The only treatment which gives complete and permanent relief under these circumstances is to make two or three free incisions down to the bone, along the whole extent of the osseous part of the canal. A convenient instrument for this purpose is a small sharp-pointed curved bistoury. As to the propriety of opening abscesses in this situation there can be no question, for, owing to the extreme denseness of the tissues and their approximation to bone in the external auditory canal, abscess in this part is slow in its progress and attended with very great suffering. These abscesses being especially liable to recur, a proper regimen and medicines appropriate to the failure in general health are required.

**5. Fungi.**—The external auditory meatus has been occasionally found to be the seat of two varieties of vegetable fungus, namely, *Aspergillus flavus* and *nigricans*. The symptoms which they have given rise to have been great irritation and a slight discharge. They have been readily destroyed by syringing, and the local application of diluted spirit of wine or weak mercurial ointment.

**6. Polypus.**—Polypus of the ear is usually preceded by inflammation in the tympanic cavity and perforation of the membrane; it is considered along with diseases of the *middle ear*.

**7. Hæmatoma Auris.** See HÆMATOMA AURIS.

**II. MIDDLE EAR.**—All affections of the middle ear originate in some part of that tract of mucous membrane which, commencing where the Eustachian tube opens into the pharynx, forms the lining of this tube and of the cavity of the tympanum, finally becoming the innermost layer of the tympanic membrane. To the character of this tissue is due the term 'catarrh,' which, in its two forms of purulent and non-purulent, is used in describing any deviation from health which, directly or indirectly, is the cause of pathological change in the Eustachian tube or tympanum.

**1. Obstruction of the Eustachian Tube.**—One of the most frequent conditions under which the Eustachian tubes become

the seat of obstruction is that met with in children or young persons. The subjects of this affection present a very characteristic aspect. They breathe almost entirely through the mouth, which, sleeping or waking, is kept partially open; their tonsils are often enlarged, and they snore loudly during sleep. The mucous membrane of the nares and pharynx is swollen, and secretes in excess. Owing to this tumid state of the fauces the passages to the Eustachian tubes in this situation do not admit of the constant necessary supply of air to the tympana. The air in these cavities undergoes partial absorption, and thus becomes more rare than that external to the tympanic membrane; the density of the outer air remaining the same, the equilibrium from pressure is destroyed; the membrane, consequently, is retracted, the chain of ossicles is pressed inwards, and thus the conduction of sound becomes interfered with—in short, the patient is more or less deaf. In these cases inspection of the tympanic membrane at once reveals the state of affairs. As the cavity of the tympanum is not involved in the catarrhal change, its translucency and lustre are not impaired; the handle of the malleus is tilted inwards, the head of this bone is unusually prominent, and there is a distinct fold crossing the upper part of the posterior section of the membrane. Where the obstruction has lasted for a long period, the membrane will appear to be almost fallen in upon the walls of the tympanum, and the promontory and incus may be distinguished. If under these conditions the tympanum be inflated on Politzer's plan,<sup>1</sup> an instant return to good hearing follows, but in the course of a few days the improved hearing partially dies away, leaving the patient, however, in some degree better than before the operation.

In all these cases a thorough examination of the pharynx should be made, inasmuch as it will be frequently found that the symptoms above mentioned are due to masses of adenoid growths in the vault of the pharynx. This condition is especially indicated when, in addition to the apparent nasal obstruction, the intonation of the voice is of that peculiar kind as of a person speaking with a bad cold: thus—"diney-dine" instead of "ninety-nine." The only effectual way to explore the pharynx is to place the forefinger behind the soft palate as far up as the posterior nares. When adenoid vegetations are present, they can in this way be readily felt, and their size and position estimated. The posterior nares will often be found to be completely closed,

<sup>1</sup> This method of inflating the middle ear (now in such general use) consists in passing a stream of air from an india-rubber bag through one nostril whilst the patient swallows some water. The operator at the same time closes one nostril with the forefinger of the left hand, and completes the closure of the other with the thumb. The mouth must be kept firmly shut.

and in severe cases the pharynx throughout is absolutely filled with them. A rhinoscopic examination gives but a very inadequate idea of the condition.

**TREATMENT.**—When adenoid growths are present they should be removed, and if this is done thoroughly the patients recover their hearing completely in the course of a few weeks, and what is almost of equal importance (this applies to the general health), nasal respiration is quite restored. The best plan is without doubt to clear the pharynx at one sitting. This may be done either under ether, or without. In each case the patient's body should be well bent forward, in order that the blood should pass out through the nostrils. Unless this position is carefully attended to in the case of a patient under ether, there is great risk of blood dribbling into the larynx and being inhaled; for, these growths being very vascular, there is a good deal of bleeding. With this precaution, however, the pharynx can be cleared of adenoid growths by the nail of the forefinger in cases where they are very soft and friable; or in other cases (the larger proportion), when they are of firmer consistence, by the use of a steel nail (introduced to notice by the writer), adjusted to the forefinger. When it is found upon examination that adenoid growths are not present, the treatment of obstruction of the Eustachian tubes will be of a more simple character. It should include the gentle syringing of the inferior nares with warm alkaline solutions. The most useful of these contain bicarbonate of sodium and borax. In using them the head should be bent forward, and a small nasal syringe employed; this is preferable to the nasal douche, since the stream from the douche is so powerful as sometimes to rush into the Eustachian tubes, and so excite inflammation of the tympanum. Politzer's inflation should be employed, but not more frequently than two or three times in a week.

If the tonsils are so much enlarged as to interfere with the respiration, it will be necessary to remove them; but the reason for this proceeding is not that they press upon the openings of the Eustachian tubes, but because their presence keeps up the unhealthy condition of the pharynx.

Under this routine of treatment the patients completely recover their hearing; the space of time during which it is necessary to continue treatment varying according to the obstinacy which each case manifests.

Obstruction of the Eustachian tubes in *adults* presents certain well-marked differences from the affection as it prevails in *children*. An ordinary cold is the beginning of the trouble. It is more usual to find one instead of both tubes obstructed, and more often than not the tympanic cavity is involved in the catarrh. Where this is not the case—and it will be evident from the

retained lustre and transparency of the membrane—the same principles of treatment as are pursued in the case of children will hold good, except in so far that the affection in grown-up persons is less persistent after the tube has been once artificially opened; and that, to effect this, Politzer's method is sometimes not sufficient, or, even if so, not as efficacious as the Eustachian catheter. It must also be borne in mind that in the treatment of cases in which one ear is healthy, by means of the catheter the affected ear exclusively may be subjected to the air-douche, whilst with Politzer's method it is impossible to avoid forcing a stream of air into the healthy tympanum, and this is not always an advisable proceeding.

*The Eustachian Catheter.*—The following is the mode of using the Eustachian catheter: 'Place the patient in a chair, and let him lean back, and steady his head with the left hand firmly fixed on the top of it; hold the catheter lightly in the right hand, with the curve downwards, and pass it quickly in this position through the inferior meatus of the nose to the posterior wall of the pharynx. When this is felt, withdraw the catheter about half an inch, and tilt the point of the curved end rather upwards, and to the left or right, according to the side which is being operated upon. Now hold the catheter and end of the patient's nose steadily between the thumb and the first two fingers of the left hand. All this time the ear of the patient and that of the surgeon are connected with the otoscope. The point of the catheter is now supposed to be in the pharyngeal orifice of the Eustachian tube; but the only certain sign of this being the case is that when air is forced into the catheter it will be heard through the otoscope to impinge upon the tympanic membrane when a stream of air is passed down the catheter.'

The catheter may be made of silver or of vulcanite, but, of whatever material, it must be inflexible whilst being used. Beyond this, suffice it to say here that in practised hands its employment is invaluable, and indispensable in the treatment of most affections of the middle ear, not only in overcoming obstruction of the Eustachian tube, but also as a means by which injection of fluids may be applied to the cavity of the tympanum. In making use of the air-douche an india-rubber bag fitted to the catheter should be employed, and in using injections to the tympanum a similar arrangement is necessary.

**2. Catarrhal Inflammation of the Tympanum.**—When the tympanic cavity has become involved in the catarrhal state, or when the affection, instead of proceeding up the Eustachian tubes, begins in the tympanum, as it frequently does, those changes have commenced which, of all others, form the most frequent impediments to the conduction of sound—in other words, which make

the subjects in whom they are found more or less deaf; and it may be broadly stated that the extent to which this affection is remediable depends directly upon the time at which the patients suffering from it apply for treatment. In the early stages, the obstruction to the passage of sound through the tympanum is solely due to the effusion of mucus in this situation, and this is easily demonstrated by the moist gurgling sound which inflation of the tympanum produces, as may be heard upon connecting the ears of the patient and surgeon by means of a piece of india-rubber tubing.<sup>1</sup> Afterwards comes what may be termed the dry stage, *i.e.* when the fluid portion of the mucus has suffered absorption, and when any of the products of inflammation may have become more or less organised, or at least in a condition which, if not interfered with, suffers no further change. The morbid conditions which result from non-purulent catarrh of the tympanum are twofold. First, those which affect the tympanic membrane, and are, therefore, demonstrable during life; secondly, those which are met with after death in the tympanic cavity. The first of these include changes in curvature, in colour, and in consistence.

The slighter changes in curvature have been noticed in speaking of obstruction of the Eustachian tubes, which condition is necessarily more or less present in all cases where the tympanum has been the seat of catarrh, and these changes are met with indefinitely increased until the state of complete collapse is reached.

In this condition the membrane has the appearance of being in close apposition to the walls of the tympanum, and lapped round the ossicles, so that the forms of the malleus, incus, and sometimes the stapes are distinctly traceable.

In so extreme an example, the membrane is generally bound down to the tympanic wall by adhesions. The first change which the membrane exhibits is a loss of its lustre and transparency: it becomes opaque. Further alterations in colour, in cases of long standing, consist in the formation of patches of brown, yellow (colour of parchment), and white. Variations in consistency will include thickening throughout the membrane, or in parts of it, especially in the cases of dense chalk deposits (phosphate of lime); and thinning in places, so observable sometimes that inflation will induce bladder-like protrusions, which, as inflation is suspended, fall back again: changes in all these respects completely metamorphosing the appearance of the membrane. After death, within the

<sup>1</sup> This tubing should always be used, whether air or fluids are being injected through the Eustachian tube, for upon the sounds thus heard, as well as on the patient's own perception, depends the answer to the question whether the inflation is complete.

tympa-num may be found collections of dried mucus around the ossicles; thickening of the lining membrane; bands of adhesion in all directions; and ankylosis of the ossicles to each other, as well as between the stapes and fenestra ovalis.

As additional evidence during life of obstruction in the tympanum, it may be mentioned that sounds from a vibrating tuning-fork placed on the vertex are intensified when such obstruction exists, and the nerve remains unimpaired. This test is especially valuable where one ear is healthy, inasmuch as the sound will be heard exclusively on the deaf side, this being due to the fact that vibrations of sound thus conveyed to the auditory nerve, on their passage outwards through the tympanum, meet with the obstruction in this position, and are reflected on to the labyrinth. The appearances above described, together with the history of the case, serve sufficiently to distinguish affections of the conducting from those of the nervous apparatus; and the sounds which are produced upon inflation of the tympanum, whether of a moist or dry character, give evidence as to whether the mucus in the cavity of the tympanum is in a more or less fluid state, or has reached the dry stage where the fluid part of the secretion has become absorbed, the more solid portion remaining.

**TREATMENT.**—In the first of these conditions, the inflation at once increases the hearing power; in the second, it produces no change in the hearing. An indication in this direction is a most useful guide in respect of treatment, which may be said to include injections of fluid or vapours into the cavities of the tympana. In the matter of the selection of these remedies, the greatest diversity of practice exists in the hands of capable authorities. Astringent solutions of sulphate of zinc, alkaline injections of bicarbonate of sodium, iodide of potassium, chloride of ammonium, and the vapours of iodine, chloride of ammonium, each and all find favour. If fluids are injected, a few drops only are to be introduced through the Eustachian catheter, a small bulb of india-rubber being used to force the fluid through the catheter. Vapours are employed by means of the many apparatus in general use. The diversity of opinion on this subject shows how difficult a problem is the treatment of the ulterior effects of catarrh of the tympanum.

The degree of improvement varies within wide limits, but the greater benefits may always unhesitatingly be predicted during the moist stage of the catarrh. Indeed, the necessity for early treatment is abundantly shown, in the instance of catarrhal affection of the middle ear, by the extremely satisfactory termination of cases treated early in the disease, and the slight relief which but too often follows when the affection has been allowed to proceed for years unchecked. In

such cases as the latter, the fact that considerable quantities of inspissated mucus have been found in the tympanic cavities, first suggested the operation of making an incision into the tympanic membrane, and attempting the removal of mucus through the incision by passing a stream of air through the tympanum. This proceeding, with certain modifications afterwards introduced, is no doubt very useful in cases favourable for its employment, but it should be reserved for those which have defied the less severe means, and where there is unmistakable evidence of an obstruction to the passage of sound through the tympanum. Experiments with the tuning-fork, already referred to, give valuable evidence in this direction; but for a detailed account of this method of treatment, introduced some years ago by the late Mr. Hinton, the reader is referred to *Questions of Aural Surgery*. Suffice it to say here that an incision about one-eighth of an inch in length is made in the posterior section of the membrane with a cataract-needle, and through this opening an attempt is made to expel from the tympanum the morbid secretion. It is not within the compass of this article to refer to the many operations which have during the past fifteen years been suggested for the relief of tympanic affections, but as in most of these cases there is no doubt that the mucous membrane which lines the tympanic cavity is the primary seat of the disorder, not sufficient success has attended these efforts to stand the test of time, or probably some of them would be practised habitually by aural surgeons in this country, on the Continent, and in America. Such, however, is not the case. One of the causes of failure in all operative measures on the tympanic membrane is to be found in the fact that it is impossible to keep any opening in this structure from healing. That such attempts at relief should have been made is not surprising when it is remembered how intractable some of these cases are, how one of the forms of catarrh which affects the tympanum is slow and insidious in its progress, and sufficiently distinctive in its character, to have applied to it by general consent the term *proliferous progressive catarrh*.

**3. Purulent Catarrh of the Tympanum.—Perforation.**—The form of tympanitis in which the effused products become purulent, is an acute and generally an extremely painful affection. Usually the pus rapidly makes its escape from the tympanum into the external meatus, by a process of ulceration through the tympanic membrane, leaving as its result a perforation of this structure. In quite the early stage the affection may often be cut short by the free application of leeches in front of the tragus, followed by fomentations, but more often than not the membrane has given way before the patient comes under observation. Even then,

if the tympanic cavity be emptied of the pus by the free use of Politzer's inflation and repeated syringing, the opening will often close, and leave very little, indeed sometimes hardly any, appreciable deafness. If, however, a purulent discharge through the opening be allowed to go on unheeded for any length of time, it is the exception for the perforation to heal. This condition is constantly seen after scarlet fever, measles, or any of the exanthemata. A perforation of the tympanic membrane presents an infinite variety of aspects, from a small pinhole to nearly complete loss of the membrane, but there will always be a slight remaining external rim of membrane. This latter is perhaps the most frequent of all forms of perforation, and especially when the ulceration dates from an attack of scarlet fever. Although the handle of the malleus occasionally remains, it more usually comes away in these and other cases where the loss of tissue is very extensive. The head of this bone, however, may always be distinguished, unless there has been complete disorganisation of the tympanic cavity. Among other forms of perforation commonly met with may be mentioned those in which the anterior or posterior half of the membrane is left, and is bounded internally by the handle of the malleus; the so-called *reniform* perforation, where the lower part of the membrane is lost, and the umbo of the malleus indicates the position of the hilus of the kidney; and the small, smooth-edged circular perforation which is common alike to all parts of the membrane. Occasionally, though not very often, the tympanic membrane is the seat of a double perforation. Similar variations in hearing accompany this condition, between slight deafness and total loss of hearing power. The size of the perforation affords no guide in this respect, extreme loss of hearing being met with when the perforation is very small, and very slight deafness where the loss of tissue has been most extensive, so that it may be unhesitatingly stated that the loss of the membrane is but in a very small degree the cause of the deafness in these cases, the disorganisation in the tympanic cavity mainly accounting for this. Such disorganisation is at times so complete (especially after scarlet fever) as to include the loss of all the ossicles, total deafness, and paralysis of the muscles supplied by the portio dura. A very small perforation in the anterior and superior part of the membrane may from its position escape notice, but the diagnosis can be always verified by the facility with which air may be made to pass through the opening, or the reverse, provided that the communication between the Eustachian tube and the tympanum is not closed by cicatricial tissue—a very rare condition when so little of the membrane has suffered ulceration.

One other form of perforation deserves mention, rather on account of its frequency than its importance, namely, perforation of Shrapnell's membrane, which is that part of the membrane proper lying just above the short process of the malleus. Ulceration of this small area, which is merely a protective membrane, may exist without any impairment to hearing so long as the remainder of the tympanic membrane is entire, so that it would appear to perform no function so far as a conducting medium is concerned, but it is important to bear in mind that when it is perforated the lesion is at times connected with exposed bone.

**TREATMENT.**—The treatment of purulent tympanitis and perforation will include assiduous cleanliness; keeping the Eustachian tube free from obstruction; and the use of local remedies. Amongst the most useful of these are weak solutions of alcohol, and insufflation of gallic acid or boric acid. As the condition of the ear improves under these measures, so will the hearing power vastly increase when it has not been completely lost; but there still remains the oft-times invaluable application of what is spoken of as the *artificial* membrane. Of all kinds, the best undoubtedly is the flattened pad of moistened cotton-wool, applied by the patient, every morning, with a pair of forceps constructed for the purpose. Until this be tried in each case it is impossible to say whether it will do good; but when it is useful—as it is in a large number of cases—by its help the patient will recover very good hearing, and this even when the perforation has existed for a period of many years. That its effects depend upon the support which it gives to the ossicles, thus re-establishing the normal pressure of the stapes upon the fenestra ovalis, has been unquestionably demonstrated.

**4. Polypus.**—One of the most frequent complications in cases of perforation of the tympanic membrane is polypus, a term employed to designate a fleshy tumour in the ear. Although polypi are occasionally present in the meatus independently of perforation, the most usual situation from which they arise is the lining membrane of the tympanum. Sometimes the exact point of origin is the edge of a perforation, and still more rarely the sides of the meatus. In size these growths vary from a small protrusion through a perforation, to a tumour which entirely fills the meatus and projects externally from the ear. In this latter instance the growth presents a very distinctive appearance, not unlike a raspberry. Sections of aural polypi hardened in chromic acid with few exceptions show the structure to be fibro-cellular, the fibrous element preponderating over the cellular in proportion to the age of the tumour.

**TREATMENT.**—In all cases polypi should be removed, and the best instruments for this purpose are the rectangular ring polypus

forceps, or, in the case of a large growth, a Wilde's snare. Owing to their remarkable tendency to recur, removal is only the preliminary step in treatment. The principal part of this consists in their complete eradication by caustics. Of these the most efficacious and convenient is chloro-acetic acid, and later in the treatment nitrate of silver. The acid should be applied with great care by using it on the point of a probe defended by a small twist of cotton, and a convenient form of nitrate of silver is a bulb of the melted salt fused on to a probe or platinum wire. The caustic should be applied daily for some time after the polypus has been removed, and then less frequently. The treatment should also include the same scrupulous cleanliness and application of astringents, so desirable in the case of perforations. It is simply to the want of attention to details that failure in the treatment of aural polypus may be ascribed.

#### COMPLICATIONS OF TYMPANIC DISEASE.—

(a) *Facial Paralysis*.—This is due to inflammation around the portio dura in its passage through the aqueduct of Fallopius. When suppuration in the tympanic cavity, with caries of the bony canal, precedes or accompanies the loss of function in the nerve, recovery is hopeless; but when the paralysis follows a subacute catarrh of the tympanum, not ending in a perforation, as is sometimes the case, the paralysis in time disappears no less certainly than when it is dependent upon an affection of the nerve at a point after its exit from the temporal bone.

(b) *Pyæmia; Cerebral Abscess; and Meningitis*.—When the mastoid cells become the seat of inflammation, the pain, tenderness, and pitting on pressure over the mastoid process, will at once suggest an early incision down to the bone, and it may be truly said that this is often delayed too long, and perhaps is never done too soon. Again, when the symptoms point definitely to pus within the mastoid cells, the bone should be pierced so as to make the external opening communicate freely with the cells. Relief given in this way will occasionally be the means of saving life, by preventing the absorption of poisoned material into the lateral sinus. Besides pyæmia thus induced, other fatal issues which suppuration in the middle ear frequently entails have their starting-point in the tympanic cavity, and in such instances cerebral abscess or meningitis may be the immediate cause of death. In the latter case a *post-mortem* examination reveals pus in the arachnoid cavity, or between the roof of the tympanum and the dura mater; in the former case, the seat of the abscess may be either in the cerebrum or cerebellum; this portion of the brain (the cerebellum) being more generally, though not always, the part affected when the mastoid cells are involved as well as the tympanum.

The experience of the past few years has amply demonstrated the success which has attended the operation of trephining in those cases where symptoms point to cerebral abscess rather than to general meningitis. The operation should be done under strictly antiseptic conditions; the point selected should be over the temporo-sphenoidal lobe; and when pus is found after an exploration through this opening the wound should be kept carefully drained.

Fatal terminations of this nature most frequently occur when cleanliness and local treatment of the ear have been neglected. Hence the necessity for such care.

But the fact that even under the most favourable conditions such events are possible, when there is a fistulous opening in the tympanic cavity, should induce caution with insurance offices in accepting the lives of persons with this lesion, at the ordinary premiums. Fatal cases of this kind might seem in practice to be almost divisible into two classes—namely, those in which cerebral symptoms come on soon after the establishment of the perforation; and others where there has been a purulent discharge from the ear (that is, from the tympanum) for many years before the advent of such symptoms. In this latter class must be included those cases in which the temporal bone has become the seat of caries; and it may be stated, subject to no exceptions, that whenever exposed bone can be detected by means of examination with a probe within the cavity of the tympanum, the subjects of this condition are always more or less in a perilous state, and that at any time fatal symptoms may commence with a severe rigor, the earliest of all succeeding symptoms. For this reason, even when exposed bone cannot be absolutely demonstrated in the way mentioned, the existence of bone-granulations where there is a perforation of the tympanic membrane should be regarded as a most serious complication. The same danger, though in a very much less degree, may be said to be present when dead bone can be detected in the mastoid process—in a less degree, because the outer table of the bone is often affected whilst the inner remains healthy. The dead bone then in the former position becomes exfoliated, and the external wound heals. Such are briefly the points of importance in connexion with caries of the temporal bone. How caries of the mastoid process may be obviated by a timely perforation of the mastoid cells, and how the chief part of the temporal bone may when carious be removed and the patient survive, may be seen on reference to a paper entitled 'Disease of the Mastoid Bone,' by the writer of this article, in the *Transactions of the Medical and Chirurgical Society* for 1879.

(c) *Malignant Disease*.—In the paper just mentioned is also reported a case of malig-

nant disease of the ear, in which the cavity of the tympanum, having been the seat of suppuration for some time, became affected with epithelial growth, which caused the death of the patient. From all recorded cases of malignant disease of the ear, as well as those which have come under the writer's notice, it would seem that the seat of origin of the new-growth will be found to be the lining membrane of the tympanic cavity, and that a purulent discharge from this surface always precedes the appearance of the cancer, and must therefore be regarded as the exciting cause of the growth. In its early stages cancer in this situation bears a strong resemblance to the ordinary forms of polypus. The same cause, then, which in some cases calls into being a polypus, may occasionally give rise to malignant disease, and this without any predisposing cause (so far as can be ascertained) in the patient towards cancerous growth.

III. INTERNAL EAR.—Apart from deafness due to local changes in the external or middle ear, the function of hearing is subject to impairment from causes which have their seat in the nervous structures of the ear; in other words, although the conduction of sound may be good, the perception of sound may be faulty. The inability to hear the vibrations of sound conveyed through the cranial bones, such as from a vibrating tuning-fork placed upon the vertex, is indicative of this condition. For the rest, the absence of tympanic disease, and the history of the case, must supply the evidence required for a diagnosis. Familiar examples of this nature are the deafness which often accompanies old age, or which is left after fevers when the middle ear has not suffered; the two forms of syphilitic nervous affection to be presently mentioned; the sudden and sometimes total loss of hearing which occasionally follows severe mental shock; the deafness after loud explosions near the ear, so common in artillerymen and naval men; and that which is caused by blows on the head and boxes on the ear. An attack of mumps will sometimes leave behind an irremediable loss of hearing in one or both ears, unattended with any discoverable change in the tympanum. In a similar way weakly women occasionally become more or less deaf during their confinement, and this symptom becomes aggravated as each successive child is born. In connexion with this subject, prolonged suckling may be mentioned as one of the numerous debilitating causes which undoubtedly aggravate the trouble of an already impaired ear.

Among the nervous affections of the auditory apparatus possessing certain characteristics which serve to distinguish it from others is the so-termed Menière's disease, an attack of which at times gives rise to symptoms which would be alarming if their true origin passed unrecognised. A patient who suffers

in this way is seized with an attack of vertigo so severe that he not infrequently falls, and for some hours afterwards requires assistance in walking; occasionally vomiting succeeds the giddiness; and he recovers to find himself very deaf in one ear, with which previously he had heard well. Milder attacks of the same nature generally follow the first, and each one leaves the patient more deaf. Although with many writers it is a favourite theory that the seat of morbid changes in this disease is in the semicircular canals, up to the present the point has not been satisfactorily determined. No treatment appears to exercise any influence upon the disease. *See VERTIGO.*

**Syphilis.**—The affections of the ear due to syphilitic disease demand separate consideration. They occur under the following varieties: Firstly, in the form of sores and warts in the external meatus, which yield to local treatment. Secondly, affections of the middle ear during the secondary ulceration of the throat, the treatment for which, beyond specific medicines, in no way differs from what is useful in the ordinary catarrh of the same parts. Thirdly, failure in hearing power during the secondary stages, unattended with any change in the middle ear. This disappears under constitutional remedies. Fourthly, the loss of function in the auditory nerve, so commonly met with in the subjects of inherited syphilis. In these patients the hearing power begins to fail between five and fifteen years of age (very seldom later in life), and proceeds to very great and often total deafness, the period between good hearing and the extreme point of deafness arrived at varying from a few weeks to several years. From this cause children sometimes become in the course of a month or six weeks totally deaf, but such rapidity is exceptional. Experience has shown how powerless treatment is to arrest the progress of this affection, so that attention should be confined to preventing its subjects from becoming dumb, if they are attacked after they have acquired speech, and before they are likely to forget it, namely, from about four to seven years of age. This is best attained by teaching them lip-reading; and if they can read, by making them do so (*aloud*) several times each day. In this way a child will retain its recollection of language when otherwise speech would pass away. W. B. DALBY.

**EAUX-BONNES, in France.**—Sulphur waters. *See MINERAL WATERS.*

**EAUX-CHAUDES, in France.**—Sulphur waters. *See MINERAL WATERS.*

**EBURNATION** (*ebur*, ivory).—A state of bone-tissue in which it assumes the whiteness, smoothness, and hardness of ivory, in consequence of an increased deposit of calcareous matter. It occurs chiefly in rheumatic arthritis. *See RHEUMATIC ARTHRITIS.*

**ECBOLICS** (ἐκβολή, abortion).—This name is given to the measures which produce abortion, or expulsion of the contents of the uterus. In moderate doses ecbolic drugs may act as emmenagogues. See EMMENAGOGUES.

**ECCHYMOISIS** (ἐκ, out of; and χυμός, juice).—An extravasation of blood into the cellular tissue, due either to injury or to disease. It presents at first a more or less blue or bluish-black appearance, which changes with age, passing through green to yellow. See EXTRAVASATION.

**ECHINOCOCCUS** (ἐχίνος, a hedgehog; and κόκκος, a grain or berry).—See ENTOZOA.

**ECLAMPSIA** (ἐκλάμπω, I flash, I explode).—This term is now used as a synonym for convulsions, whatever may be their cause. See CONVULSIONS.

**ECPHYMA** (ἐκ, out of; and φῦμα, a swelling).—A growth from the integument. The term was employed by Mason Good as a designation for warts and corns, but is at present almost obsolete.

**ECSTASY** (ἐξίστημι, I amaze).—DEFINITION.—The term 'ecstasy' has been applied to certain morbid states of the nervous system, in which the attention is occupied exclusively by one idea, and the cerebral control is in part withdrawn from the lower centres and those for certain reflex functions. These latter centres may be in a condition of inertia, or of insubordinate activity, presenting various disordered phenomena, for the most part motor.

DESCRIPTION.—The subjects of ecstatic phenomena are commonly of the female sex, or are men who lead celibate and ascetic lives. To such individuals, they are in the present day almost confined. In the middle ages, on several occasions, under special circumstances, an intense dominant emotion, with some attendant ecstatic manifestations, was known to spread widely by a sort of moral contagion.

Women who are the subjects of this morbid state are usually single, frequently present menstrual irregularities, and often distinct evidences of hysteria, of which the ecstatic condition may be but a part. The immediate cause of the attack is usually some repeated vivid emotion, commonly religious, sometimes one of fear. The direction taken by the motor or other phenomena of the ecstatic state is often very obviously determined by imitation. With this direction there is associated, in some cases, the assertion of supposed facts, which transcend the ordinary course of natural phenomena, and which have been proved, in many instances, to depend on intentional fraud.

As forms of ecstasy we have the condition of some religious enthusiasts, in whose domi-

nant state all other mental processes are merged. Dreams and visions are determined by the ecstatic emotion and add to its intensity. Consciousness of the body may even be lost, so that all sensation is gone for a time; while the corporeal functions, including ingestion and egestion, are reduced to a minimum, and a little exaggeration may represent them as in complete abeyance. Hence the 'fasting girls' that have arisen in various countries, by whom 'stigmata' marks, in the positions of the nails employed in crucifixion, are sometimes presented, probably by artificial production, but possibly by the influence of the mental state on the processes of nutrition in the parts to which attention is directed. In some hysterico-epileptics a state of ecstasy—of rapt, intense emotion—forms part of the paroxysmal seizures, and then wild muscular spasms replace the tranquil repose of the more volitional ecstatics. Occasionally—when an intense emotion is shared by many persons—insubordinate muscular movements occur, of a rhythmical character, seen in the Jumpers and Shakers of the present day, and more strikingly in some of the dancing religious ceremonies of half-civilised races, and in the dancing epidemics of the middle ages. Such was the original dance of St. Vitus, in which the exciting emotion was religious; and such also the 'tarantella,' in which the excitant was terror at the supposed consequences of the bite of the tarantula, which the dance was intended to avert. The phenomena now manifested in the epidemics that have been described under the name of the 'Jumpers,' &c., merit, indeed, more detailed notice, since they have been observed in remarkable degree and conspicuous form in certain races in whom the process of brain development concerned in what we term 'civilisation' is taking place—with a slowness that is probably alike typical as regards the past, and instructive as to the process. The conditions especially favourable for the development of these disorders appear to be generated by the close contact of such races with those in whom the process of evolution of the nervous system has been carried farther; and the less developed organisation is, as it were, strained by a connexion which it is not strong enough to endure. At all periods a pathogenic influence has probably also been exerted by the excessive tension placed on some mental faculties by certain religious systems, but the effect is greater when the strain comes from without, and the nervous constitution is warped by the inequality between its own strength and that of the race with which it is not only in contact but attachment. The cerebral functions may be deformed by emotions that would be powerless to disturb a brain whose functions were strengthened by equal development in all parts.

It is especially among the negro tribes in contact with more civilised white races, that the most conspicuous forms of ecstatic disease have been observed. A condition described by Hammond under the term 'Miryachit' (Russian, *Miriatchit* = 'playing the fool') is almost identical with that met with in the so-called Jumpers. The subjects of this condition have been met with chiefly in Siberia; but an exactly similar condition is met with in Java, where it is known as 'Lata' or 'Latak.' Both sexes are liable, but most sufferers are females. It occurs in epidemics, especially involving several members of the same family; although sporadic cases are often met with. Contact with a person who is the subject of this affection is often the exciting cause; but sometimes the malady is distinctly hereditary. Natives and acclimatised immigrants are exclusively attacked, and of them only children and persons of a low intellectual development. The characteristic of the disease is that its subjects are compelled to imitate any sound, gesture, grimace, or act, even if it gives rise to pain, anger, or remonstrance. As a rule the malady is chronic, but presents spontaneous remissions. The general health is unaffected, and the patient is not incapacitated from work. Thus the chief difference between this condition and most other ecstatic states is, that the subjects of *Miryachit* re-act only and simply to impulses entering through the afferent optic and auditory channels; and when an order is given they do not perform the acts, but simply repeat the words of the order.

The state of 'sommolentia,' or 'sleep-drunkenness,' is closely allied, psychically, to the conditions under discussion, although differing in its associations, by which it is connected more closely with epileptic mania than with the ecstatic disorders. The subjects of this condition perform some act, it may be even of violence or murder, on being suddenly awakened from sleep—the action being often prompted by a dream.

The close resemblance between ecstasy and some forms of hysteria has already been alluded to; but what is of still greater interest is the fact that suggestion plays a most important part in all the conditions that have here been discussed, and that all these conditions seem, as it were, to form links in the chain which connects the dancing epidemics of the middle ages with the phenomena of suggestion and hypnotism of the present day.

A remarkable case is quoted by Hammond, of a gentleman who would perform any act he was told to by a person whispering into his ear while he was asleep.

**TREATMENT.**—It is rarely now that ecstatic manifestations have to be treated except as part of pronounced hysteria, and the treatment is that of the hysterical state which underlies the ecstasy. The measures of para-

mount importance are the substitution of a 'healthy moral atmosphere' for that under which the symptoms have arisen; and the exposure of actual fraud. Occasionally, even now, examples of solitary ecstasy come under observation. In these cases considerable care and tact are needed. Ecstasies are not amenable to the motives which influence most persons, and, if there is actual fraud, will sometimes die rather than be found out. In the case of fasting girls, due observation of the body-weight during a short time will answer as well as, and is much safer than, a long exclusion of food. But the removal of the ecstatic to other surroundings is the most important step for both detection and cure.

W. R. GOWERS. J. S. RISIEN RUSSELL.

**ECTHYMA** (ἐκθύω, I burst out).—A pustule or pimple; pathologically occupying a mid-place between a pustule and a furuncle. The so-called tar-acne, the small inflammatory pustules developed around a mother-boil, and the commoner eruptions produced by iodine and bromine, are examples of ecthyma. See SKIN, Diseases of.

**ECTOPIA** (ἐκ, out of; and τόπος, a place). An abnormal protrusion or displacement of a part; for example, *ectopia vesicae*, protrusion of the bladder. See ORGANS, Displacement of.

**ECTOZOA** (ἐκτός, without; and ζῶον, an animal).—A term employed by some naturalists to embrace all the external parasites. See EPIZOA.

**ECTROPIUM** } (ἐκ, out of; and τρέπω, I turn).—A condition in which the eyelid becomes everted, so that the conjunctival surface is exposed. See EYE AND ITS APPENDAGES, Diseases of.

**ECTROTIC** (ἐκτιρώσκω, I miscarry).—A term applied to arrest of the course of a morbid process, for example, the development of small-pox. The agent by which the pustule is made to abort, named the ectrotic, may in this instance be a point of nitrate of silver. A coating of plaster, and especially substances which will exclude the light, such as mercurial ointment, or an ointment of lampblack, are likewise employed as ectrotics of small-pox.

ERASMUS WILSON.

**ECZEMA** (ἐκζέω, I bubble up).—SYNON.: Fr. *Eczéma*; Ger. *Eczem*.

**DEFINITION.**—An inflammatory disease of the skin, which in its typical form is associated with the formation of papules and vesicles which quickly burst, and leave a more or less excoriated surface, discharging a gummy serous fluid, which stiffens into a thin crust as it dries. The disease is always attended with itching.

**ÆTIOLOGY.**—Eczema is by far the most common disease of the skin, and is said to constitute about one-third of all the cases of skin-disease. It occurs at all periods of life, from infancy to old age, but is probably rather more common during the first and second dentitions, and in the later period of life, than in youth or middle age; on this point, however, there is some difference of opinion, and in any case, age is not a very constant factor in its development. It is generally admitted to be somewhat more common in males than in females.

The principal causes of eczema may be conveniently divided into three groups: (1) those that are strictly local, such as varicose veins, and the various forms of local irritation; (2) more general causes, such as teething, errors of diet, overwork, and anxieties of all kinds; (3) a constitutional tendency to the disease. It must be admitted, however, that there are cases of eczema for which no definite assignable cause can be found.

With regard to strictly *local* causes, varicose veins are perhaps the most important. Irritants, however, such as tincture of arnica, stimulating plasters and embrocations, often give rise to local attacks of eczema in those who are predisposed to it. Exposure to extreme heat or cold is amongst the well-recognised exciting causes of the malady, which is often a source of annoyance to Europeans living in the tropics. A Turkish bath will sometimes produce a papular eczema such as we also meet with in stokers. The handling of irritating materials, which is incidental to certain trades, occasionally gives rise to local eczema of the hands. Too frequent washing in hard water may be mentioned as a common exciting cause in those predisposed to the disease.

The *general* causes of eczema are of more importance than the local causes, although they are less certain and definite in their action. In children, the process of teething, an injudicious diet, and worms may be mentioned as generally recognised exciting causes of the disease; of these three, injudicious feeding is the most important, and is by no means confined to infancy or childhood. Constipation and a general neglect of the action of the bowels, is another common cause of the perpetuation, if not of the development, of the malady.

With regard to a *constitutional* tendency to the disease, it is, without doubt, common. In some instances, this tendency is so strong that eczema may be said to be always present throughout life, or with but short intervals of freedom. It is generally believed that gouty families and individuals are more liable to eczema than those that have no tendency to gout; the writer does not doubt this fact, but that is very different from the common statement that gout *gives rise to eczema*, for which there is not much foundation.

**SYMPTOMS.**—Eczema is remarkable for the

varying appearance it assumes, according to the acuteness of the inflammation, the stage of the disease, and the parts affected. In an early stage it generally consists of a red and slightly swollen patch; later on, this may be covered with papules and vesicles, which are usually of short duration, to be followed by the development of a moist excoriated surface, discharging a serous or gummy fluid. When the process of healing begins, the patch becomes dry, and covered with thin scaly crusts, which ultimately fall off, and leave the skin in a comparatively healthy condition. Although this is the description of typical eczema, yet the varieties in the course and character of the eruption are so great that they require a brief notice.

There are three principal variations in the development of the eruption: (1) The inflammation may be very acute, and the exudation of serous fluid into the skin and subcutaneous structures so rapid and excessive as to produce great swelling of the tissues. This is especially met with in acute eczema of the face, in which there is sometimes closure of the eyelids, and a complete alteration in the features, so that the patient can be hardly recognised. (2) A very common variation from the normal course of the eruption is the development, from the first, of dry, red, slightly scaly patches, which are never moist, and but little swollen. Eczema of this kind is always attended with much itching, but with no pain or tenderness on pressure. (3) A general eruption of minute red papules over the body and limbs, is another very common form of the disease. The papules usually develop rapidly, and are distributed symmetrically over the body, reminding one, at a little distance, of a scarlatina rash. The eruption generally aborts, dries up, and desquamates without the formation of visible vesicles, though now and then a few may be found amongst the bright red papules.

The various forms or stages of eczema are sometimes indicated by qualifying names; thus the earliest phase consists simply of hyperæmic patches and is called *eczema erythematosum*; later on we may have *eczema papulosum*, and *vesiculosum*; soon the vesicles burst, and leave a raw, discharging, excoriated surface, to which the term *ichorosum* has been applied; when the congestion is great, it is called *eczema rubrum*. The formation of a puriform discharge is indicated by the name *impetiginosum*; and lastly, *eczema squamosum* is applied to the disease in its scaly condition. One or all of these phases of eczema may sometimes be seen in different regions of the body at the same time.

Although the skin of any part of the body is liable to be attacked by eczema, yet it may be said to have a preference for certain regions, as, for example, the flexor surfaces of the limbs, the hands, the scalp, and the points of junction of the skin with the mucous

membranes. In some of these regions, such as the hands, the lips, and the anus, local causes play an important part in the perpetuation, if not in the development, of the malady. Eczema is generally distributed symmetrically, though there are many exceptions to the rule. With regard to subjective sensations, it may be said that they are always present, and form one of the most troublesome symptoms of the disease. In most cases itching is a very characteristic feature; the only exception to this rule is met with in acute eczema, in which the sensations of pain, burning, and smarting for a time mask or take the place of itching; but when the inflammation subsides, itching is sure to arise. In no other affection, except prurigo, is itching so constantly present.

**DIAGNOSIS.**—Eczema is liable to be mistaken for the following diseases: erysipelas, scabies, psoriasis, sycosis, tinea tonsurans, superficial lupus, and some syphilitic eruptions.

*Eczema and Erysipelas.*—Acute eczema of the face, with much exudation into the subcutaneous tissue, is the only form liable to be mistaken for erysipelas. The following points will aid the differential diagnosis: (1) In erysipelas, the eruption is preceded by rigors. (2) The pulse, temperature, and febrile symptoms generally are higher than in eczema. (3) There is almost always headache, and often albuminuria. (4) Erysipelas has a more sharply defined margin, and a smoother and more glazed surface. (5) Erysipelas commonly begins round or below one orbit, and is, more often than eczema, unilateral at first, although, in the end, both sides may be affected.

*Eczema and Scabies.*—These two diseases are often confounded, for the very obvious reason that scabies leads to a kind of artificial eczema. Apart from the use of the microscope, which cannot always be applied, the following considerations will aid the differential diagnosis. Scabies especially attacks certain regions, and when a pimply eruption is met with between the fingers, and on the flexor surface of the wrists, and also at the same time on the lower part of the abdomen, penis, and nates, a very careful examination should be made for other signs of scabies. An examination over the wrist with a common magnifying-glass will often lead to the detection of the scabies cuniculus, looking very like a pin-scratch. The history of the case will also aid the diagnosis.

*Eczema and Psoriasis.*—Dry scaly forms of eczema may be mistaken for psoriasis, though the more sharply defined and raised margin of the latter, with a tendency to form rings, usually makes the diagnosis easy. The development of the eruption on the points of the elbows and just below the knee-caps, is very distinctive of psoriasis.

*Eczema and Sycosis.*—The very chronic

character of sycosis, and the absence of any running exudation, together with its strict limitation to the hairy parts of the face, will generally serve to distinguish it from eczema. In a recent attack the differential diagnosis is difficult, but not of much importance, as the two diseases are very closely allied.

*Eczema and Tinea Tonsurans.*—It is only ringworm of the body that can be mistaken for eczema. A solitary patch of dry eczema-like eruption, spreading at the margin, at once suggests the probability of ringworm. The diagnosis will be rendered almost certain if a narrow margin of the round patch is sharply defined, slightly raised, and of a brighter colour than the other parts. If no treatment has been adopted, the microscope will complete the diagnosis; but if any active remedies have been applied, the microscopic examination is useless.

*Eczema and Lupus.*—Patches of superficial lupus sometimes become covered with scales and eczema-like crusts, which mask the real nature of the disease. A careful examination will, in any doubtful case, lead to a correct diagnosis.

*Eczema and Dermato-syphilis.*—Pustular syphilitic eruption of the scalp may be mistaken for impetiginous eczema. The presence of superficial ulceration, and a peculiar offensive smell, are characteristic of the syphilitic eruption, apart from other signs of syphilis. Dry, cracked eczema of the palm is very common, and is sometimes confounded with a similar condition which is due to syphilis, and often mis-called 'psoriasis palmaris.' This kind of eczema is generally attended with much itching, which is not the case with the syphilitic eruption. Eczema is, moreover, rarely confined to one palm, while the syphilitic eruption is often unilateral. The history of the case will also aid the diagnosis.

**TREATMENT.**—The variations in the character of eczema are so great that it is impossible to lay down rules of treatment that will meet all cases. Some general directions may, however, be given, which will serve as guides, though nothing can supply the place of practical experience.

*Acute Eczema.*—The treatment of acute eczema is simple. In all very severe cases the patient must be confined to bed or the couch. Moderate or free purging with sulphate of magnesium or sulphate of sodium is always indicated, some care being of course required in the case of weakly or elderly people. When the attack is of gouty origin, the purgative treatment may be supplemented by the use of alkalis with iodide of potassium or colchicum, according to circumstances. In the local treatment of acute eczema, all ointments of a stimulating kind should be strictly avoided. If the inflammation is confined to the limbs, water-dressing or a weak lead lotion is often the best application, but it must never be allowed to

get dry. During the day, when the dressing can easily be kept moist, it is better not to cover it with gutta-percha tissue; but at night this plan may be adopted, in order to prevent the lint from getting quite dry. It is difficult to apply water dressing to the head and face, and under these circumstances fuller's earth or starch and zinc powder is a useful application. If, however, this does not suit, the *linimentum calcis*, made with sweet almond oil, may be used; it should be either applied on lint or simply painted on several times daily. Some patients derive great benefit from thin linseed meal poultices, which do not require changing so often as water-dressings, otherwise the principle of treatment is the same in both.

*Sub-acute or Chronic Eczema.*—The *general treatment* of sub-acute or chronic eczema will depend more on the character of the eruption, and the age of the patient, than on the parts affected; the *local treatment*, on the other hand, must be modified to suit the regions attacked. The general treatment would include rest, change of air, diet, and medicine, according to the severity of the case, and other attendant circumstances. If the patient is suffering from gouty symptoms, dyspepsia, or constipation, the plan of treatment should be adapted to meet these conditions, with a considerable probability that it will also be the most suitable treatment for the eczema, which may of course be dealt with at the same time by local means. When, however, the patient is young and in good health, arsenic in moderate doses will be found the most generally useful remedy. In children between the ages of two and five, one-drop doses of the liquor arsenicalis given after meals three times daily is usually enough; it may be combined with alkalis or laxatives or tonics according to circumstances. The routine practice of treating ordinary eczema in young children with iron and cod-liver oil generally ends in failure, especially when the eczema is of an irritable kind. In the pustular forms of eczema, however, iron is useful, and then the arsenic may be combined with the *vinum ferri* or some other preparation of iron. In people of middle age, arsenic is not so generally useful as in children. In the sub-acute irritable forms of eczema, *vinum antimoniale* in small doses, with diuretics, may often be given with advantage instead of arsenic.

In all forms of eczema the greatest attention should be paid to the action of the bowels. The malady is often associated with constipation, and even when this is not the case, some increase in the natural action of the bowels is highly beneficial. The kind of purgative to be used will depend on the age of the patient and other circumstances. In infancy, fluid magnesia in milk is one of the best; in childhood, sulphur powders or lozenges, or mild saline purgatives, are

most suitable; for adults, sulphate of magnesium or sulphate of sodium, given either before breakfast in full doses, or in smaller doses with some tonic two or three times daily, is suitable; lastly, in old age, mild aloetic pills may be given.

*The use of Tar in Eczema.*—For many forms of chronic dry eczema, the various preparations of tar are among the most useful local remedies. They do more to relieve the intolerable itching than any other class of remedies, the chief drawbacks being their disagreeable colour and smell. Before applying tar preparations to eczema, it is always advisable to try the effect in a much diluted form, or over a very limited area; for when applied to unsuitable cases they sometimes produce a great aggravation of the disease. The ordinary tar ointment may be freely diluted with any other ointment, such as zinc ointment, and subsequently the quantity of tar increased or diminished according to circumstances. The tar lotions are less disagreeable than tar ointment; they are made by combining the liquor carbonis detergens (made from coal tar), or the liquor picis compositus (made from vegetable tar) with water, or some simple lotion, such as the ordinary lead lotion. One to four per cent. of the tar solution is usually enough.

*Local forms of Eczema.*—*Eczema of the Scalp.*—Special local treatment is sometimes required when eczema attacks certain regions of the body. On the scalp, when it is attended with much discharge, the hair should be cut and kept quite short. All the scabs should be softened by a poultice, and then removed by bathing with warm water. The head should then be carefully dried, and dressed with an ointment consisting of acetate of lead gr. xv, zinc ointment ʒij, calomel gr. xx, nitrate of mercury ointment gr. xx, vaseline ʒij, benzoated lard ʒss. When the inflammation has subsided, the white precipitate ointment, diluted with an equal quantity of vaseline, may be used. In dry, scurfy eczema of the scalp, the best salve is the red oxide of mercury ointment ʒij, white vaseline ʒvj; this should be applied in small quantities every day. Sometimes when there is much active inflammation, a lotion may be used instead of an ointment until the inflammation has subsided. The most useful are weak sulphate of zinc or boric acid lotions, or carbolic-acid lotion of strength of about one part in a hundred parts of distilled water. Whenever a lotion is used it should never be allowed to get dry; the dressing may be conveniently kept in its place by a light thread or cotton nightcap.

*Eczema of the hairy parts of the face* should be treated very much like eczema of the scalp. If, however, there is pus-formation in the hair-follicles, it is best to remove the hairs that pass through pustules, and allow the pus to escape; then to wash with

weak, strained gruel, and after carefully drying the skin to apply a lotion of calamine and oxide of zinc, so as to dry up the discharge as quickly as possible. The crusts which form, if firmly attached, should be allowed to remain, but when loose from exudation underneath, they should be removed, and, the hair being carefully cut, the surface should then be dabbed with a little medicated wool, and the lotion again applied.

*Eczema of the Anus.*—This is a most troublesome malady, the itching being often out of all proportion to the amount of visible eruption; when this is the case, it is commonly called *pruritus ani*. The most important point in the local treatment of eczema in this region, is always to wash *directly* after the bowels have acted, *without the use of paper*. For this purpose a jug and basin and a very soft old towel should always be kept in the water-closet. After washing, the eczema should be very carefully dried, and some suitable ointment immediately applied. Among the best are the calomel, the diluted oxide of mercury, and the calamine ointments; a very small quantity of the creasote ointment may be advantageously combined with any of these whenever pruritus is the chief feature of the disease. An occasional painting with a solution of nitrate of silver (gr. x ad ʒj) is very useful. Although it is essential to wash directly after the bowels have acted, yet this should not generally be repeated until the bowels act again, too frequent washing being bad for the eczema. A little cotton-wool placed so as to keep the skin on the two sides from touching is very useful.

*Eczema of the Auditory Meatus.*—Any accumulation of crusts should be first removed by syringing with warm water; this should be followed by the use of a tepid sulphate of zinc lotion (gr. iij-vj ad ʒj) several times daily; an occasional painting with a solution of nitrate of silver (gr. viij ad ʒj) is often useful. When the eczema is of the dry kind, citrine ointment diluted with six parts of oil or vaseline, and applied to the inside of the ear carefully with a camel's-hair brush, is the best remedy.

*Fissures in Eczema.*—Fissures are apt to occur in eczema at the corners of the mouth, between the toes, about the anus, and along the lines of flexure in the palm of the hand. The best treatment for these is to carefully paint a solution of nitrate of silver (gr. xv ad ʒj) into the *bottom of the crack*, with a very fine camel's-hair brush, once a day.

*Eczema of the Nipple* is often difficult to cure. It should be *protected from touching the clothes by a shield*, and painted daily with a solution of nitrate of silver (gr. x ad ʒj).

*Chronic Eczema of the Palm* is a most obstinate malady. This arises from the natural thickness of the cuticle, which does

not readily allow the escape of exudation, but cracks and leaves troublesome fissures, especially at the lines of flexure. Moreover, the hands are constantly exposed to the unfavourable conditions of movement and frequent washing. In these cases, if the outer cuticle is much hardened and thickened, it is necessary to remove it. This may be done either by macerating it with a weak lotion of liquor potassæ and distilled water (ʒij ad ʒviij) applied on lint, or by painting it for a few days with a saturated solution of salicylic acid in flexible collodion. When the hard cuticle is removed, the hand should be dressed with the oleate of zinc, or some other soothing ointment. Gloves should be constantly worn, so as to avoid the necessity of washing until the hand is quite well.

*Eczema of the Leg.*—The special indication in the treatment of eczema of the leg is careful bandaging from the foot up to the knee. In some few cases the solid india-rubber bandages are most useful, but they are not always easily borne.

*Pruriginous Eczema.*—Some forms of dry eczema affecting large areas of the body are attended with such intense itching that the patient loses all power to resist the temptation to scratch. As a consequence the skin is torn and lacerated with the nails of the sufferer, which only gives temporary relief. Under these circumstances Professor Unna recommends a kind of gelatine paint made by combining zinc oxide and gelatine, each one part, and glycerine and water each four parts. This is melted when required by placing the jar in hot water, and then painted on with a soft shaving brush. The remedy is rather a disagreeable one, but sometimes useful to prevent scratching in severe cases.

*Diet.*—A careful diet is of considerable importance in the treatment of eczema, especially in children, and in gouty or dyspeptic people. Children suffering from *pustular eczema* require a liberal diet, and especially a little beer or wine, and plenty of fresh meat and vegetables. Even infants with pustular eruptions often require alcohol in small quantities, the best form being from 15 to 20 drops of French brandy in milk two or three times daily according to circumstances. It is the formation of pus which appears to indicate the use of small doses of alcohol. On the other hand, dry irritable eczema in adults is often benefited by a reduction in the usual amount of alcohol. Some kinds of food, such as highly salted meat and fish, sweets—including dried fruits, coffee, and oatmeal, appear to be unsuitable for most kinds of eczema.

*Washing in Eczema.*—Perhaps there is no more common mistake in the treatment of eczema than too frequent washing; indeed, the mistake is a very natural one. In all ordinary cases of eczema, *washing with soap and water must be forbidden*; and

this is most essential when the eczema is healing, and the new, delicate cuticle is forming, for then water macerates and destroys it, and thus the duration of the disease is needlessly prolonged. As a matter of fact, starch or fuller's earth powder keeps the skin quite clean, and also favours the healing process. If washing is absolutely necessary, as it is sometimes in an early stage of the disease where there is much discharge, weak, strained gruel, made with soft or distilled water, is always preferable to soap and water, but even that should not be used often.

*Baths in Eczema.*—It is difficult to lay down any definite rule with regard to the use of baths in eczema. In some forms of eczema, especially in young children, baths do much harm. In gouty people, on the other hand, who suffer from dry chronic eczema resembling psoriasis, a course of mild alkaline baths is often very beneficial. Great discrimination, however, is required in the choice of baths. The summer is the most suitable time of the year for the bath treatment.

ROBERT LIVEING.

**ECZEMA MARGINATUM.** — See *TINEA TRICHOPHYTINA*; and *ERYTHRASMA*.

**EFFUSION** (*e*, out; and *fundo*, I pour). The escape of a fluid from its natural channel or cavity into the substance of organs or the cellular tissue, or from free surfaces. As examples may be mentioned dropsy in its various forms, and effusions resulting from inflammation.

**EGYPT, UPPER.**—A very dry, tonic, winter climate. Mean winter temperature, 62° F. Season, October to March. See *CLIMATE, Treatment of Disease* by.

### ELECTRICITY IN MEDICINE.

The purposes for which electricity is employed in medicine are various. It is used as a stimulant to excite muscular and nervous tissue which is the seat of paralysis or pain, and as a stimulant to the tissues generally; its chemical action may be employed for dissolving tissues and coagulating blood within aneurysmal sacs; and its thermal effects are employed in surgery for heating cauteries. The incandescent electric light has been adapted for the illumination of the throat, as well as of the bladder and other cavities of the body. Electricity is used in its three forms of—(1) *Franklinic, static, or frictional* electricity; (2) *Faradism, i.e.* electricity generated by induction, whether voltaic or magnetic; and (3) *Galvanism or Voltaism*, which owes its existence to chemical action.

**APPARATUS.**—1. *Franklinic electricity*, which is generated by the friction of glass discs or cylinders, and which was formerly much in vogue, may again possibly take its place as a

recognised therapeutic agent. Charcot contends that in some cases of muscular wasting, franklinism will produce a contraction where both faradism and galvanism fail to do so; and in some cases of 'hysteria' also franklinism seems to be of undoubted service.

2. *Faradic batteries* should consist of (a) a coil of insulated copper wire, the ends of which are in connexion with the plates of a galvanic cell. This is called the *primary coil*, and contains a bundle of soft iron wire in its interior. (b) A second coil of insulated copper wire, made of finer wire, and containing a greater number of spirals than the primary, over which it is made to slide; this is called the *secondary coil*. (c) An *interrupter*, capable of interrupting the current automatically and with great rapidity, by means of the constantly recurring magnetic action of the bundle of iron wires in the interior of the primary coil. These batteries should have means of graduating the intensity of the current of either coil, and of including the patient in the circuit of either coil without altering the connexions of the conducting wires. Sometimes the galvanic element is replaced by a large magnet, as in the well-known rotating magneto-induction apparatus.

3. *Galvanic batteries*, when used for therapeutic or electrolytic purposes, should be composed of (a) a large number of small cells of low electromotive force, and as constant in their action as possible. The cells known as the 'Leclanché' are the best. (b) Means of including in the circuit any number of cells at will, so that thereby the intensity of the current may be regulated. (c) Means of opening or closing the circuit at will, and of reversing the direction of the current without altering the position of the current-carriers (rheophores) on the patient's body. This is effected by means of a simple contrivance known as a *key and commutator*. (d) A galvanometer. Practitioners who only make occasional use of electricity should select batteries of the simplest construction; and for rough therapeutic purposes a box of cells provided with screw terminals (one terminal for every third or fourth cell) is all that is necessary. Accessories, such as interrupters, reversers, and galvanometers, should, if needed, be purchased independently and connected with the battery when required. The galvanometer is, for the purposes of exact diagnosis, for physiological experiment, and for electrolytic operations, quite indispensable, as by its use alone are we able to say what strength of current is operating. Not only does the electromotive force of the cells employed vary momentarily, but the resistance of the body varies in the same and in different individuals, so that to say that a current from 'so many cells' has been used is no guide. The galvanometer alone is able to measure the intensity of the current em-

ployed, and these instruments are calibrated to register intensity in milliamperes; and in recording we say that 'the muscle contracted with a current of so many milliamperes,' &c.

Batteries which are required for heating wires and cauteries must be composed of cells of high electromotive force, and as large as is practically possible. The number of cells or elements is of less importance than their size. It is not unusual to have, for this purpose, Groves's or Bunsen's cells capable of containing a pint of liquid each. Accumulators or storage batteries are now much used for this purpose.

The essential and distinctive differences of the two forms of current (galvanic and faradic) will now be described.

**The Galvanic Current.**—The galvanic current is (1) *continuously evolved*; hence it is spoken of frequently as the 'continuous current;' (2) It *flows always in the same direction—i.e. from the positive pole*, which is in connexion with the copper or receiving plate, to the negative pole, which is in connexion with the zinc or generating plate. This fact must be constantly borne in mind, because the action of the two poles is markedly different, and it has been asserted (on very doubtful evidence, however) that the direction of the current in the body, whether towards the nerve-centres or towards the periphery, has an important effect upon the physiological and therapeutical results. (3) It has well-marked *chemical and thermal effects*. This action is most marked at the negative pole, with which, if a moderately intense current be used, heat, redness, inflammation, and even sloughing of the skin may be readily produced. It is therefore always necessary to frequently change the position of the negative pole on the body. (4) It has *electrolytic effects*. When a galvanic current is passed through a conducting compound liquid, decomposition of the liquid results, oxygen (if water only be used) and acids (if saline solutions be used) being evolved at the positive pole, while hydrogen or alkalis, as the case may be, are evolved at the negative pole. Since the human body consists of a mass of cells which contain and are bathed in saline fluids, many of the phenomena observed on passing galvanic currents through the human body, or any part of it, are probably due to this electrolytic action. Faraday called the positive pole the *anode*, and the negative pole the *cathode*, and these terms are frequently employed.

**Induced Current.**—With regard to the induced current, the following points must be remembered. (1) It is *momentary in duration*. (2) Its *direction is constantly changing*, so that, in using it, it is less necessary to distinguish between the poles. (3) Its *chemical, thermal, and electrolytic effects are nil*. (4) It has much greater '*tension*' than the galvanic current—that is, it over-

comes the resistance of the body with far greater ease. (5) It *causes the contraction of healthy muscle* far more readily than the galvanic current. Muscular contractions only occur at the moment of making or breaking a current, and it is mainly to the rapid interruptions of the induced current that its high stimulating power is due. The current of the secondary coil has greater tension, more penetrating power, and greater stimulating power than the current of the primary coil or 'extra-current.' This is due to its being composed of finer wire, and having a greater number of turns.

The stimulating effects of the galvanic current which cause muscular contraction occur only at the moment of making or breaking the current, and not during its continuance, and the stimulating effect of the two poles is different, as may be demonstrated by the 'polar method of investigation,' instituted by Brenner of St. Petersburg. If one rheophore be placed on an indifferent part of the body, as the back, and the other be placed over a nerve-trunk or muscle, we are able by means of the commutator and key to study the action of either pole on nerves and muscles during the making and breaking of the current. With weak currents it is found that contraction ensues only when the stimulating rheophore is negative (cathode), and only on closing the circuit. This is called Cathodal Closure Contraction (C.C.C.) If the strength of the current be slightly increased, we get contraction also when the stimulating rheophore is positive (anode), and the circuit is opened (A.O.C.) Next follows Anodal Closure Contraction (A.C.C.), but Cathodal Opening Contractions (C.O.C.) never occur in healthy muscles with any currents short of those of unbearable intensity.

The galvanic current, unlike the induced current, affects the nerves of special sense. If it be applied in the neighbourhood of the eyes, flashes of light are seen, and blindness has resulted in one case from the incautious application of strong currents to the face. The gustatory nerve is affected in a similar way, and the 'galvanic taste' is perceived when the rheophores are placed on the cheek. The taste is acid with the positive, but metallic and coppery with the negative pole. If the rheophores are held to the ear, rumbling noises are produced, and it is said that stimulation of the olfactory nerve will give rise to a peculiar smell.

Onimus has pointed out a further distinction between the induced and the galvanic current. 'An induced current,' he says, 'only acts during the infinitely short time of its passage, after which everything returns to order. . . . It can never be anything else but a series of slight excitements. With constant currents real excitement is determined only at the times of making and breaking.

. . . It is during the silent period, however, that the principal action of the continuous current makes itself felt.'

It is certain that the rapid interruptions of the induced current, and the strong muscular contractions caused thereby, are capable, if the current be moderately strong, of rapidly and completely exhausting the irritability of a muscle, and, if this fact be not constantly borne in mind, harm instead of good will result from the application of faradism. The galvanic current, on the contrary, possesses remarkable refreshing effects, a fact which has been demonstrated by Heidenhain on frogs, and by the writer on the human subject. It is found that a man can sustain a weight at arm's length far longer than he otherwise could, if a galvanic current be passed through the nerves of the limb. The feeling of fatigue can be removed by the application of the current, and the force of muscular contraction is increased thereby. These facts have important therapeutic bearings.

MODES OF APPLICATION.—To apply electricity we need to have conducting wires and rheophores or current-carriers attached to the battery. The best conductors are made of ordinary telegraph wire, which should be as thick as is compatible with pliability. Telegraph wire is not damaged by moisture, and can be readily connected to all forms of batteries and rheophores, and is therefore economical as well as convenient. Rheophores vary much in design. They should all have insulating handles, and the junction between the conductor and rheophore should be about the middle of its length, so that both conductors may be held in one hand without risk of the metallic junctions coming in contact. The most generally useful rheophore is the sponge-holder. An excellent sponge-holder, which retains the sponge with absolute firmness, is that designed by Kidder of New York. Rheophores should be of different sizes, from a sponge as big as half-a-crown to the pointed extremity of an olive-shaped conductor. They should be made of metal and not carbon, because the latter is too brittle. They may be obtained in the form of discs, balls, points; and of endless design for reaching particular organs and regions, as the eye, ear, larynx, bladder, rectum, and uterus. They are usually covered with wash-leather, and used moist. A wire brush is useful for influencing the skin. The dry hand of the operator, who allows the current to pass through his own body to that of the patient, may be used for the same purpose. It is often convenient to fix one rheophore to the patient's body, which may be accomplished by placing an ordinary sponge on the surface of the body, laying thereupon the naked end of the conducting wire, and securing the whole with one turn of a bandage. If we wish to influence the surface only, we may use one rheophore dry; but if we wish to affect

deeper-lying structures, we must overcome the resistance of the epidermis by thoroughly moistening it with hot salt-and-water. We may use one rheophore dry, and one moist; or we may use as a rheophore a porcelain or gutta-percha vessel containing water, into which the limb is placed.

There are two methods of applying electricity, known as *general electrification* and *localised electrification*. By the former method we pass the current through the entire body or great part of it; it has been employed for many diseases, but its utility is doubtful. By the latter method, which we owe to Duchenne, we seek to influence special nerves, muscles, or organs, and to limit the action of the current strictly to these parts. If we wish to influence a muscle, we may do so either by applying the rheophores directly over the fibres of a muscle, both the rheophores being held in one hand and 'promenaded' over the whole surface of the muscle. This is the *direct* method, and is the method advocated by Duchenne. Or, instead of trying to influence the muscle itself, we may stimulate the nerve supplying it, and so cause the muscle to contract. This is the *indirect* method, advocated by Ziemssen. It is effected by using two rheophores of different sizes. A large rheophore is affixed to an indifferent part of the body, while with a small pointed rheophore an endeavour is made to touch the exact point where the nerve we seek to influence is most superficial. Neither of these methods is to be exclusively adhered to. Certain deep-lying muscles, such as the diaphragm and the supinator brevis, are only capable of indirect stimulation. It will be found also that, in certain diseased conditions, muscles will not respond to stimulation through the nerves, but only to the direct application of the current. When we wish to use the refreshing effects of the current, as in cases of paralysis which are on the road to recovery, or in cases of fatigued muscles, &c., it is advisable to combine the application of the galvanic current with a rhythmical exercise of the affected muscles. Benedict lays it down as a rule that the *locus morbi*, be it brain, spinal cord, nerve, or muscle, should always be included between the rheophores.

DIAGNOSTIC AND THERAPEUTICAL USES.—

1. **Diagnostic Uses of Electricity.**—For purposes of diagnosis, electricity is of undoubted service, since by its means we are often enabled to distinguish between paralysis due to central lesion and paralysis due to peripheral lesion. We are accustomed to speak of a paralysis as 'central' so long as that portion of a nerve-centre is sound from which the nerves supplying the paralysed muscles take origin. Thus in cases of damage to one internal capsule, the spinal cord and the greater part of the brain being healthy, we speak of the case as one of central paralysis. In cases, too, of paraplegia from

local injury, the cord below the injury being healthy (notwithstanding that all mental control is cut off), we speak of the paralysis as central. In such cases of central paralysis we find (a) that reflex stimulation of the muscles is possible, (b) that the muscles undergo but little wasting, and (c) that the irritability of the muscles to faradism is scarcely diminished. We speak of paralysis as due to a 'peripheral' lesion whenever the paralysed muscles are cut off from communication with their nerve-centres, or directly communicate with centres whose physiological activity has been destroyed by disease. In such cases we find (a) that reflex stimulation of the paralysed muscles is no longer possible, (b) that the paralysed muscles waste with remarkable rapidity, and (c) that the irritability of the muscles to faradism is rapidly diminished, and, as a rule, is ultimately destroyed. To establish the fact of diminished irritability to faradism is generally not difficult, and in cases of paralysis affecting one side of the body only it is done by comparing the paralysed muscles with their healthy fellows. We must take care that the current does not vary in intensity, and that it passes through exactly similar lengths of the body for the stimulation of both sets of muscles. It will be found convenient to fasten one rheophore to the middle line of the body (a big sponge tied to the back of the neck answers well), and then, when the patient's limbs are arranged exactly symmetrically, test the healthy muscles first with a small or pointed rheophore, and determine the current of least intensity which will cause contraction. Then the rheophore is to be applied to exactly the same spot on the opposite limb, and, if contraction follows as readily as on the healthy side, we know that there is no diminution of irritability. If, however, contraction do not follow, we increase the strength of current, and so determine to what extent the irritability is diminished, and whether or no it is completely extinguished. We should add that in cases of peripheral paralysis the diminution and extinction of irritability do not supervene immediately on the occurrence of the paralyzing lesion, but only after the lapse of a week or ten days. In paraplegia or other morbid conditions in which paralysis affects both sides of the body it is, of course, impossible to compare a paralysed muscle with its healthy fellow, and in such cases we can only judge of the amount of irritability by experience and mental comparison with previous instances. It is found, in cases of peripheral paralysis, that after complete extinction of faradic irritability the muscles will respond to a slowly interrupted galvanic current, and that not infrequently the irritability of the muscles to galvanism is greater on the paralysed than on the sound side of the body. It sometimes happens that not merely quan-

titative but qualitative changes take place in the irritability of these muscles, and that the Anodal Closure Contraction (A.C.C.) soon becomes very marked, and equals or even surpasses in force the Cathodal Closure Contraction (C.C.C.); and further that the Cathodal Opening Contraction soon becomes more marked than the Anodal Opening Contraction. These reactions, which are supposed to depend upon degenerative changes in the muscle and motor nerve-endings, have been spoken of as the *degenerative reactions*. These quantitative and qualitative changes in irritability are found (1) in some forms of paraplegia due to degenerative changes in the cord; (2) in so-called spinal paralysis both of infants and adults; (3) in traumatic paralysis due to injury of the nerve-trunks; (4) in rheumatic paralysis, that is, paralysis due to 'rheumatic' thickenings of the neurilemma; and (5) in lead-paralysis.

Its occurrence is probably determined by the degeneration of the motor nerve. In one form of 'rheumatic' paralysis—paralysis of the musculo-spinal nerve from cold, as described by Duchenne—the reactions of the paralysed muscle remain normal.

In ordinary hemiplegia the irritability of the muscles remains, as a rule, unchanged. In some cases, however, the irritability is increased in the early stages; and occasionally after the paralysis has lasted some time we find slight diminution of irritability. In some of the conditions confusedly known as progressive muscular atrophy, the irritability of the wasted muscles to faradism remains undiminished to the end.

## 2. Therapeutical Uses of Electricity.

*a. In Paralysis.*—The treatment of paralysis by means of electricity must be conducted rationally and with discrimination. By means of electricity we may attempt to remove the cause of the paralysis, by influencing the nutrition of the parts where such cause is situated, by acting on the sympathetic nerve-branches supplying the walls of the blood-vessels. Thus it is asserted that the absorption of a clot in the brain may be hastened, and that the nutrition of a damaged brain may be improved, by acting upon the cervical sympathetic nerve. To influence the cervical sympathetic we place one rheophore over the superior cervical ganglion (which may be reached by pressing inwards behind the angle of the jaw), and the other on the back, over the first and second cervical vertebræ. The use of such a proceeding is more than doubtful, and in case of any improvement occurring it would be impossible to know to what such improvement ought to be attributed, since the passage of a current across the upper part of the neck must influence many important nerves besides the sympathetic. On the whole, we think that galvanisation of the sympathetic is not to be advised in the early stages of paralysis. Galvanisation of

the sympathetic has been employed in chronic degenerative changes in the brain or cord, but with very doubtful success.

The localised application of the electric current to the paralysed muscles is of undoubted service, and in employing it we should bear in mind the following rules.

1. As to the object of our treatment, we should remember the words of Sir Thomas Watson, 'That our aim is to preserve the muscular part of the locomotive apparatus in a state of health and readiness, until peradventure that portion of the brain from which volition proceeds having recovered its function, or the road by which its messages travel having been repaired, the influence of the will shall again reach and reanimate the palsied limbs.'
2. Always to employ that form of current to which the muscles most readily respond. Thus if the muscles act readily to faradism, then faradism is to be used. In some cases of peripheral palsy we find that contraction follows only on the application of a very strong galvanic current, very slowly interrupted, and accordingly a slowly interrupted galvanic current must be used. As the case improves we shall find that a weaker current produces similar results, and that the muscle contracts with moderately rapid interruptions. And so will improvement gradually take place in favourable cases till faradic irritability and lastly voluntary power are restored.
3. Always to employ the weakest currents which will cause muscular contraction, and never to run the risk of exhausting a muscle by causing a too prolonged contraction. Each muscle should be taken in turn, and be made to contract two or three times in succession, and having gone over the whole of the paralysed muscles seriatim the process may be repeated. An application of this kind every other day is usually sufficient.
4. If the paralysis to the will remain absolute, and if the contractility of the muscles be perfect, we do no good by persevering with electrical treatment. This condition is often met with in hemiplegia. The patient is absolutely helpless on one side, although the paralysed muscles are in no degree wasted, and their irritability remains normal.
5. If the paralysis to the will remain absolute, and if the irritability of the muscles be diminished, then electricity is useful, in so far as it helps to improve the nutrition of the muscles and restore their normal degree of irritability. The normal degree of irritability and nutrition being restored (the paralysis to the will remaining absolute) electrical treatment may be discontinued.
6. If the irritability to both forms of current has completely disappeared, we are not justified in persevering too long, nor in holding out delusive hopes to the patient. Nevertheless, treatment should not be abandoned without a patient trial. [For the treatment of special forms of paralysis by electricity, the reader is re-

ferred to the appropriate sections of this work.]

3. *In Painful Affections.*—The power of electricity to relieve pain is very great. The relief is usually temporary, but in many cases is permanent. Electricity may act by serving to divert the mind from troubles real or fancied, or its counter-irritating effects may serve the same purpose as other counter-irritants whose power to relieve pain is well recognised. Occasionally electricity will give relief when every known remedy has failed, and in such cases we must suppose that it acts by bringing about some change in the nerves themselves by its specific action on nervous tissue. All three forms of electricity are employed for the relief of pain, but the galvanic current will be found the most generally applicable. Some writers insist that the anode (positive pole) shall be applied to the painful spot. Strong faradisation serves in some cases to give relief. The effects of galvanism should be tried in every case of neuralgia, but it is not capable of relieving all cases, and disappointment is not infrequent. Where muscular movements increase the neuralgic pains, a rhythmical exercise of the affected muscles should be conjoined with the galvanism. Headache of all kinds not infrequently yields to electricity; lumbago, sciatica, and those painful muscular conditions which we call 'rheumatic,' are quickly relieved by it. Tinnitus aurium will sometimes yield to the galvanic current when all other remedies have failed.

γ. *In Spasmodic Diseases.*—In the treatment of spasmodic diseases, electricity is of limited utility. Some forms of tremor are relieved by it. Some aggravated cases of writer's cramp have yielded to it when all other remedies have failed, and a few cases of clonic torticollis have received undoubted benefit by its judicious application. Tonic spasm of internal organs, such as the bowel and bladder, has been relieved by the galvanic current.

δ. *In other Diseases.*—In addition to the treatment of diseases of a purely nervous character, electricity has been employed as a remedial agent in diseases whose origin is not so obviously connected with the nervous system. It has relieved the paroxysms of angina pectoris, and the burning pains which accompany herpes zoster. It has been employed in the treatment of many obstinate skin-diseases by American physicians, and Dr. Cheadle has recorded a case in which the dilated vessels in acne rosacea were made to contract by faradisation. Rheumatic gout is said to have been benefited by a 'central' application of galvanism (one pole to the nucha, and the other to the epigastrium). The flow of urine in diabetes has been stated to be diminished by a similar process; and the symptoms of exophthalmic goitre have (it is said) been diminished by

galvanisation of the cervical sympathetic. There is in fact scarcely a disease, from epilepsy to chilblains, in which it has not been alleged that electricity has been of use. In obstetric medicine, for the arrest of *post-partum* hæmorrhage, faradisation is now one of the recognised means to be employed; and it has been of more doubtful service for the rectification of displacements of the uterus. Ovarian pain and tenderness have been relieved by the galvanic current, and amenorrhœa has often yielded to electricity.

ε. *Galvano-cautery and Galvano-puncture.* The chemical and thermal effects of galvanism are largely employed both in surgery and medicine. Its thermal effect has been used for the heating of cauteries; and cauteries so heated have very obvious advantages over all other forms. The chemical effect of the negative pole has been used as a caustic for the destruction of tissues, and tumours of considerable size have, it is said, been 'dispersed' by this means. The treatment of uterine fibroids, as recommended by Apostoli, by passing a current of 250 or more milliamperes by means of a big clay rheophore applied to the abdomen and a special platinum rheophore inserted in the uterus, is a mode of treatment which is occupying the attention of obstetricians, and is still *sub judice*. The same may be said of similar measures applied to cancerous and other tumours. Galvano-puncture has been used in the treatment of hydatid cysts of the liver, but it is at least doubtful whether simple puncture is not quite as serviceable. Galvano-puncture seemed at one time likely to take a recognised position among the means at our disposal for the treatment of aortic aneurysms. Several cases were recorded in which improvement followed this method of treatment, but the success attending it has not on the whole been encouraging, and it has fallen almost entirely into disuse. G. V. POORE.

**ELEPHANTIASIS ARABUM** (ἐλέφας, an elephant).—SYNON.: Fr. *Eléphantiasis*; Ger. *Elephantiasis*.

DEFINITION.—A non-contagious disease, characterised by recurrence of febrile paroxysms, attended by inflammation and progressive hypertrophy of the integument and areolar tissue, chiefly of the extremities and genital organs; and occasionally by swelling of the lymphatic glands, enlargement and dilatation of the lymphatics, and in some cases by the co-existence of chyluria, and the presence in the blood of certain nematode hæmatozoa; together with various symptoms indicative of a morbid or depraved state of nutrition.

ÆTIOLOGY.—Elephantiasis is endemic in India, the Malayan peninsula, China, Egypt, Arabia, the West Indies, and parts of America, chiefly in localities within the influence of the sea air; and it occurs sporadically all

over the globe, excepting, perhaps, in the extreme north and south. Certain conditions of soil and climate, such as humidity, heat, malarious influences, and proximity to the sea-coast, seem to be concerned in producing the disease and influencing its development. Removal from the endemic area frequently checks the disease, whilst return there reproduces it. Elephantiasis affects both sexes, and persons of all ages and conditions of life, but more men suffer from it than women. No race is exempt, but it is much more frequent in dark than in fair races. It occurs at all ages, but is most common in adult and middle life, comparatively rarely beginning in young children or in the aged. Elephantiasis is doubtfully hereditary; but Richards found that of 236 persons, 73 per cent. had one or both parents affected. Various causes are assigned for the disease. Air, water, food, and, as it is common near the sea-coast, eating fish have been frequently credited with producing it. Certain forms of vegetation, and the geological formation of the soil, have also been regarded as predisposing and determining causes. Climate and locality, combined with bad living, are doubtless the real predisposing causes; and it is probable that, as the late Dr. T. Lewis suggested, it may be found to be intimately associated with, if not entirely dependent on, the presence in the blood of certain parasites. No race is exempt from the disease, but, whatever may be the explanation, the white suffer less than the dark races. It does occur occasionally, though very rarely, in the pure European in India, but more frequently in those of mixed descent; it will generally be found that where it occurs in persons of apparently European parentage, there is a mixture, however slight, of dark blood.

ANATOMICAL CHARACTERS.—The hypertrophy of elephantiasis in most cases appears to be simply an increase in the natural elements of the part, the blood-vessels and lymphatics sharing in the growth. In other cases the lymphatics and lymph-spaces are most concerned, giving rise to a condition that has been described as *navoid elephantiasis*, in which the appearance is presented of a soft and fluctuating swelling, which when punctured gives issue to a white or pinkish fluid, very closely resembling chyle. The lymphatic glands also share in the enlargement. In other respects the progress of this variety is like that of the ordinary form of the disease.

The *Filaria sanguinis hominis* is sometimes found in great numbers in the blood of persons suffering from elephantiasis; and the mosquito is considered to be the medium by which the hæmatozoon is transmitted to water, and thence to the human body; but its causal relations to the disease are not absolutely established. See FILARIA SANGUINIS HOMINIS.

**SYMPTOMS.**—The ordinary form in which elephantiasis presents itself is hypertrophy of the integument and areolar tissue of some part of the trunk or limbs, and notably of the legs and genital organs. The skin becomes enormously thickened by hypertrophy of all the fibrous elements of its structure, attended by the deposit of a quantity of albuminous fluid in the interstices of the areolar tissue. The papillæ are prominent and much increased in size. The integument is formed into hard masses or folds, with a rugose condition of the surface, not unlike the appearance of an elephant's leg. The feet and toes are sometimes almost hidden, and the scrotum or labia form enormous outgrowths. The scrotum often attains great weight, and may be accompanied by large hydroceles. Scrotal tumours have been removed weighing upwards of 100 lbs.

The onset of elephantiasis is frequently violent and attended with great suffering. There is high fever; intense pain in the lumbar region, groin, spermatic cords, and testes, which become swollen; while acute hydroceles form. These symptoms are often attended with sympathetic vomiting, nausea, and rapid erythematous swelling of the external parts; and, if the extremities be attacked, the swelling may be tense and painful, accompanied by much effusion into the areolar tissue. The surface of the integuments is much inflamed, and sometimes discharges a serous ichor or chyle-like fluid, according to the extent to which the lymphatics are engaged in the particular case. The great tension and swelling of the spermatic cords are apt to dilate the abdominal rings so widely, that after recovery the patient may suffer from hernia.

In some cases of elephantiasis the integuments are also the seat of a dilated and turgid condition of the lymphatic vessels, which during the periods of vascular excitement, when the febrile attacks occur, give way and discharge a chyle-like fluid; in other cases the surface temporarily assumes a herpetic condition, which exudes an acrid and offensive serous fluid. See *CHYLURIA*.

Elephantiasis not infrequently occurs without much or any obvious injury to, or disturbance of the general health during the intervals between the febrile attacks, which in some cases are few and slight. The appetite, spirits, and strength are good, the functions are all normally performed, and the only inconvenience is that due to the size and weight of the outgrowth. On the other hand, it is frequently quite the reverse: the rapidly recurring febrile attacks, pain, exhaustion, suffering, and visceral complications induce a state of cachexia and debility sometimes so serious as to render even surgical interference impracticable. Withal, hepatic and splenic enlargements do not as a rule result from the persistence of the elephantoid fever alone;

though not infrequently, as a more direct result of malarious poisoning, they seriously complicate the evils of the sufferer's condition. Albuminuria, as well as chyluria, is occasionally present.

In some cases, after the outgrowth has attained a certain bulk, it ceases to grow altogether, or increases slowly and insidiously without febrile disturbance, and in such cases the general health remains good. But there is generally a tendency to recurrence of the fever once or twice a month, when the parts affected become tense, hot, painful, and swollen, and often discharge a serous or lymph-like fluid, which may be acrid and offensive. Some tumours, on the other hand, are very slightly, if at all, so affected, and remain perfectly dry. In all cases, however, some growth goes on; and even when, as occasionally happens, fever has ceased to recur, there may be a gradual, but slow and painless, increase of the hypertrophy. The greatest variety and uncertainty obtain in the duration and progress of the growth: sometimes it is very rapid, at other times it is slow, with intermissions of activity and indolence of development. The disease elsewhere than in the genitals, unless it be accompanied by exhaustion and debility, causes no failure in the generative powers in either sex. Women may have a tendency to miscarry when suffering from elephantiasis.

**COURSE, DURATION, AND TERMINATIONS.**—According to Richards, the average duration of the disease, as deduced from the observation of 636 cases, was 11½ years; and he notes that the earliest age was nine years, whilst the latest at which he observed it was eighty years. It appears from this that the disease has little influence in shortening life.

**PATHOLOGY.**—The outgrowths in elephantiasis are the local expressions of a constitutional disease, and are not to be regarded merely from their local point of interest. They are the result of certain climatic influences whose exact nature is not at present determined; though, considering the geographical range of the area where the disease is endemic, it seems probable that, whatever other cause may be at work, the so-called malarious influences play some part in its production.

The researches of the late Dr. T. Lewis into the pathology of chyluria in India, and his discovery of certain hæmatozoa in the blood of those affected with that disease, coupled with the fact that the subjects of chyluria and hæmatozoa are also frequently, if not always, affected by elephantiasis with its febrile paroxysms, hypertrophied integument, and lymphatic disturbance, are very suggestive of a community of origin of these morbid conditions. Dr. P. Manson's investigations into the connexion of the mosquito with the transmission of the filaria are also most important and interesting.

**TREATMENT.**—Little has yet been done by constitutional treatment in cases of elephantiasis. Remedies useful during the febrile paroxysms have little power in preventing recurrence of, or in checking the disease. Iodine, combined with quinine, arsenic, and iron, has been found useful to a certain extent. During the febrile state salines, diaphoretics, and such remedies as are needed during the pyrexial state of malarious fevers, are indicated. Opium may be necessary to relieve the intense pain which often accompanies the onset of the stage of excitement. When the febrile stage has passed, quinine is useful, which, if anæmia exist, should be combined with iron. The local application of iodine, in such forms as the iodide of lead or biniodide of mercury ointment, has been thought useful; but as this is generally combined with pressure in the recumbent posture, the benefit is probably due to the latter. Such measures, along with improved hygienic conditions, may no doubt control the progress of the disease and relieve suffering. No remedy, however, is so potent as change of climate, by removal from the endemic site of the disease. This, if effected in the earliest stages, may completely arrest the disease, and perhaps even disperse any incipient structural change. This has been observed in the rare cases in which elephantiasis occurs in Europeans, who, on returning to Europe, have after a time lost the disease, and almost, or entirely, any hypertrophic changes that may have occurred. Natives of India improve if they leave the endemic area during the early stages, and go and reside in other and drier localities. However, when the hypertrophy is advanced, the paroxysms of fever are still liable to recur, even when the climate is changed, though with less violence.

*Surgical treatment*, when the hypertrophy is advanced, is often most successful in relieving the sufferer, not only of the local trouble, but also of the fever. Tumours of the genital organs, sometimes of enormous size, are now removed with complete success and comparatively small mortality. Before commencing the operation, especially in the case of a large scrotal tumour, it is well to drain it of blood by placing the patient on his back, and elevating the tumour on the abdomen for an hour or so, during which time pressure by a bandage (a modification of Esmarch's) may be tried, and cold (ice) may be applied. During the operation, the application of a whipcord ligature drawn tightly round the neck of the tumour also prevents loss of blood.

The removal of a scrotal tumour is effected by incisions along the course of the cords and the dorsum penis. The cords, testicles, and penis are turned out by a few touches of the knife, and then reflected and held up on the abdomen, while the mass of the tumour is

rapidly swept away by a few bold incisions in the perinæum. The numerous venous and arterial bleeding points should then be arrested, and the wound dressed with antiseptic applications. No attempt should be made to preserve flaps of integument either for the penis or testes. It is unnecessary, and almost certain to be followed by recurrence of the disease. The process of cicatrisation goes on rapidly, and in from two to four months all is closed in by cicatricial tissue, which gradually perfects itself, and has no liability to become the seat of a return of the disease.

JOSEPH FAYRER.

**ELSTER, in Saxony.**—Alkaline sulphated waters. See MINERAL WATERS.

**EMACIATION** (*emacio*, I make lean). Wasting or loss of flesh. The term is applied both to the process of wasting and to the condition that results therefrom. See ATROPHY; and ATROPHY, GENERAL.

**EMBOLISM** (*ἐμβολισμ*, a plug).—SYNON.: Fr. *Embolie*; Ger. *Embolie*.

**DEFINITION.**—The arrest in the arteries or capillaries of some solid body, which has been carried along in the course of the circulation.

**PATHOLOGY.**—Emboli usually consist of portions of fibrin derived from thrombi of the veins or heart, or of vegetations detached from the cardiac valves. They may, however, be formed by fragments of tumours which have grown into the blood-vessels, or of other foreign bodies, including fat, which have obtained entrance into the circulation.

The effects of embolism may be divided into two classes: First, those which are caused by the arrest of the circulation; and secondly, those which are due to any specially irritating properties of the embolus.

1. The embolus may, first, be supposed to consist of some *indifferent* substance not possessing any irritating qualities. The effects which may then be caused by arterial embolism are mainly these: First, a transient anæmia of the territory supplied by the blocked artery. This may pass away without leaving any permanent consequences. Secondly, necrosis of this territory. This may be either sudden, in the form of gangrene; or more gradual, in the form of softening or withering. Thirdly, the formation of a *hæmorrhagic infarction*, that is, congestion of the territory, followed by extravasation of blood into the tissues, and so the formation of a firm, solid patch of a dark red colour, usually of a wedge shape, with the apex towards the embolus, and the base towards the periphery. In very soft organs, as the brain, the extravasation may break down the tissue and cause the ordinary phenomena of an apoplectic clot. These hæmorrhagic infarctions undergo various subsequent changes. Usually a process of degeneration sets in;

the blood-pigment passes through its familiar transformations; the patch changes from dark red to tawny and yellow, undergoes molecular disintegration, shrinks away, and ultimately leaves a depressed fibrous patch, in which the remains of the altered blood, crystals of hæmatoidin, &c., may often be recognised. Sometimes the patch softens down into a puriform fluid, which may become surrounded by a fibrous capsule, and ultimately dry up, or even calcify. When recent, these patches are usually surrounded by a halo of congested vessels.

The cause which determines these different results of arterial embolism is, in the main, the anatomical arrangement of the blood-vessels. Supposing the embolus to be lodged in an artery which gives off anastomotic branches between the seat of the embolus and the final capillary distribution, the effect in most cases will be transient anæmia, the collateral channels will enlarge, and the circulation will be again restored. A thrombus will form on the embolus and will extend back to the next arterial branch, and the changes described in the article on thrombosis will take place in it (*see* THROMBOSIS). If the blocked artery be of large size, and supply important organs, the symptoms of temporary arrest of function of the part supplied by the artery will follow, as transient paralysis, dyspnœa, coldness of the extremities, &c., according to the artery affected. Should, however, the artery be small, and not supply important organs, no symptoms whatever will be caused; and this is the case in the majority of embolisms. Supposing, however, the artery is what Cohnheim calls a *terminal* artery—*i.e.* one which gives off no anastomotic branches between the embolus and the final capillary distribution, and that the capillary anastomosis with other arterial territories is insufficient to supply a collateral circulation, and that the presence of valves prevents the reflux of blood into the territory from the veins, it is manifest that the embolism must completely cut off the blood-supply, and consequently, in some form or other, cause necrosis of the territory.

The network of anastomosing channels is, however, so close in most parts of the body that, in order to produce this effect, it is necessary either that the main artery of the part be obstructed, or else that there be multiple embolisms blocking up at the same time several arterial branches, and so stopping the channels of collateral circulation.

The mode in which the hæmorrhagic infarction is produced is still a subject of dispute. According to Cohnheim, whose views until recently were generally accepted, the hæmorrhage is due to a reflux from the veins into the territory supplied by the blocked artery. This first causes congestion, and then extravasation, in consequence of impairment

of nutrition of the walls of the blood-vessels for the integrity of which the circulation of the blood is essential. According, then, to Cohnheim, in order to produce the phenomena of the hæmorrhagic infarction it is necessary that the artery be a terminal one—*i.e.* one which gives off no anastomotic branches for some distance before its final capillary distribution, and that the veins be not furnished with valves. These conditions are met with in the spleen, the kidney, the brain, certain branches of the pulmonary artery supplying surface lobules, and the central artery of the retina; and on these grounds he accounts for the frequent occurrence of hæmorrhagic infarctions in these organs, though there is no reason to suppose that, with the exception of the lungs, embolisms are more frequent in them than in other parts.

The more recent researches of Dr. M. Litten, *Zeitschrift für klinische Medicin*, vol. i. p. 189, render, however, these views no longer tenable. He shows by experiments on the kidney, spleen, lung, &c., that if the blocked artery be a strictly terminal one—*i.e.* one whose area of distribution has no other arterial supply, the phenomena of the hæmorrhagic infarction do not occur, even though the vein have no valves; and under other circumstances that the infarction takes place although the vein has been ligatured; hence the cause of the infarction cannot be venous reflux. Thus, if both the renal artery and vein be ligatured, infarction of the kidney takes place, the kidney receiving a sufficient collateral supply of blood from other sources; but if the capsule be first stripped off, and the kidney left attached only by the renal artery and vein, no infarction takes place, though the vein is left pervious. Similar results were obtained in other organs. Hence it would seem evident that the congestion and infarction following embolism are produced by an afflux of arterial blood into the territory from collateral channels. Should these be numerous, and should small arteries open directly into the anemic territory, the circulation will soon be restored, and no infarction will take place; should, however, the communication be imperfect, and chiefly by means of capillaries, a congestion of the territory, leading to diapedesis, and infarction will result, the *vis a tergo* being insufficient to propel the blood onwards into the veins.

Litten has also shown that the vessels in which the circulation has been arrested retain their integrity much longer than was supposed by Cohnheim; and that in the kidney, long before the vessels suffer, necrosis of the epithelium takes place, the nuclei of the cells disappear, their protoplasm coagulates, and they become converted into swollen hyaline masses (*coagulation necrosis*), which have a remarkable tendency to calcification. Hence the wedge-shaped white embolisms often seen

in the kidney are not produced by decolorisation of hæmorrhagic infarctions, but are simply the result of the necrosis of the epithelium; and the halo of injection which is often seen to surround them is due to inflammatory congestion caused by the presence of the necrosed patch.

This explanation of the mode of production of the hæmorrhagic infarction is more closely in accord with the view originally propounded by Virchow, who regarded the hæmorrhage as due to collateral fluxion.

2. We have now to consider the effects of emboli which possess irritating or *poisonous* qualities, such as those derived from the puriform softening of venous thrombi in cases of septic inflammation. The mechanical effects will be the same as those of the previous class; but, in addition, these emboli set up a suppurative inflammation in their vicinity, quite independent of any obstruction of the circulation. Hence it is that we meet with pyæmic abscesses, as the result of infecting emboli, in all parts of the body, while the effects of obstructed circulation are, for the most part, confined to certain organs. Thus the liver is very frequently the seat of embolic abscesses, while hæmorrhagic infarctions do not occur there. In the lung, where in parts terminal arteries are found but for the most part there is free anastomosis, the two processes are often seen side by side. The different effects of these two classes of embolism are very manifest in the capillaries. Simple emboli, of such small size as to become first arrested in the capillaries, either cause no permanent change at all, or, at most, produce a punctiform hæmorrhage. Infecting emboli, on the other hand, give rise to the miliary abscesses so often seen in pyæmia.

W. CAYLEY.

**EMESIS** (ἐμέω, I vomit).—A synonym for vomiting. See VOMITING.

**EMETICS** (ἐμέω, I vomit).—SYNON.: Fr. *Émétiques*; Ger. *Brechmittel*.

**DEFINITION.**—Agents that produce vomiting.

**ENUMERATION.**—Copious draughts of Luke-warm Water, Mustard, Sulphate of Zinc, Sulphate of Copper, Carbonate of Ammonium, Common Salt, Alum, Chamomile, Tartar Emetic, Ipecacuanha, and Apomorphine.

**ACTION.**—The act of vomiting consists in the simultaneous spasmodic contraction of the diaphragm and abdominal muscles, and relaxation of the cardiac orifice of the stomach, so that its contents are expelled. When the diaphragm and abdominal muscles contract, but the cardiac orifice remains closed, so that the contents of the stomach cannot escape, the expulsive efforts are termed *retching*. The nervous centre which regulates these movements is situated in the medulla oblongata; and it may be excited either directly by the action upon it of drugs

carried to it by the blood, or reflexly by irritation of various nerves. The drugs that act directly upon it have the same action, whether they are introduced immediately into the circulation or absorbed by the stomach. They may thus produce vomiting and evacuation of the stomach without being taken into the stomach at all, and on this account they are termed *indirect* emetics, although they act directly upon the vomiting centre. Such are ipecacuanha, apomorphine, and tartar emetic. Similarly the drugs that excite it reflexly are still termed *direct* emetics, because they are applied directly to the stomach. Such are the sulphates of zinc, copper, and aluminium; carbonate of ammonium; salt; mustard; and chamomile, which irritate the nerves of the stomach. Ticking the fauces with a feather, or with the finger, also excites reflex vomiting, and may be adopted either alone, or in order to aid the action of other emetics. The terms *direct* and *indirect*, therefore, as applied to emetics, relate to the stomach, and not to the centre for vomiting.

Direct emetics, as they stimulate the nerves of the stomach only, have little action except that of simply exciting vomiting. The indirect emetics, which excite vomiting by their action on the medulla oblongata, act also on other parts of the nervous system, and cause secretion of saliva, secretion of mucus from the œsophagus, stomach, and bronchial tubes, and perspiration. They also cause much nausea, depression of the circulation, and loss of nervous and muscular power. Further, the vomiting they induce is more continuous and violent, and often expels the contents of the gall-bladder, causing part of the bile to flow into the stomach, and be thus evacuated.

**USES.**—Emetics are employed to remove the contents of the stomach under various circumstances. Firstly, when the food is causing irritation, and not undergoing proper digestion, as, for example, in dyspepsia or sick-headache; and in such cases large draughts of lukewarm water, of mustard and water, or of an infusion of chamomile are usually found beneficial. Secondly, in cases of poisoning; and here mustard, sulphate of zinc, and sulphate of copper are best, as they empty the stomach most quickly and effectually. Thirdly, to cause the expulsion of bile from the gall-bladder, or remove bile from the body, in biliousness, fevers, and ague. When the bile-duct is stopped by a small gall-stone, the pressure exerted on the gall-bladder during vomiting has been known to cause the expulsion of the calculus. In biliousness, excess of bile is more readily removed by vomiting than by purging, as there is no opportunity for the bile to be absorbed on its way from the gall-bladder to the mouth, whereas it may undergo absorption on its passage through the intestines. It is supposed

by some that various poisons circulate occasionally in the bile, such as the malarious poison which occasions ague, and possibly certain septic poisons which give rise to fevers. The advantage of emetics in ague is undoubted, as it can certainly sometimes be cured by them without quinine, and the action of quinine is always aided by their use. They have also been recommended in the early stages of continued fevers. In such cases tartar emetic or ipecacuanha is most serviceable. Fourthly, to cause expulsion from the air-passages of false membrane in croup or diphtheria, or of secretions in bronchitis and phthisis. For these purposes ipecacuanha is the emetic most frequently chosen; but if it do not act rapidly in croup, sulphate of zinc or sulphate of copper may be employed, or a teaspoonful of powdered alum and honey. In cases of either croup or bronchitis, where there is great depression of the circulation, carbonate of ammonium may be used with advantage, as it not only causes vomiting, but at the same time stimulates the circulation.

T. LAUDER BRUNTON.

**EMMENAGOGUES** (*ἔμμηνα*, the menses; and *ἄγω*, I move or expel).

**DEFINITION.**—Emmenagogues are remedial agents which stimulate or restore the normal menstrual function of the uterus, when it is irregular or absent.

**ENUMERATION.**—Emmenagogues may be either *indirect*, as Iron, Strychnine, and other tonics, Warm Hip-baths, Leeches, Mustard, Aloetic purgatives, &c.; or *direct*, as Rue, Borax, Savin, Myrrh, Cantharides, Guaiacum, Apiol, Quinine, Digitalis, and Ergot—most of which, when given in larger doses, produce abortion, and are called *ecbolics*. The most efficient means, however, of obtaining this last-named action are those of a mechanical nature, so well known to obstetricians, and directed either to the actual rupture of the membranes, or to their separation from the cervix.

**ACTION.**—The *indirect* emmenagogues act by improving the quality of the blood, giving tone to the nervous system, or irritating adjacent parts or organs, from which a stimulating influence is conveyed by reflex action to the womb.

The *direct* drugs in moderate doses gently stimulate the uterus, thus promoting the menstrual flow, or even checking it when in excess; but when further pushed they may cause powerful contraction of the unstriped muscular fibre, of which its walls are mainly composed. Ergot is believed to act either directly on the muscular tissues themselves, or through the intermediate intervention of some central or peripheral nervous influence.

**USES.**—Checked or retarded menstruation frequently results from anæmia or general

debility, and the indirect emmenagogues will under these circumstances usually effect a cure. If, however, the case prove more obstinate, one or more of the direct emmenagogues will often restore the suspended function. Should even this do no good, some mechanical impediment probably exists, or some altered physical condition of the womb which mere drugs cannot rectify.

ROBERT FARQUHARSON.

**EMOLLIENTS** (*emollio*, I soften).—**SYNON.**: Fr. *Émollients*; Ger. *Erweichende Mittel*.

**DEFINITION.**—Substances that soften and relax the parts to which they are applied.

**ENUMERATION.**—The principal emollient applications are:—Warm water, Steam, Poultices made of substances which retain heat and moisture, for example, linseed-meal, bread, bran, flour, oatmeal, and figs; Fatty substances, as linseed, olive, almond, and neat's-foot oil, lard, and suet; Spermaceti, Wax; Soap Liniment and other liniments; Glycerine; Paraffines; and Lanolin. To these may be added such substances as do not properly relax the tissues, but protect the surface from irritation, such as White of Egg, Gelatine, Isinglass, Collodion, and Cotton-wool.

**ACTION.**—Emollients relieve the tension and pain of inflamed parts by their action both upon the blood-vessels and upon the tissues themselves. They cause all the contractile tissues to relax and dilate, and thus, lessening pressure upon the nerves of the part, they relieve pain. They soften superficial parts by supplying them with either fat or moisture, and by increasing the supply of blood. In this way they prevent the skin from cracking after exposure to cold. When the cuticle is lost they form a covering, under which the skin may heal; and they prevent the injurious consequences of friction from without.

**USES.**—Fatty emollients are used to prevent the skin or mucous membranes from cracking; to prevent irritation or ulceration between parts constantly in contact, as on the limbs of children near the joints; to prevent bed-sores; to aid the healing of blisters; or in skin-diseases, such as eczema. They are also used, especially in the form of linimentum calcis, as applications in burns and scalds, for which purpose such substances as cotton-wool are likewise frequently employed. Mucilaginous substances are useful, when swallowed, to relieve pain and irritation in the throat, and to lessen irritable cough; and such substances as figs are employed to protect the intestines from injury by hard and pointed substances which have been swallowed. Warmth and moisture are applied in the form of poultices to the surface in pustules, boils, carbuncles, and deep-seated inflammation of the limbs, and

in inflammation of the internal organs (*see* POU LTICE). In the form of vapour they are useful in inflammation of the air-passages. *See* INHALATIONS, Therapeutic Uses of.

T. LAUDER BRUNTON.

**EMPHYSEMA OF LUNGS.**—*See* LUNGS, Emphysema of.

**EMPHYSEMA, SUBCUTANEOUS** (*év, in;* and *φύσα, wind*).—*SYNON.*: Fr. *Emphyseme*; Ger. *Emphysem*; *Windgeschwulst*.

**DESCRIPTION.**—Subcutaneous emphysema is the distension of the spaces of the areolar tissue with air or any other gas. There is thus produced a swelling, in slight cases affecting a very limited area, in extreme cases extending to the subcutaneous tissue of the whole body. Unless the tension is great, the swelling is slightly lobulated; it is elastic, and although the finger sinks readily into it, no lasting impression is left. When the area affected is small, the gas can be driven in any direction by the pressure of the hand. On palpation a peculiar fine crepitation is felt, which is absolutely diagnostic. On percussion there is superficial resonance, the note resembling that obtainable from a bladder loosely filled with air. Unless the gas causing the emphysema is the product of decomposition of gangrenous tissues, as in spreading gangrene, there is no redness of the skin. The swelling usually forms rapidly, and may extend in a few minutes over the greater part of the body. In such cases it is most marked where the subcutaneous tissue is lax. In the face the features are obliterated, and the eyes closed by the swelling of the lids. The scrotum and penis become enormously distended.

**ÆTIOLGY AND PATHOLOGY.**—Wound of the lung from a broken rib or from a stab is the most common cause of subcutaneous emphysema. If from a stab, it can only occur when the opening in the pleura and that in the skin no longer correspond with each other, in consequence of an alteration in the position of the patient, or when the wound has been artificially closed. When from wound of the lung, it may occur with or without pneumothorax. Nothing is more common than to find a limited emphysematous swelling round a fractured rib, without any signs of air in the pleural cavity. This arises either from adhesions existing between the parietal and visceral layers, or from the escape of air being very limited. In severe cases with pneumothorax the mechanism of the production of emphysema is as follows: A wound in the lung always allows air to pass from it readily, but from the way in which the soft pulmonary tissue falls together, no amount of force can drive air through the wound in the opposite direction. It thus acts

somewhat like the valve of an air-pump. The first effect of the escape of air into the pleural cavity is to cause collapse of the lung. As the chest expands with each inspiration air rushes from the wound in the lung into the pleural cavity; as it contracts in expiration, the air, being unable to pass back by the wound, is driven through the opening in the parietal pleura into the subcutaneous cellular tissue. In such cases there is gradually increasing dyspnoea, with great distension of the subcutaneous cellular tissue; and unless relief is given, the patient dies asphyxiated.

Emphysema occasionally occurs from rupture of some of the air-vesicles during a violent expiratory effort. Dr. Walshe states that this accident has happened from 'the efforts in parturition, defecation, raising weights, coitus, violent coughing, paroxysms of rage, excessive laughter, and hysterical convulsions.' The air usually escapes first into the cellular tissue between the lobules of the lung, giving rise to the condition known as *interlobular emphysema*. It then finds its way into the mediastinum, and thence to the root of the neck. Interlobular emphysema gives rise to intense dyspnoea, and has been known to cause sudden death. Emphysema has also been seen as a consequence of ulceration of the trachea, and in a few very rare cases as the result of ulceration proceeding from a cavity in the lung through the adherent pleura and intercostal muscles to the subcutaneous cellular tissue.

Localised emphysema of the face is a symptom of fractures implicating the antrum.

Emphysema of the flanks is an occasional symptom of rupture of the third part of the duodenum, behind the peritoneum, and of perforation of the cæcum at its posterior part.

Emphysema of the perinæum and scrotum may arise from a wound of the bowel in the administration of an enema.

In non-penetrating wounds of the thorax and abdomen, a small quantity of air may find its way into the areolar tissue in the immediate neighbourhood, in consequence of the movements of respiration. In compound fractures emphysema is often found extending some distance above and below the wound, if the patient has been carried some distance and the injured limb much shaken.

Emphysema from the gases produced by decomposition is only seen in cases of rapidly spreading moist gangrene—*emphysematous gangrene*.

**PROGRESS AND TERMINATIONS.**—The effects of emphysema differ with the source of the gas. When the air comes from a superficial wound of the lung, it has no tendency to cause decomposition of the effused blood with which it may come into contact. Thus, in surgical cases, no evil consequences result from emphysema around a simple fracture of

a rib. This is explained by the fact that the gas in the air-vesicles is absolutely free from solid particles of any kind, including micro-organisms, as shown by Tyndall's experiment, in which the residual air, in forced expiration, makes a gap in the beam of an electric light when breathed across it. On the other hand, air admitted from without, as in a compound fracture, tends to favour the decomposition of the effused blood, and renders treatment by occlusion or by antiseptic dressing difficult and uncertain. In ordinary cases the effused air is rapidly absorbed without causing inconvenience of any kind. If the amount of air in the tissues be very great, and the case be complicated by pneumothorax, fatal dyspnoea may occur, unless relieved by treatment.

**TREATMENT.**—The swelling itself requires usually no treatment, the gas being absorbed without difficulty. If it is complicated by pneumothorax, or if the dyspnoea be such as to threaten death, the wound, if one exists, must be opened up; or, if there is none, a free opening must be made into the pleural cavity. If the swelling be such as seriously to inconvenience the patient, a few punctures may be made with a triangular needle. Emphysema from intestinal flatus is always limited, and requires no treatment beyond that applicable to the cause of the escape of gas.

MARCUS BECK.

**EMPIRICAL** (ἐν, by; and *πέιρα*, experience).—This term is applied to treatment founded on experience, as contrasted with *rational*, which is founded on scientific reasoning. See DISEASE, Treatment of.

**EMPROSTHOTONOS** (ἐμπροσθεν, forwards; and *τείνω*, I stretch).—A bending or drawing forwards of the body, due to tonic contraction of the muscles, observed in some cases of tetanic convulsions. See TETANUS.

**EMPYEMA** (ἐν, in; and *πύον*, pus).—Strictly speaking, this term signifies a collection of pus within the cavity of the pleura, but it is often conventionally used to denote any inflammatory effusion in that situation which has assumed a chronic character. See PLEURA, Diseases of.

**EMS, in Germany.**—Thermal muriated alkaline waters. See MINERAL WATERS.

**ENCEPHALITIS** (ἐγκέφαλος, the brain). Inflammation of the brain and its membranes; or, more properly, inflammation of the brain-substance itself. See BRAIN, Inflammation of.

**ENCEPHALOCELE** (ἐγκέφαλος, the brain; and *κύλη*, a tumour).—A hernial protrusion of a portion of the brain-substance through an opening in the skull, which may be either congenital or the result of accident, of surgical operation, or of disease. See BRAIN, Malformations of.

**ENCEPHALOID** (ἐγκέφαλος, the brain). A form of cancer, so named on account of its obvious resemblance to brain-tissue. See CANCER.

**ENCHONDROMA** (ἐν, in; and *χόνδρος*, cartilage).—A new-growth consisting of cartilaginous tissue. See TUMOURS.

**ENCYSTED** (ἐν, in; and *κύστις*, a bladder).—Contained within a cyst. A term applied to new-growths or collections of fluid thus enclosed, or limited by adhesions.

**ENDARTERITIS** (ἐνδον, within; and *ἀρτηρία*, an artery).—Inflammation of the internal coat of an artery. The disease is generally chronic or subacute, rarely acute. Two special forms of endarteritis have been described—namely, *endarteritis deformans*, or atheromatous disease (see ARTERIES, Diseases of; and ATHEROMA); and *sypilitic endarteritis*, which frequently affects the vessels of the brain. See SYPHILIS; and BRAIN, Vessels of, Diseases of.

**E endemic** (ἐν, in; and *δῆμος*, a people). This term is applied to diseases that prevail in particular localities or districts, and which are due to special ætiological conditions existing there. See DISEASE, Causes of.

**ENDERMIC MEDICATION** (ἐν, in; and *δέρμα*, the skin).—Fr. *La Méthode Endermique*; Ger. *Intracutane Arzneiapplication*.

**DEFINITION.**—The method of using remedies either by rubbing them into the skin or by sprinkling them on a surface which has been previously denuded of its epidermis.

Endermic medication was strongly advocated and practised by Lembert, Valleix, Trousseau, Anthony Todd Thomson, and others, but it has almost, if not entirely, given place to hypodermic injection, (1) because the endermic method is the more painful; (2) because it is slower in action—thus, Trousseau found that twelve minutes were required for one-sixth of a grain of morphine endermically administered to take effect, whereas, hypodermically, the action of the same dose is observed in less than one minute, in fact, almost immediately; (3) because in many parts of the body endermic medication is totally inapplicable.

**METHOD AND USES.**—The epidermis should first be detached either by some ordinary vesicant, such as cantharides plaster, or by the application of a cautery. The drug to be used is then laid on the raw surface in the form of powder rubbed up with starch, sugar, sulphate of sodium, or some other unirritating substance, paste made with water or thin mucilage, or ointment. In this way, neuralgia, sciatica, and other painful affections may be treated with the morphine salts in doses of

one-sixth of a grain and upwards; local paralyses and other nerve-troubles with strychnine in doses of one-fiftieth of a grain and upwards; and many other alkaloids may be used in the same way in various diseases.

A. S. CURRIE.

**ENDOCARDITIS** (*ἔνδον*, within; and *καρδιά*, the heart).—Inflammation of the lining membrane of the heart. See HEART, Inflammation of.

**ENDOGASTRITIS** (*ἔνδον*, within; and *γαστήρ*, the stomach).—Inflammation of the mucous membrane of the stomach. See STOMACH, Diseases of.

**ENDOMETRITIS** (*ἔνδον*, within; and *μήτηρ*, the womb).—Inflammation of the lining membrane of the uterus. See WOMB, Diseases of.

**ENDOPERICARDITIS**.—Inflammation of the endocardium and pericardium together. See HEART, Inflammation of; and PERICARDIUM, Diseases of.

**ENDOSCOPY** (*ἔνδον*, within; and *σκοπέω*, I look).—DEFINITION.—The inspection of internal surfaces. Taken inclusively, this definition comprises ophthalmoscopy, otoscopy, laryngoscopy, rhinoscopy, urethroscopy, cystoscopy, and inspection of the gullet, stomach, and rectum. But in ordinary parlance, the word signifies inspection of the urethra only; special terms being used when inspection of other parts is meant.

INTRODUCTION.—An internal surface may be illuminated by letting light, such as daylight, fall through a straight tube (*direct illumination*); or by rays collected and thrown by mirrors placed conveniently through a tube on to the surface to be examined, and reflected directly to the eye of the observer, or through lenses set at suitable angles (*reflected illumination*); or, lastly, by light generated within the body and reflected to the eye of the observer (*internal illumination*). This last method, in which the electric lamp was placed at the inner end of the tube, and its light thrown directly on to the exposed part, has been abandoned for examining the urethra, on account of the difficulty in shielding that canal from the heat of the glow-lamp; though it is still the mode of illuminating the bladder or other cavities, the walls of which are not in contact with the instrument.

The present mode of examining the urethra and other surfaces is the outcome of the work of many experimenters, the earliest of whom was probably Bozzini (1807), followed by Segalas in 1826. In 1840-46, Avery of London devised the tubes which are still in general use. But the methods of illumination now employed, by gas or electricity, are far superior to those obtainable in Avery's time. Desormeaux improved Avery's

method of illumination, and this was further bettered by Cruise. Still, the apparatus remained too cumbrous for easy use until Grünfeld simplified the application of illumination between 1875 and 1880, and Leiter between 1880 and 1887 perfected the electric method by reflecting light from a glow-lamp along the tubes on to the exposed surfaces. About this time Grünfeld, Dittel, and others, employing these more manageable instruments, were able to describe methodically the appearances of the normal urethra in its several parts, and also many of the changes caused by disease.

APPARATUS.—The apparatus may be arranged in three varieties: in Desormeaux' and Leiter's the lamp and tube are fixed together; in a second, the light is distinct, but the reflectors and lenses are fixed to the tube; in a third, the light and reflector, a concave mirror fastened to the forehead or held in the hand, are both separate from the tube.

The best apparatus is that of Leiter, where the light of an Edison's glow-lamp of 0.5 to 1 ampère is affixed below the outer end of the tube, and its rays reflected through the tube on to the exposed portion of the urethra and back again to the eye of the observer. It comprises a set of straight silver tubes five inches long and of internal diameter ranging between 18 and 26 millimètres. Their inner ends are cut off either transversely or obliquely, and the outer ends have a wide mouth and flange, on to which the funnel and mirror may be fitted. Each tube has a vulcanite plug with a smooth or rounded end that projects beyond the tube, to facilitate its passage along the urethra. The interior is blackened to prevent confusion by reflection from its sides. Dr. William K. Otis, of New York, has contrived curved tubes which can be passed along the urethra to the posterior portion with ease; but curved tubes require a reflector at the end, and the one supplied with the tubes is too clumsy to be of much service. Grünfeld has improved the windowed tubes of Avery—that is, tubes with some part of the side or the end closed by a piece of glass—and for some purposes these windowed tubes are more useful than the open ones. In addition to tubes, lamps, and mirrors, small mops of cotton wool are needed to wipe the mucus, pus, and blood from the displayed surfaces, so that the cleaned and dried membrane may be viewed. For the treatment of the various affections, additional armamenta are strong astringent or caustic solutions, hæmostatics, such as hamamelis, iron, or tannin, and pencils of cupric sulphate or of lunar caustic. To them may be added small electric cauteries, probes, forceps, snares, knives, scissors, curettes, and tubes for insufflation of astringent powders. Grünfeld has also contrived to place outside his endoscopic tube another fine one, through which he can

inject drops of astringent solutions on to inflamed patches.

Batteries of various make are used: that for illumination should have a power of eight to twelve volts; that for cautery, of twenty to thirty volts. Carbolic oil (1 in 16) or glycerine for lubricating the tubes is also needful.

APPLICATION.—When inspecting the urethra it is convenient to divide the canal into two portions—that before the triangular ligament being the anterior, that behind it the posterior. Not infrequently the inflammation set up by gonorrhœa is confined to the anterior portion; while, on the other hand, in gleet of long standing the posterior part may be alone affected, when the anterior section is quite clear of disease.

In the anterior portion are the meatus and the fossa navicularis, the spongy and bulbous parts, into which, with a forward direction, open the ducts of mucous glands and those of Cowper's glands. In the posterior are the verumontanum, the sinus pularis, and the openings of the spermatic ducts and of those of the prostate itself.

Before examining the urethra, it is advisable to pass along the canal a sound or bougie slightly larger than the inspecting tube, in order to be sure that no constriction exists which would prevent the passage of the tube, and also to familiarise the urethra with the presence of instruments. It is well also to refrain from introducing the tube into the posterior portion until that part has become accustomed to instruments by the repeated passage of bougies or sounds. The calibre of the tube should not be large enough to stretch the urethra; if it is, the walls are more or less blanched, and the operation is apt to give pain. These drawbacks avoided, the larger the tube the more easy the examination.

*Examination of the anterior portion of the urethra.*—The best position is the half-recumbent one on a high couch, with the knees bent and the legs hanging over the edge. The observer seats himself on a low stool opposite his patient, his eyes about level with the urethra. If the patient be well accustomed to manipulation he may stand upright on a stool against a wall, to bring his pelvis level with the eyes of the observer sitting before him. All being ready, the operator introduces the tube. If the meatus be much contracted, either congenitally or by fibrous bands after inflammation, it is well to divide the tight bands by the touch of a probe-pointed bistoury, after rendering the part insensible with cocaine. A No. 24 or 26 tube, with its plug, is then slowly passed along the passage until the bulb is reached. The plug is then withdrawn, and the area of the urethra exposed at the end of the tube is well mopped and dried, and the light adjusted. If the urethra is healthy and not scarred or

blanched by previous inflammation, the part now seen is of a delicate rose-pink or purplish pink hue, faintly striated with longitudinal lines. The most distinct streaks or furrows are in the floor at each end of the transversely disposed lumen. Faintly marked crimson papillæ are visible in the anterior portion, but are lost near to the navicular fossa. As the tube is slowly withdrawn, the urethra collapses closely behind it evenly on all sides. The lumen or central figure is an even transverse line or slightly oval figure of symmetrical thickness. The orifice of a lacuna Morgagni is indicated by a little dark dot; though these lacunæ may be difficult to distinguish unless sought for in the way to be described presently. When inflamed, ulcerated, or indurated, these openings are easily distinguished. As the tube continues to retreat at about three inches from the meatus, the colour of the mucous membrane grows paler in many persons, to become again dull pink or bluish pink at the sinus navicularis. Here, also, the lumen changes from a transverse position to a vertical one, being oval or triangular in form. In the roof of the urethra it is not difficult to detect in this fossa the mouths of two or three or more ducts of muciparous glands, and the wall of the passage is not so evenly resilient as it is more posteriorly. To find the lacunæ Morgagni in a healthy urethra, it is often necessary to slightly divert the tube from the axis of the urethra to cause the wall to bulge slightly into the open end of the tube. By this, a larger area of the wall is brightly illuminated. If a lacuna be within the illuminated area, it is distinguished by a dark dot due to want of reflexion, because the floor of the lacuna is lower than the general surface. So, also, the position of a lacuna is revealed during slow retraction of the tube by the sudden appearance of a fine furrow or seam in the wall, radiating from the lumen and made more distinct by the greater brilliancy of the light reflected from each side of the furrow. Very shortly the seeming furrow or line disappears as the tube passes from it. The openings of the ducts of Cowper can be more easily found, as they have a more constant position in the urethra. They open into the bulbous portion, where they appear as two dark dots, side by side, soon after the lumen has assumed its transverse linear form.

*Examination of the posterior portion of the urethra.*—In this portion, proceeding from behind forwards, are to be seen the os internum urethræ, the walls of the prostatic portion, the verumontanum, the openings of the prostatic ducts and of the ejaculatory ducts, and the membranous portion of the urethra.

The ordinary straight tube with open end, five inches long and of the calibre suitable for the anterior part of the urethra, is the best to use for this purpose. A special move-

ment is required to pass the tube through the triangular ligament without exciting contraction of the voluntary muscles which encompass that portion of the canal, and which resist the passage of the tube, and thereby hurt the patient.

In introducing the tube, the end rounded by the plug should be pressed gently down the urethra so that it follows the floor, not the roof, until it reaches the triangular ligament. There, while steady pressure is maintained, the hand is lowered deeply between the thighs until the deeper end rises and slips on in spite of the slight resistance always made to its onward passage. When resistance ceases, the tube is pushed backwards for another inch or so, and held steadily while the plug is withdrawn and the light adjusted. If this is done slowly, the pain, should the patient feel any, ceases as soon as the instrument stops moving. It is often well to get the patient to hold the tube himself during the adjustment of the lamp, as he is less likely than the operator to move it inadvertently. But a steady, gentle pressure must be kept on the tube while it is in this region, or it will be ejected by the muscles of the urethra, and will be suddenly thrust forth into the bulbous portion. The part being well wiped and the light thrown on, this portion of the urethra will appear funnel-shaped, and of full rosy hue or pale purple, or yellowish white if there have been inflammation or induration of the prostatic portion at some time before. At the *os internum* the lumen is long, horizontal, and marked at each end by two radiating creases. If now the tube be pushed back a little, it is quickly filled by urine, and a misty yellow light is all that is visible. This accident is easily avoided by practice; but if it happens, the tube must be withdrawn a little, and its outer end depressed that the urine may flow out. This done, the mops dry up the remaining moisture, and the inspection may begin. Besides the conditions already mentioned, in some cases fine longitudinal striæ or creases are seen to radiate pretty uniformly from the centre. These are probably rows of fine papillæ along shallow folds of the membrane. The tube is slowly withdrawn, bit by bit; and, as it recedes, here and there a dot or depression may be noted placed in no definite position on the wall. These dots are orifices of ducts of the prostate. Gradually, while the end of the tube recedes, there rises into view a saddle-shaped eminence like an inverted U, with the top at the highest point of the exposed surface, around which the walls closely conform. This is the *verumontanum*, or *caput gallinaginis*. It has usually a paler colour than the wall, but is never white unless diseased. Presently, when the saddle-shaped eminence is at the highest, two small red fossæ or furrows can often be seen on either side of the central ridge, and

equidistant from it. These are the outlets of the ejaculatory ducts; and usually near them, but more outward, some more dots appear, signifying the orifices of the prostatic ducts. Occasionally, if the tube be kept still or pressed firmly against the floor, a little whitish-grey bead forms on one of the symmetrically placed hollows. This is semen, and, if wiped away and put under the microscope, is seen to contain the characteristic spermatozooids. Not infrequently the mouths of the ejaculatory ducts are not found by this method. They may then be generally detected by pushing the tube back a little and pressing it up against the roof to allow the floor to rise more. If the outer end of the tube be now raised, the light shines more strongly on the *veru*, and as the tube recedes the furrows generally become distinct. When they are clearly seen, a fine whalebone bougie or silver probe may, it is said, sometimes be coaxed into them for a short distance. On still further withdrawing the tube the saddle-shaped eminence widens at its base, the lumen loses the shape of an inverted U, and grows circular or oval, with the long diameter vertical. The walls also rise up into the area before the tube, and have a paler hue, making a broad pinkish ring inside the tube. When this appears, the membranous portion has been reached. If there have been bygone inflammation of the part, the ridge of the *veru*, instead of being like an inverted U, is like an inverted V, and of much lighter colour, even white, from induration and wasting. Small veins, showing as dark sinuous lines, are not uncommonly seen in the prostatic portion, and, but less frequently, in the anterior portion also.

BERKELEY HILL.

**ENEMA** (*ἐνίημι*, I inject).—SYNON.: Lave-ment; Clyster; Fr. *Clystère*; *Lavement*; Ger. *Klystier*.

**DEFINITION.**—An enema is a liquid injected by means of a suitable instrument into or through the rectum.

**INSTRUMENTS.**—Various instruments are used for the administration of enemata: 1. A simple elastic bottle with ivory or gum-elastic pipe, which has superseded the old bladder and pipe. 2. An indiarubber bottle with flexible tube at either end and double action, as in Higginson's instrument. 3. An ordinary piston syringe, worked by the hand, which is either simple, or provided with a double action, so as to supply a continuous stream. 4. A French instrument, known as the *irrigateur*, worked by a spring. 5. The hydraulic enema, which consists of a piece of indiarubber tubing about six feet long, furnished with an ordinary ivory rectum-pipe at the one end, and a metal cone, or a screw nozzle, at the other. The tube, being filled with the injection, has one end placed in the containing reservoir, or is connected

by the screw; while the pipe at the other end is introduced into the bowel. The vessel supplying the injection being placed on an elevation, the liquid gravitates into the bowel, filling the large intestine. When it is desirable to inject a large quantity the patient should lie first on the left side, then on the back, and lastly on the right side, to promote the filling of the intestine. In all cases care should be taken to prevent the injection of air into the bowel, and also to ascertain that the nozzle of the injecting pipe is free in the rectum, not in contact with the sphincter, not thrust against the sacrum or into a hard fecal mass. The process of injection must be carried on slowly, with occasional pauses, otherwise premature contraction of the bowel with expulsion of the enema will result.

**VARIETIES AND USES.**—The chief varieties and uses of enemata are as follows:—

1. **Anthelmintic Enemata.**—To cure thread-worms injections of salt and water, or lime-water, or from two to four drachms of spirit of turpentine diffused by yolk of egg in four ounces of water, are serviceable. The enema of aloes or of asafœtida may also be employed in the small quantity just named.

2. **Antispasmodic Enemata.**—Puerperal convulsions have been relieved by the injection of half a drachm or more of chloral hydrate. Injections of asafœtida or of rue are also given. Injections of warm water with two or three fluid-drachms of sulphuric ether have sometimes relieved spasmodic invagination of the bowels. When the intestine is tympanitic and distended, the enema terebinthinæ, or enema asafœtidæ, will act well as a stimulant and carminative.

3. **Astringent Enemata.**—These are used either to check diarrhœa, to arrest hæmorrhage, or to remedy ulceration and mucous discharges. For the first of these purposes the enema opii is valuable. In cases of hæmorrhage from the bowels, as well as from the womb, injections of ice-cold water are frequently used. Ulceration with mucous discharge is often successfully treated with enemata of nitrate of silver (five grains to one pint of distilled water), of sulphate of zinc or alum (one or two grains to the ounce of water), or of sulphate of copper (one grain to the ounce of water).

4. **Emollient Enemata.**—Demulcents, such as decoctions of starch, linseed, or barley, or pure linseed oil, are at times used with the object of imparting nourishment to the system, and of soothing an irritable mucous membrane.

In dysentery, from four to six pints of warm water, or of milk and water, have been injected as a form of internal fomentation.

5. **Nutrient Enemata.**—In cases of exhaustion, enemata of peptonised milk, beef-tea and eggs beaten up are used. About

four or six ounces should be given at once by means of an indiarubber bottle with an elastic-gum rectum tube. When properly given, they will remain and be absorbed. Should the rectum become irritable, the irritability may be often lessened by adding a few drops of laudanum to each enema. A solution of defibrinated blood, in its recent or dried form, has been recommended as a material for nutrient enemata (see paper by Dr. Sansom on Supplementary Alimentation, *Lancet*, 1881, vol. i. p. 288). The digestion and assimilation of nutrient enemata may be facilitated by the addition of preparations of pancreatin and pepsin. See PEPTONISED FOOD.

Injections of brandy and water, or beef-tea and brandy, have been given with benefit in prostration from uterine hæmorrhage or other causes.

6. **Sedative Enemata.**—These are often employed in painful affections of the rectum and bladder. The enema opii is very useful. In spasm of the bowels and in hernia the enema tabaci has been resorted to, but it must be used with caution, as it may produce faintness and collapse.

7. **Purgative Enemata.**—These are used to overcome constipation. For this purpose—in the case of an adult—from one to two pints of fluid must be slowly pumped into the bowel. When the process is conducted gradually, stopping occasionally and making pressure on the anus if the injection threaten to come away, as much as four or five pints can be got into the bowel. The injection should be retained as long as possible, as thus a complete evacuation is ensured. As a general rule, about a pint of liquid is enough for an adult; for an infant an ounce; for a child of four years, four to six ounces.

**Composition.**—Soap and water, gruel with olive-oil, castor-oil, and sometimes oil alone, may be used. The enema magnesii sulphatis is an efficient purgative. Enema aloes is also valuable.

Glycerine is now used in cases of defective action of the bowels. From one to two drachms are injected by a suitable syringe into the rectum, and in from five to thirty minutes action of the bowels will follow. There is often more or less straining, with expulsion of flatus; and, if the quantity used be too large, violent and spasmodic action may result.

Glycerine suppositories have been added to the Pharmacopœia. Their action is attended with less spasm than is that of the fluid injection. The glycerine acts as a stimulant and irritant to the rectum; for while some amount of burning is felt, a thermometer shows increased temperature in the rectum. Where the accumulation lies high up in the intestine, glycerine injection has little or no effect in causing its expulsion.

In cases of intestinal obstruction the introduction of a large volume of soap and water—with oil, perhaps, added—by means of the gravitation-tube, may be had recourse to with advantage. Obstruction due to impacted feces generally yields to this method of treatment, especially if assisted by judicious massage of the abdomen.

The gradual injection of air by means of a bellows and rubber tube, or a Lund's insufflator, into the large intestine has proved a successful method of treating intussusception of the bowel in young children, especially in cases where the intussusception can be easily felt by the rectum, and where enemata are at once returned. *See* **INTESTINAL OBSTRUCTION**.

The frequent use of very large injections is undesirable, lest undue distension result, with weakness and loss of tonicity in the bowel. The habitual use of injections washes away the mucus designed to lubricate the bowel.

JOHN C. THOROWGOOD.

**ENGADINE, UPPER; in Switzerland.**—A bracing mountain climate. Elevation of valley 5,000 to 6,000 feet. Season, June to September. Some places are also open during the winter. *See* **CLIMATE**, Treatment of Disease by.

**ENGHIEN, in France.**—Sulphur waters. *See* **MINERAL WATERS**.

**ENGLISH CHOLERA.**—A synonym for simple cholera or choleraic diarrhœa. *See* **CHOLERAIC DIARRHŒA**.

**ENGORGEMENT.**—Overloading of the vessels, or of the heart, with blood. A synonym for congestion. *See* **CIRCULATION**, Disorders of.

**ENTERALGIA** (*έντερον*, the intestine; and *άλγος*, pain).—**SYNON.**: Enterodynia; *Neuralgia mesenterica vel mesaraiica*; Colic.

The terms enteralgia and colic—generally regarded as synonymous—include all forms and degrees of paroxysmal intestinal pain in cases where there is no febrile disturbance. Enteralgia, implying more especially the neuralgic nature of the sensori-motor disturbance, is sometimes preferred, as by those who hold that colic proper—of which lead-colic is a typical example, as distinguished from symptomatic colicky pains—is a visceral neuralgia. It is likewise frequently applied to colic occurring in the neurotic—in whom there is not uncommonly a record of other forms of neuralgia in the individual or family history—the asthenic, the anæmic, or the gouty, even when there is a local exciting cause, such as flatus, retained feces, &c.; and to conditions in which pain predominates over spasm. The clinical features of enteralgia are, however, as a rule, indistinguishable from those of colic. A similar local disturbance—pain either sudden and sharp

or gradual and dull, either about the umbilicus or in the right iliac region—according to its intensity, is reflected by the nerves to the heart and the peripheral arteries. The action of the former becomes slow and feeble, and the latter contract; hence arising the small, infrequent, tense pulse, the cool pale skin, and the other signs of collapse which mark the distant effects of colic. And, as in this disorder, the termination of the attack may be sudden, perhaps following the expulsion of flatus or feces—a free perspiration, a copious flow of pale urine, the menstrual or lochial or other discharge, or a fit of the gout. As a rule, the more severe the paroxysm the shorter will be its duration. The exciting cause is not uncommonly a chill (wet feet, &c.), or mental over-strain and worry. Persistent enteralgia is now and then indicative of spinal disease, such as caries.

**TREATMENT.**—The main indications in the treatment of enteralgia are to endeavour to remove any cause of the pain: to administer opium or belladonna by the mouth or rectum, or subcutaneous morphine, and to apply hot fomentations, with laudanum and turpentine, for the relief of the suffering (*see* **COLIC**, **INTESTINAL**); and, during the intervals between the seizures, to restore the nutrition and tone of the nervous system, and improve the general health. Among medicines, courses of the hypophosphites, belladonna, quinine, and arsenic may be found most useful.

GEORGE OLIVER.

**ENTERIC FEVER.**—A synonym for typhoid fever. *See* **TYPHOID FEVER**.

**ENTERITIS** (*έντερον*, the bowel).—Inflammation of the intestines. *See* **INTESTINES**, Diseases of.

**ENTEROCELE** (*έντερον*, the bowel; and *κήλη*, a tumour).—A hernia containing a portion of bowel. *See* **HERNIA**.

**ENTOPHYTE** (*έντός*, within; and *φυτόν*, a plant).—A plant parasitic in any part of the body. *Entophytic diseases* are diseases that are supposed to depend upon the growth of such plants, as, for example, fungus-foot. *See* **FUNGUS-DISEASE OF INDIA**.

**ENTOZOA** (*έντός*, within; and *ζῶον*, an animal).

Man, in common with other animals, vertebrate and invertebrate, is liable to entertain various forms of entozoa. In comparison with other vertebrates, even with members of his own order, Primates, the number known to infest man is singularly small. This is attributable in a large measure to the fact that his food is submitted to the process of cooking. The various entozoa found in the human subject are truly parasitic, and not examples of commensalism. A parasite inhabits a living organism, and

obtains nourishment from its body. In *commensalism* creatures live within the bodies of larger animals, like parasites, but share its food, or live upon the refuse of its body instead of the juices and tissues of the host. The more important entozoa found in man are:—

- I. *Cestoda*, Tapeworms.
- II. *Trematoda*, Flukes.
- III. *Nematoda*, Round-worms.
- IV. *Gregarinidæ*, Psorosperms.
- V. *Rhizopoda*, Amœba coli.

I. *Cestoda*, or Tapeworms (κεστός, a girdle).—Fr. *Cestoïdes*, *Ténia*; Ger. *Bandwurm*.

Varieties:—

- a. *Tænia solium*.
- b. *Tænia mediocanellata* vel *saginata*.
- c. *Tænia bothriocephalus* *latus*.
- d. *Tænia cucumerina* vel *elliptica*.
- e. *Tænia nana*.
- f. *Tænia echinococcus*.

a. *Tænia solium*.—This worm when fully developed may attain a length of 2 to 3 feet. The vertex or head, about the size of a small pinhead, is furnished with four suckers, a rostellum, and twenty-six hooklets (fig. 34). A narrow neck succeeds the head, and at a short distance the segments begin to be visible. At first their breadth exceeds their length; but farther from the head they lengthen, until the mature segments are reached. These average 12 mm. in length and 5 mm. in width.

Each mature segment contains male and female generative organs. The genital openings are at the sides of the segments near the middle: the ovary presents seven to ten branches, each ending in a dendritic manner (fig. 36).

*T. solium* inhabits the small intestine of man, and is acquired by eating pork. Infection takes place in the following manner: The eggs, either free or contained in the segments (proglottides), are evacuated from the host, and are conveyed either by means of food or drinking-water into the body of a pig or other animal. When the eggs reach the stomach of the pig the capsule is dissolved, and the liberated embryo makes its way into the wall of the intestines or is carried by the blood-current into other parts of the body. The young tapeworm, when it emerges from the egg, possesses six hooklets, and is known as the 'six-hooked embryo.' After settling in the tissues it becomes metamorphosed, and is now furnished with a head and becomes enveloped by a membrane. In this condition it is known as a 'measle' or *Cysticercus cellulosa* (κύστις, a bladder; and κέρκος, a tail) (fig. 32). Pork containing the cysticercus is said to be 'measled.' The cysticerci are destroyed by thorough cooking, either by boiling or the more satisfactory process of roasting. When a cysticercus is introduced into the intestine,

the head (scolex) fixes itself into the mucous membrane, the cyst-membrane is dissolved, a chain of segments or proglottides develop, and a tapeworm is the result.

The cysticercus occurs also in man, and is probably due to the introduction of the eggs of *T. solium* into the stomach. Cysticerci have been found in the aqueous and vitreous chambers of the eye, in the subcutaneous tissue, and in muscles. When encysted they retain their vitality for many years. The presence of these small bodies excites local irritation, inducing inflammatory thickening. In muscles and intermuscular tissue they occasionally cause swellings resembling tumours. In the brain, when numerous, they induce fatal results. A few may exist in the cortex of the cerebrum without producing any obvious effect, but in the neighbourhood of the medulla two cysticerci have been known to cause death. In the eye cysticerci have been detected by means of the ophthalmoscope. In this situation they present a striking and characteristic appearance.

b. *Tænia mediocanellata* vel *saginata*.—This tapeworm differs from *T. solium* in several important particulars. When fully developed it may attain a length of 4 feet. The segments are thicker and wider than in *T. solium*; it has no rostellum or hooklets, but four suckers or proboscides and



FIG. 32.—*Cysticercus (telæ) cellulosa*, removed from the human eye by Mackenzie.  $\times 5$  diam. After Allen Thomson.

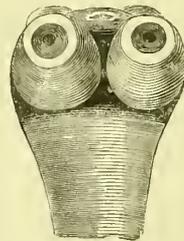


FIG. 33.—Unarmed Head of *Tænia mediocanellata*.  $\times 10$  diam. After G. Fritsch.



FIG. 34.—Armed Head of *Tænia solium*.  $\times 10$  diam. After G. Fritsch.

a median canal (fig. 33). The mature segments have a larger number of uterine diverticula, and they do not end dendritically (fig. 35). The cysticercus of this tapeworm infests the muscles of oxen. The tapeworm itself is acquired by man in consequence of eating underdone beef. In the accompanying sketches the heads and proglottides of *Tænia solium* and *Tænia mediocanellata* are arranged side by side for comparison.

c. *Tænia bothriocephalus latus*.—This is the largest tapeworm infesting man; when mature it attains a length of 5 to 8 feet, and contains two, three, and even four

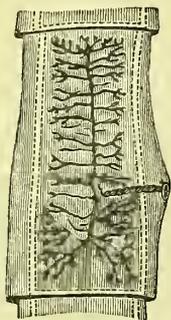


Fig. 35.—Proglottis of *Tænia mediocanellata*.  $\times 1\frac{1}{2}$  diam. After G. Fritsch.

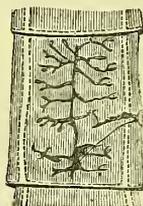


Fig. 36.—Proglottis of *Tænia solium*.  $\times 1\frac{1}{2}$  diam. After G. Fritsch.

thousand segments. The uterus has the form of a coiled ribbon, and the genital opening is near the middle of the ventral surface.

This worm lives in the small intestine, and is met with mainly in Switzerland and north-eastern Europe. It has been reported as occurring in Ireland.

It has long been suspected that fish were the intermediate host of this cestode, and Braun has demonstrated its occurrence in the larval stage in the muscles and viscera of pike, trout, and turbot.

d. *Tænia cucumerina* vel *elliptica*.—This is a small tapeworm, common in cats and dogs, rarely found in man. It has a rostellum and about sixty hooklets arranged in four irregular rows. The intermediate host is the dog-louse, *Trichodectes canis*. This fact explains the large number of *tænia* found in dogs not cleanly kept. The lice worry the dog, and it licks the irritated parts, and thus takes the lice with the cysticerci into the alimentary canal. Stroking dogs, or receiving their lingual caresses, is probably the means by which man is infected. It is noteworthy that children have furnished nearly all the specimens of this worm which have been reported in the human subject.

e. *Tænia nana*.—This is a small tapeworm. The head has four suckers, a rostellum, and circlet of twenty-two to twenty-eight hooklets. Bilharz found it in great numbers in the duodenum of a boy in Egypt. It is very rarely entertained in the alimentary canal of man.

**SYMPTOMS.**—The presence of tapeworms is indicated by a great variety of symptoms. Among the more important may be mentioned interference with the digestive process, irritability, and restlessness at night. Anæmia, headache, and vertigo are not uncommon. Irritation about the anus is sometimes a source of annoyance. Reflex pheno-

mena, such as chorea, convulsions, or epileptiform seizures, insanity, mania, squint, and other untoward conditions, have been known to coincide with the presence of tapeworms in the alimentary canal, and to disappear when the worms have been expelled. Many other grave phenomena have been attributed to their presence. It is equally true that hundreds of individuals entertain tapeworms and suffer no inconvenience in consequence, nay, even are unaware of their guests until apprised of the fact by the appearance of two or three feet of cestode segments passed *per anum*.

**TREATMENT.**—Many drugs have been recommended, such as turpentine, kousso, kamala, areca-nut, and other anthelmintic remedies. The late Dr. Cobbold, after a long experience, came to the conclusion that the liquid extract of male fern is the most reliable drug. The patient, if adult, should take a dose of castor-oil at night, a drachm of extract early next morning on an empty stomach, to be followed in three hours by a small dose of castor-oil. The fragments of worms should be examined in order to ascertain if the head be expelled.

f. *Tænia echinococcus* and *Hydatidis (vâdaris)*, a drop of water).—Fr. *Hydatide*; Ger. *Blasenwurm*.—*Tænia echinococcus* is especially interesting because its larval stages occur in man under the name of hydatid cysts.

The mature *tænia* inhabits the intestine of dogs, jackals, and wolves. It is about 4 mm. in length, and consists of only four segments, of which the terminal one alone is mature. Its head has four suckers, a rostellum, and two rows of hooklets, varying in number from fourteen to twenty-five in each row.

The eggs of this worm are introduced into the alimentary canal with the food, but more commonly with drinking-water. The six-hooked embryo, emerging from the egg, makes its way into surrounding tissues, and is frequently carried along by the blood-current in the veins as an embolus, to find a resting-place in lungs, kidney, liver, omentum, or brain, and becomes gradually transformed into an hydatid cyst.



Fig. 37.—*Tænia echinococcus*.

For the history of the early stages of these cysts we are indebted to the zeal and industry of Leuckart. At first they are small white dots, with thick, homogeneous, transparent capsules, with concentric lamination, enclosing coarsely granular contents (fig. 38). After the cyst has been growing five months, and has attained a diameter of half an inch,

echinococcus-heads begin to bud from its walls. Each of these heads, when fully formed, is about 0.3 mm. in length when fully distended, and represents the head and neck of an adult echinococcus (fig. 39). Each head is furnished with four suckers and an armed rostellum, the hooklets of which are

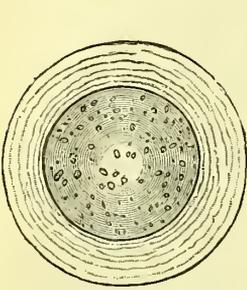


FIG. 38.—Hydatid of four weeks' growth, showing ectocyst and endocyst.  $\times 60$ . After Leuckart.

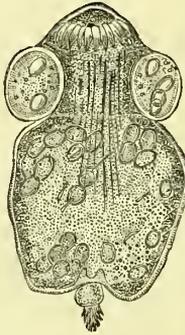


FIG. 39.—The so-called *Echinococcus* head, showing hooks, suckers, cilia, and corpuscles.  $\times 250$  diam. After Huxley.

very small. Numerous calcareous particles are lodged in its parenchyma. The rostellum with its hooks and suckers can be retracted and extended. In examining such specimens under the microscope it is usual to find them in the inverted or retracted condition shown in fig. 40. The heads or scolices are formed within brood-capsules, and Leuckart emphatically states that they are throughout life

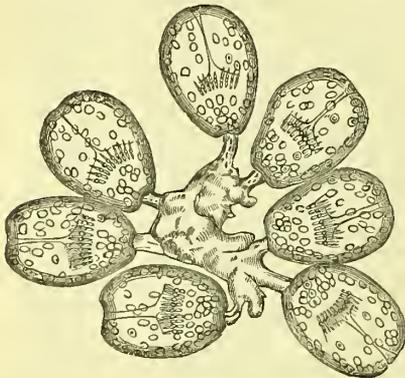


FIG. 40.—Group of *Echinococci*, with their hook-crowns inverted.  $\times 180$  diam. After Busk.

directly continuous with each other by means of the parenchyma of the main cyst, and cannot roam about the cavity and retain their vitality. The walls of hydatids consist of two layers, an outer or cuticular and an inner layer or parenchyma. The cuticular layer is highly elastic and curls up when divided; the parenchyma consists of granular matter, cells, and muscle-fibre. The brood-

capsules are developed from the parenchyma; each brood-capsule, like the parent-cyst, has two layers, an *outer* parenchymatous and an *inner* cuticular, thus reversing the conditions of the mother-cyst. Leuckart maintains that the scolices bud from the outer wall of the capsule; but when fully developed invaginate, so that what was formerly the internal cuticular surface of the head now becomes external. This view requires confirmation. The mother-cyst grows larger as fresh brood-capsules are formed. Every echinococcus cyst does not develop brood-capsules, and may attain a large size yet remain sterile. The presence of the cyst irritates the surrounding structures, and leads to the formation of a spurious capsule of fibrous tissue, sometimes of great thickness and usually very vascular. The brood-capsules, with their contained scolices, occur in clusters; each capsule is of the size of a No. 5 shot. Occasionally the capsules become much larger and form internal daughter-cysts attaining in some specimens the size of a Tangerine orange. Such daughter-cysts may develop cysts of their own, or granddaughter-cysts. When endogenous daughter-cysts are present, the mother-cyst is, as a rule, of very large size, and contains as many as two or three thousand daughter-cysts varying in size from a pea to an orange. Occasionally, we find in echinococcus cysts occurring in cattle, that daughter-cysts are formed from the cuticle and protrude beyond the *external* wall of the parent-cyst, and form brood-capsules. A few examples of this condition have been reported in the human subject.

*Echinococcus multilocularis* is a variety of hydatid, which has been described as affecting the liver. It forms a hard tumour made up of alveoli, separated by fibrous tissue. The alveoli are filled with gelatinous transparent material, stained at times with bile. Such specimens were formerly described as colloid cancer of the liver, until Virchow pointed out that they were composed of echinococcus cysts, and occasionally contained scolices. Nearly all the reported cases have occurred in South Germany and Switzerland. The true nature of this form requires elucidation. The recognition of *E. multilocularis* is rendered more difficult by the circumstance that they sometimes ulcerate. This condition may easily be confounded with actinomycosis of the liver.

**ANATOMICAL CHARACTERS IN MAN.**—The human body frequently contains echinococcus cysts; there is scarcely an organ in which it has not been found, even in the medullary cavities of bones. Like the *cysticercus cellulosa*, the embryo of *T. echinococcus* has its favourite situations: the former selects chiefly the intermuscular connective tissue and brain, the latter selects mainly the viscera, especially the liver. The liability of organs to entertain hydatids may be in-

ferred from Neisser's tables as quoted by Leuckart. They are based upon an analysis of 986 cases : Liver, 451; lungs and pleura, 84; kidney, 80; brain, 68; muscles, dermis, and orbit, 72; spinal cord, 13; pelvis, 36; circulatory organs, 29; spleen and bones, 28, and so on. This table is only useful as showing the greater liability of viscera to harbour this parasite. In the bones, eye, orbit, mamma, thyroid body, and heart it is of extremely rare occurrence.

It is strange that, though the liver is such a favourite viscus for hydatids, it is rare to find more than one cyst present in this organ at the same time in the human subject; yet in swine they are frequently multiple. In man, one large cyst may be lodged in the liver, and twenty, thirty, or even a hundred occupy the omentum, mesentery, &c. This condition is, however, very uncommon.

**SYMPTOMS.**—The danger caused by these cysts arises mainly from the mechanical pressure they exercise on surrounding parts. Thus, in the brain or heart, a cyst the size of a hazel-nut gives rise to serious disturbance, but one as large as a man's head is often tolerated in the liver or pelvis. These cysts are often found *post mortem* when their presence had never been suspected during life, although they attained goodly proportions. The parasite may die early in its career, and the cyst-wall calcify; hydatid membranes seem very prone to this change, the calcareous matter resembling very old dried mortar. Hydatid cysts cause death in a variety of ways. In the brain they simulate cerebral tumour, and induce death early, especially when growing near the bulb. In the spinal cord they produce paraplegia. In the liver they may be accidentally ruptured, and cause death in a few hours from shock and hæmorrhage combined, or, later, from peritonitis. In some instances, after injury or meddlesome tapping, suppuration takes place with septic troubles, which the formation of pus entails. Liver cysts may perforate the diaphragm, and rupture into the pleura, or communicate with the lung and its bronchi. Occasionally they open into stomach or bowel, the scolices being discharged *per anum*. They have been known to rupture into the bile-passages, the transit of the scolices causing biliary colic. Among the rarer sites of rupture may be mentioned the pericardium, vena cava inferior, or through the parietes of abdomen or thorax.

In the lung pressure upon the air-cells or bronchi may cause gangrene and other troubles which follow bronchial obstruction. Hydatid cysts of the thyroid body are very rare; they usually rupture into the trachea and cause death by suffocation.

**DIAGNOSIS.**—The diagnosis of hydatid cysts, in organs other than the liver, is attended with very great difficulty; and as these cysts frequently appear at *post-mortem* examina-

tions and astonish the physicians, so are they one of the most fertile sources of surprise to enterprising surgeons. In the liver a hydatid cyst may be suspected when a tumour can be made out unaccompanied by pain, cachexia, high temperature, or wasting.

The conditions most likely to be confounded with hydatid cysts in the liver are abscess, distended gall-bladder, effusion in the right pleura, aneurysm of the abdominal aorta or hepatic artery, cancer of the liver, renal cysts, dilated stomach, pancreatic cyst, ovarian tumours, and dropsy due to closure of Winslow's foramen. See LIVER, Hydatid Disease of.

It is not wise to rely on the fact that the patient has lived among dogs or in a pastoral country. Individuals who have never kept or lived with dogs often develop hydatids.

When an abdominal or thoracic tumour is suspected to be a hydatid cyst (these are the situations in which physicians are most frequently called upon to diagnose them), the nature of the swelling should be ascertained by puncturing it with a trocar, and drawing off some of the fluid. The characters which determine its echinococcus nature are the following:—

The fluid is limpid, colourless (occasionally bile-stained when growing in the liver), and in specific gravity varies from 1009 to 1015. Free from albumen, it contains chloride of sodium, and is said to furnish traces of succinic acid, leucin and tyrosin. The three last are present in quantities too small to be of any clinical value. The most positive signs are the presence of brood-capsules, scolices, hooklets, or the gelatinous, trembling, laminated membrane which forms the true wall of the cyst, and is in itself absolutely peculiar to hydatids.

It must also be remembered that hydatid cysts are occasionally sterile—that is, they contain no brood-capsules and furnish no hooklets. Such cysts frequently attain a large size, and seem to occur most frequently in the pelvis; they are as a rule mistaken for other forms of cysts.

**TREATMENT.**—The treatment of hydatid cysts is entirely surgical. In such an organ as the liver the cyst should be freely opened, and its edges stitched to the sides of the incision and drained. The walls collapse, the cyst-membrane comes away during the operation, or is subsequently discharged piecemeal. The cavity then slowly granulates.

When hydatids are lodged in the omentum they are easily removed by abdominal section. When deeply placed in the pelvis they may be situated between the bladder and the rectum in the male, or burrow between the layers of the broad ligament in the female. These are better treated by incision and drainage. Hydatid cysts have many times been recorded in the kidney, but it is very unusual for them to exceed the dimensions of

an orange. They exhibit a marked tendency to discharge into the renal pelvis, the vesicles escaping by way of the urethra. Should the cyst attain such proportions as to give rise to a palpable tumour, it should be explored by a lumbar incision, and treated in the way recommended for the liver. Such cysts arising in the kidney have been known to open into the lung, into the stomach, or communicate with the intestine. Intra-thoracic hydatids are best treated on the same surgical lines as empyema. In the subcutaneous tissues and orbit they are rarely diagnosed—indeed, these cysts in such situations can only be suspected; the suspicion is confirmed when the vesicles or portions of membranes escape during removal of the tumour. Intracranial hydatids have in Sydney, Australia, been successfully submitted to surgical procedure. It is certain that no tumour or cyst connected with the brain offers a more favourable prospect to surgeons than a hydatid cyst connected with the cerebral cortex.

**II. Trematoda, or Flukes (τρήμα, a hole).—Fr. Trématodes; Ger. Trematoden; Saugwürmer.**—These forms have only one opening to the digestive cavity.

Varieties:—

- a. *Distoma hepaticum*.
- b. *Distoma lanceolatum*.
- c. *Distoma hæmatobium* vel *Bilharzia hæmatobia*.
- d. *Distoma Ringeri*.—See p. 259.

a., b. The first two are rarely seen, though they are common enough in the biliary passages of sheep and oxen. They are vulgarly known as flukes, and are the cause of the destructive disease known as 'the rot' of sheep. When flukes, with the exception of the *Bilharzia hæmatobia*, occur in man, they occupy the biliary passages, but are usually present in such limited number as to rarely cause any serious disturbance of health.

c. *Bilharzia hæmatobia*, or *Distoma hæmatobium*, unlike other forms of flukes, has the sexes distinct. The male is 12–14 mm. long, and has a flattened body, which, at its posterior part, is rolled into a kind of tube, constituting the gynæcophoric canal. The female is almost cylindrical in form, and 16–19 mm. long, being lodged within the gynæcophoric canal (fig. 41). The mature worms live in the inferior vena cava, portal, splenic, mesenteric, vesical, and hæmorrhoidal veins of men and monkeys. The ova pass into the mucous membrane of the bladder and intestine, probably escaping as a consequence of the rupture of small vessels. Occasionally the ova may be found in the ureter and the pelvis of the kidney. The ova, often present in great numbers, are furnished with either a terminal or a lateral spine. Dr. Zancarol says that ova with a lateral spine occur in the intestine; those with the terminal spine in the mucous membrane of the bladder. The use of this ap-

pendage has not yet been satisfactorily explained (fig. 42).

The *Bilharzia* is common in Egypt, Abyssinia, the Cape, and Natal. The cercaria, as

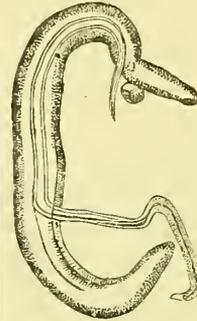


FIG. 41.—*Bilharzia hæmatobia*, male and female sexually combined. Magnified. After Küchenmeister.

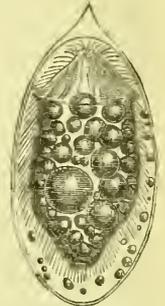


FIG. 42.—Ovum of *Bilharzia hæmatobia* with contained embryo and free sarcodermic granules.  $\times 234$  diam. Original.

the larval forms are termed, abound in rivers and canals, and are especially common in the Nile. When taken into the alimentary canal the cercaria quickly work their way into the mucous membranes, gain access to blood-vessels (veins), and develop into mature flukes.

**SYMPTOMS.**—When lodged in the intestines they give rise to dysentery; in the bladder and ureters, to cystitis, hæmaturia, and vesical calculus. The hæmorrhage caused by their presence in the genito-urinary tract is known as 'endemic hæmaturia.' The eggs, whilst still within their host, may develop ciliated embryos. The disease may be suspected when hæmaturia occurs in persons coming from countries where the *Bilharzia* abounds: the diagnosis is confirmed by finding under the microscope the ova embedded in fragments and shreds of mucous membrane passed with the urine.

**TREATMENT.**—Means must be taken to allay the vesical irritation by such remedies as buchu, uva ursi, hyoscyamus, and the like. No remedy is known that acts as a parasiticide to the *Bilharzia*. It is wise to remove the patient, if possible, from an infected locality. Cure is likely to be protracted, and as yet we are ignorant of the duration of life of the fluke.

**III. Nematoda, or Round-worms (νήμα, a thread).—Fr. Nématoides; Ger. Fadenwürmer.**

Varieties:—

- a. *Ascaris lumbricoides*.
- b. *Oxyuris vermicularis*.
- c. *Trichocephalus dispar*.
- d. *Ankylostoma duodenale*.
- e. *Filaria medinensis*.
- f. *Trichina spiralis*.
- g. *Filaria sanguinis hominis*.

Most of these inhabit the alimentary canal, and when restricted to this region rarely give rise to dangerous symptoms, unless they be very numerous; but they are apt to migrate and invade other organs, and thus give rise to dangerous and often disastrous results.

*a. Ascaris lumbricoides*.—This, the common round or maw-worm, is of cylindrical shape, pointed at each end, and of a light brown colour. The female, as is usual among nematodes, exceeds the male in size. The length of the female varies from 25 to 40 cm. The male is distinguished by the presence of two chitinous spines near the caudal end of the body (fig. 43). This extremity is also bent and hook-like. The spines indicate the male genital orifice. In the female the genital pore is situated near the middle of the body. The ova are very numerous; when mature they possess a double shell surrounded by an albuminous coating, have considerable power of resistance, and are not killed by drying or freezing.

The life-history of the worm is not fully known, and we are ignorant of its intermediate host. Dr. Cobbold was of opinion that it did not require one, but that the eggs develop in water.

This worm lives chiefly in the small intestine, but may wander into the stomach, and even pass up the œsophagus and be discharged through the mouth. An *ascaris* has been known to creep into the bile-duct and cause jaundice. In cases of perforation of the intestine in fatal cases of typhoid fever, these worms have been found lodged in the aperture. We must not conclude from this that the worm caused the perforation; it is more reasonable to believe that when the accident happened the ascaris was passively lodged in the opening during the outward rush of the fluid contents of the bowel. It is possible that

Any foreign body in the intestine is sure to attract their attention and to form a sort of 'worm-trap.' Thus they have been strangled by metallic buttons, hooks and eyes, open-topped thimbles and the like (Cobbold).

**SYMPTOMS.**—It is unusual for an individual to harbour more than two or three round worms, and as a rule they rarely give rise to symptoms. When present in large numbers—a condition of things most common in children—diarrhœa, colic, nausea, vomiting, and convulsions may occur. Children have been known to pass in the course of a few years. one hundred worms, and in one case 510 worms were voided by a child. Generally the passage of a worm by the anus or mouth is the first and only indication of its presence.

**TREATMENT.**—The most reliable drug is santonin, in the form of a powder: for children two to four grains, followed by a saline purge or castor oil; for adults five grains daily; for three days each dose to be followed by castor oil. Among other useful remedies we may mention aloes, scammony, jalap, calomel, kamala, sulphur, and turpentine.

*b. Oxyuris vermicularis*

(*Thread- or Seat-worm*).—The thread-worm is an inhabitant of the colon and adjacent parts of the ileum. The female is usually about 10 mm. and the male 4 mm. long. In addition to its greater size the female has a sharp-pointed tail, whereas in the male it is blunt and furnished with a spiculum (fig. 44). Some helminthologists are of opinion that the mature females inhabit the colon, whilst the males and immature females are found in the ileum.

Oxyurides are often present in large numbers, and wander into the rectum. They often cause intense irritation in the



FIG. 43.—*Ascaris lumbricoides*; male, with exerted spicules. Nat. size (Cobbold).



FIG. 44.—*Oxyuris vermicularis*, female. Highly magnified. After Leuckart.

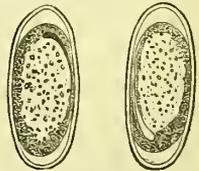


FIG. 45.—Eggs of *Oxyuris vermicularis*, enclosing tadpole-shaped embryos.  $\times 450$  diam. Original.

worms may occasionally perforate the intestinal wall, and, getting into the subserous tissue, give rise to abscess. Such cases have been recorded. The wandering habit of ascariides is often fatal to them.

neighbourhood of the anus, and in females cause great distress by creeping into the genital passages, especially during the night. The eggs of *oxyuris* are expelled with the fœces

and require to be taken into the stomach before they re-develop (fig. 45). Persons re-infect themselves by scratching the anus, and conveying the ova, by means of the finger-nails, to the mouth.

**SYMPTOMS.**—Among the local symptoms heat and irritation around the anus and nose are common. In children especially we often find restlessness, nervous irritation, choreic symptoms, and convulsions. Signs suggesting such severe conditions as meningitis are by no means infrequent. The existence of thread-worms is easily verified by administering a mild purgative and examining the fæces. As a rule, they will be found in considerable numbers. In the case of children the attention of the nurse is called by finding the worms on the sheets.

**TREATMENT.**—Brisk saline purgatives to expel the worms, and extreme cleanliness to prevent re-infection, are usually all that is required. Fruits and vegetables should be thoroughly cooked. In adults treatment requires to be carried out more vigorously, with such drugs as calomel, scammony, aloes, jalap, santonin, and asafœtida, in conjunction with enemata of infusion of quassia, or salt and water.

*c. Trichocephalus dispar*, or *Whip-worm*.—The male and female equal each other in

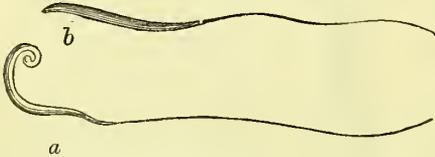


FIG. 46.—*Trichocephalus*; male (a), and female (b). Enlarged one-fourth.

length; as a rule they average 4 to 5 mm. The anterior part of these worms is fine and thread-like, the posterior is thicker, and contains the genital organs. In the male the caudal end is rolled into a spiral; near the extremity it is furnished with a spine (fig. 46).

The trichocephalus resides mainly in the cæcum. The early stages are passed in water or damp earth. The worm is very common in France: Davaine calculated that half the Parisians are infested by it. In England it is rare. Unless present in great quantities it is not often a source of trouble.

*d. Ankylostoma duodenale*.—This small worm is also known as *Sclerostoma duodenale*, *Dochmius*, or *Strongylus duodenalis*. Occasionally it is referred to as the *tunnel-worm*, on account of the disasters it caused among the men engaged in the Mount St. Gothard Tunnel. The female averages twelve mm. in length, the male is slightly smaller. The cephalic end is furnished with an oral capsule, which possesses a cleft covered by two chitinous lamellæ. The ventral lip has

four curved teeth; the dorsal, two straight ones. The caudal end in the male has a three-lobed bursa and two spines (fig. 47). This worm infests the small intestine and bores its way into the mucous membrane.

The effects of ankylostoma on the gut are very characteristic. The damaged spot is indicated by a small ecchymosis, with a central depressed white spot, in which the head of the worm lodged; occasionally it may be found still anchored to the mucous membrane. When present in large numbers these worms produce anæmia, sometimes called Egyptian chlorosis. The ankylostoma is very common in Egypt and Brazil. One stage of the worm is passed in muddy or dirty water; it gains access to the alimentary canal in drinking-water, and there attains its maturity.



FIG. 47.—*Ankylostoma duodenale*; male (A), and female (B).  $\times 5$  diam.

**SYMPTOMS.**—The presence of this worm is indicated by the usual signs of anæmia, viz. pallor of the mucous membranes, weakness, palpitation, and faintness. Diarrhœa and dropsical effusions sometimes occur. The anæmia is due to the loss of blood consequent on the injuries inflicted by these parasites on the intestinal mucous membrane.

**TREATMENT.**—The worms should be evacuated by calomel and santonin combined, given at intervals over a period of a few weeks, for it must not be imagined that one or two doses will destroy the thousands of helminths lodged in the intestines in a severe example of this disease. The liquid extract of male fern is a useful remedy. The patient will require nourishing diet, easily digestible food, and good wine, with the usual tonic remedies.

*e. Filaria medinensis*, *Dracunculus*, or *Guinea-worm*.—This is a fine thread-like worm from 60 to 100 cm. in length, and about 2 mm. broad. The female only is known. The embryos develop within the uterus. The intermediate host is a minute crustacean (*Cyclops*), which inhabits drink-



FIG. 48.—*Filaria medinensis*. Reduced to  $\frac{1}{2}$ .

ing-water. Within the cyclops they are introduced into the human stomach, and then migrate into the subcutaneous tissues, where they become mature. A common situation is the tissue beneath the skin of the legs and feet; the heel is a favourite situation.

The worm is confined to certain districts of Asia, Africa, and Brazil. It is supposed to be identical with the fiery serpents mentioned in the Pentateuch.

When the worm first lodges in the subcutaneous tissue it gives rise to little trouble; as it grows an abscess forms and gradually exposes the worm. Treatment consists in carefully winding the worm with gentle traction around a slender stick of ivory, bone, or wood, an inch or so daily, taking care not to break it. This method seems to have been employed of old by Persian surgeons.

f. *Trichina spiralis*.—The mature worm inhabits the intestinal canal. The female is 3 mm. long; the male is much smaller, and is further distinguished by presenting on the dorsal side of the tail two mammillary protuberances, which are turned towards the ventral aspect, and include between them four wart-like nodules (fig. 49). The young are developed in the uterus, and are born in a free state. The immature trichina inhabits



FIG. 49.—*Trichina spiralis* magnified; male (a), and female (b). After Leuckart.

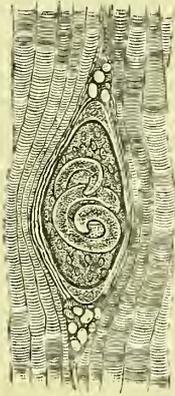


FIG. 50.—Portion of human muscle, enclosing a single capsuled *trichina*. Highly magnified. After Leuckart.

voluntary muscular fibre, where it is usually coiled into a spiral, embedded in granular tissue and surrounded by a cyst-wall, which frequently contains calcareous particles (fig. 50). These capsules are visible to the naked eye as minute white specks. Each capsule usually contains one trichina, but two, three, and even five may be present.

The relation of the two forms is as follows. When a piece of muscle containing living trichinae is taken into the stomach, the cap-

sules are digested and the worms set free. In two days and a half they become mature, and pair. The embryos commence to be born on the seventh day; and the female may continue to bring forth worms for many days. The mature worm lives but a few weeks, but during this brief life may bring forth a thousand or thirteen hundred embryos. The young then seek for striated muscle-fibre, and migrate through the wall of the intestine and the subperitoneal tissue. A few may get into vessels and are distributed by the circulation. On gaining access to a muscle they penetrate the fasciuli, becoming in about fourteen days mature muscle-trichinae. In due course they become encysted; the cyst is said to be in part due to a secretion furnished by the trichina. After a time the cyst-wall calcifies; should the trichina die, the cyst-contents also calcify. The duration of life of the worm in the muscle is very long, and even survives for a time the death of its host. The trichinae are found most abundantly in the intercostal muscles, diaphragm, and laryngeal muscles. In the limb muscles they are less abundant, and cluster near the union of the muscle with its tendon. The trichinae so accurately select the voluntary muscles that the pharyngeal constrictors and upper part of the oesophagus will lodge hundreds of them, while the rest of the tube contains none.

Symptoms.—Human beings usually acquire the disease from eating trichinosed pork. The symptoms may be divided into two stages: the first consists of gastro-intestinal disturbance due to the introduction of the parasite; the second corresponds to the migration of the worm, and is characterised by muscular troubles and febrile disturbance.

The gravity of the symptoms depends upon the number of trichinae taken. The initial gastro-intestinal catarrh lasts usually about a week. The second stage is characterised by high fever and pain in the limbs and muscles, often mistaken for rheumatism. The muscles sometimes swell, become oedematous, and occasionally are partially paralysed. In grave cases delirium ensues, the limbs become flexed and paralysed, there is excessive diarrhoea, terminating in death. The duration of the second stage lasts four or five weeks. In fatal cases, death usually occurs about the third or fourth week.

At the end of five weeks the migration is usually complete, and the trichinae once encysted give rise to no further trouble. That this is so is clear from the fact that, in dissecting-rooms, subjects occasionally occur with many hundred thousand trichinae encysted in the voluntary muscles of all parts of the body, from the head to the sole of the foot. The diagnosis may sometimes be established during life by an examination of suspected articles of food, or by the detection of adult trichinae in the evacuations during the

course of the disease. The most certain plan is to remove a fragment of affected muscle and examine it under the microscope.

**TREATMENT.**—If it can be at once ascertained that trichinosed food has been taken into the stomach, an emetic should be administered without delay, followed by a brisk purgative; this treatment should be pursued for several days to expel the worms before they become mature. Calomel alone, or combined with scammony or jalap, is recommended. Even when the young filariæ have begun their migrations, brisk cathartics still do good; but the patient's strength will require support during this critical stage by solutions of meat, broth, eggs, milk, and the judicious administration of wine or brandy. As the patient becomes convalescent ordinary tonics will be needed.

Preventive treatment consists in rejecting suspected trichinosed food, especially pork. The disease is rare in Great Britain, but fairly frequent in Germany, where it is the custom to eat 'Wurst,' or sausages made of smoked ham.

*g. Filaria sanguinis hominis.*—This parasite and its embryo are separately described. See **FILARIA SANGUINIS HOMINIS.**

**IV. Gregarinidæ** (*gregarius*, from *grex*, a flock).—Fr. *Grégarines*; Ger. *Gregarinen*.

Variety:—*Coccidium oviforme*.

The disease produced by the presence of the *coccidium oviforme* is known as *Psorospermia* ( $\psi\acute{o}\rho\alpha$  or  $\psi\acute{o}\rho\eta$ , a cutaneous disease—the itch, scab, or mange. The Latin equivalent is *scabies*). The French use the word *psore* as a generic title for vesicular and pustular maladies of the skin. J. Müller seems to have first used the word 'psorosperm' to indicate a parasitic affection of the skin, gills, muscles, and kidneys of fish and frogs. In the skin of fish the disease consisted of small sacs containing oval bodies, some with and some without tails; these he termed psorosperm saccules.

It has long been known that the destructive liver-disease of rabbits is due to the presence of small oval bodies named *coccidia oviformia*, lodged in the bile-ducts. The coccidium is thus described by Leuckart: It is egg-shaped, 0.033 to 0.37 mm. long, and 0.015 to 0.02 mm. wide, with a thick smooth shell, possessing at one end, usually the narrower, a micropyle. The coccidia resemble very closely the eggs of certain entozoa.

It has been known since 1858 that this coccidium occurs in the human subject, and since that date numerous cases have been observed. Among the recent cases, one described by Mr. Silcock may be mentioned. A woman, fifty-three years of age, was thought to be suffering from typhoid fever. The illness dated from a chill taken seven weeks before death. The symptoms were pain in the limbs, nausea, and occasional sickness, tenderness over the liver and spleen, fever

of remittent type, the temperature reaching 103° F., and slight diarrhœa. The urine was albuminous, the splenic and hepatic dulness increased, and the tongue dry and coated with brown fur. Death resulted from cardiac failure.

On *post-mortem* examination the liver weighed eighty-three ounces, and contained numerous small caseous foci; the spleen weighed sixteen ounces, and presented similar foci varying in size from a pin's head to a pea. Papule-like elevations dotted the mucous membrane of the ileum and large intestine. The naked-eye characters of these nodules resembled tubercle, but on microscopic examination they were found to be coccidium nodules.

The mucous membrane of the ureters is a favourite situation for these nodules. In this situation they give rise to hæmaturia, frequent micturition, and death from exhaustion.

There is reason to believe that these parasites invade the orifices of the follicular ducts of the skin and give rise to molluscum contagiosum and chronic eczema of the nipple (Darier). Confirmatory observations have been made in London.

**V. Rhizopoda** ( $\rho\acute{i}\zeta\alpha$ , a root; and  $\rho\acute{o}\delta\varsigma$ , a foot).—Fr. *Rhizopodes*; Ger. *Rhizopoden*.

*Amœba coli.*—Lambl appears to have been the first to detect amœbæ as human parasites, but the first satisfactory investigations were made by Lösch in 1875. He found them in the stools of a Russian peasant suffering from ulceration of the large intestine. In this case the amœbæ were five to eight times larger than the red blood-corpuscles; their protoplasm, which was coarsely granular, presented a few blunt processes, a round nucleus, and several vacuoles.

Lösch found them in great number; as many as sixty could be seen in one field of view with a magnifying power of 500. The peasant who was the subject of Lösch's researches had lived a miserable life; his illness presented all the signs of violent dysentery. Pleurisy and pneumonia supervened, accompanied by anæmia, exhaustion, and death. The fresh dejections from this patient were injected into dogs *per os et anum*, but with incomplete results. In 1883 Kartulis examined in Egypt 150 bodies of persons dead of dysentery, and found the amœba in every case. In twelve *post-mortem* examinations amœbæ were found in the ulcers. Subsequently Kartulis extended his observations to hepatic abscess complicating dysentery, and found amœbæ in sections of the abscess-wall. They resembled those found in the intestine. He has since extended his researches so as to include 500 cases of dysentery, with confirmatory results.

The amœba coli has been found in the intestines of dysenteric patients by other observers—Massentin, Baungarten, and Osler.

Dr. Osler's observations were made on a doctor, twenty-nine years of age, resident in Panama for nearly six years, who had suffered from chronic dysentery. When he came under Dr. Osler's care, abscess of the liver was detected and drained. As the pus came from the drainage-tube, samples were taken at once to the laboratory and examined. Amœbæ were present in large numbers, and continued to be present until the patient died. Amœbæ were also found in the stools, and in every respect identical with those from the abscess. They were rarely found in the brownish stools, but were more frequent in the mucus and in the greyish fragments of sloughs.

The amœbæ in size ranged from 10–20 mm., and were somewhat larger than those found by Kartulis. When once recognised there was not the slightest difficulty in distinguishing them, even when at rest, from pus-cells, not merely by their size, but from the entirely different appearance of their protoplasm.

How far these bodies are the exciting agents of the disease is uncertain, because amœbæ have been found in normal fæces; nevertheless they are deserving of extended study. They have been found in Egypt, Athens, St. Petersburg, London, and America.

J. BLAND SUTTON.

**ENTOZOON FOLLICULORUM.**—

A synonym of the animalcule of the follicles of the skin, otherwise named acarus (Simon), demodex (Owen), and steatozoon folliculorum (Erasmus Wilson). See ACARUS.

**ENTROPION** (ἐν, in; and τρέπω, I turn).

A morbid condition in which the eyelid is inverted, so that its free margin is directed towards the eye. See EYE AND ITS APPENDAGES, Diseases of.

**ENURESIS** (ἐν, in; and οὐρέω, I pass the urine).—Involuntary discharge or incontinence of urine. See MICTURITION, Disorders of.

**EPHELIS.**—SYNON.: Sunburn; Fr. *Ephélide*; Ger. *Sonnenflecken*.—This word is applied to pigmentary discoloration of the skin, of a brown, grey, or black colour, increased by the stimulus of light and heat, as of the sun's rays, or scorching by fire. Two principal varieties of the affection have been noted, namely, Ephelis solaris, and Ephelis ignealis. See PIGMENTARY SKIN-DISEASES.

ERASMUS WILSON.

**EPHEMERAL FEVER** (ἐπί, upon; and ἡμέρα, a day).—A mild form of milk-fever, so called on account of the rapidity with which it subsides, lasting not more than a day. See MILK-FEVER.

**EPHIDROSIS** (ἐπί, upon; and ἰδρώω, I sweat).—A term signifying a state of sweating, and synonymous with idrosis. See PERSPIRATION, Disorders of.

**EPIDEMIC** (*epidemicus*, affecting the people; from ἐπί, upon; and ἄνθος, a people).

The word 'epidemic' is used in two senses by medical writers and by medical men, namely, (1) in a general sense, and (2) in a technical sense.

As a *general term* the word signifies 'common to, or affecting, a whole people, or a great number in a community; prevalent; general' (*Webster's Dictionary*). It is in this sense that the word is used when it is applied to mental, moral, and social phenomena, as, for example, when we speak or write of 'epidemic suicide,' 'epidemic folly.' This employment of the word is consistent with received literary practice. Thus we read, 'There was a time when *wit was epidemic*' (*Athenæum*). Again, M. Littré, writing to the *Temps*: 'It argues great confidence in oneself and one's own enlightenment to treat with haughty disdain, and without reserving any compromise, the opinion of so many citizens, and to regard it as a case of *epidemic aberration*.'

As a *technical term*, having reference to disease, the word 'epidemic' has several different meanings attached to it. All these meanings include the notion of general prevalence among a community or a people, but some of them would go on, beyond what etymology justifies, to attach a peculiar hypothetical or theoretical conception to the term. Thus (a) Mayne restricts the term to diseases which are contagious, making contagion the essence of *epidemicity*, as he would phrase it; (b) Dunglison implies by the term a particular constitution of the air ('*constitutio aeris*, or condition of the atmosphere'); (c) other authoritative writers use the term as signifying a widespread cause, telluric, atmospheric, cosmic, as the case may be, acting at the same moment of time on many individuals, or as something occult, regarding which speculation is vain, and which they designate *epidemic constitution* or *epidemic influence*.

The foregoing technical significations attached to the word 'epidemic' are not less misleading than insufficient. Mayne's definition imposes an arbitrary limitation upon the meaning of the word, while it involves but a partial notion of the phenomena of epidemic prevalence of contagious diseases. Dunglison's definition does not rest upon a scientific foundation, and its phraseology, derived from a period when medicine was still hampered with semi-mystical speculations, cannot well be dispossessed of the vague traditional meanings which adhere to the word 'constitution.' Other technical definitions (if that can be called a definition which makes obscure what should be rendered clear) rest on mere assumptions, or relegate the term to the incomprehensible and insoluble. Of these last-named definitions Léon Colin has said: 'They signify

implicitly a common cause, apparently indecomposable, to which individuals are not exposed successively but simultaneously . . . a something isolated, impersonal, inaccessible to reason, detached from the disease itself, the epidemic genius [constitution, influence] . . . a creative force of the different epidemic affections, compelling, directing, extinguishing them.'

The promiscuous use of the word 'epidemic' in medical literature and medical talk, and the different irreconcilable significations attached to it as a technical term, have been, and still continue to be, sources of almost hopeless confusion in treating of diseases in respect to which the phenomenon of (etymologically) epidemic prevalence is observed. It is not difficult to apprehend how this has come about. The diseases in question (the *morbi populares* of some writers) include among their number plague, the eruptive and continued fevers, influenza, malignant cholera, &c. 'They have the peculiar character of attacking at intervals great numbers of people within a short period of time; they distinguish one country from another, one year from another; they have proved epochs in chronology; and, as Niebuhr has shown, have influenced not only the fate of cities, such as Athens and Florence, but of empires; they decimate armies, disable fleets; they take the lives of criminals that justice has not condemned; they redouble the dangers of crowded hospitals; they infest the habitations of the poor, and strike the artisan in his strength down from comfort into helpless poverty; they carry away the infant from the mother's breast, and the old man at the end of life; but their direst eruptions are excessively fatal to men in the prime and vigour of age' (WM. FARR). Exercising at all times in their greater and more fatal prevalence the profoundest influence over the mind, as well of the people generally as of the medical profession, these diseases, partly from the terror they inspire, partly from the extreme complexity of the phenomena they display, have formed a never-ceasing subject of the wildest speculation. No part of medicine has retained so much of the semi-mystical teachings of the older physicians as that which relates to epidemics, and general history has contributed with medical history to propagate in regard to epidemics that habit of thought which refers the unknown to the occult—using the latter term in the sense in which it is applied with regard to the imaginary sciences of the Middle Ages. Much of the speculation as to epidemics which passes current for science at the present day is in reality an unsuspected continuation of the mystical teachings of earlier medicine—magnetism, or electricity, for example, taking the place of Saturn or Mercury in the scheme of causation. The terminology is modernised, but the underlying conception remains the

same. Again, the so-called 'precursors' of epidemics, which still find a place in treatises on medicine, are the relics of the doctrine of portents of the Middle Ages. They rest on the assumption of an epidemic being determined by some common extra-mundane or intra-mundane cause, of which it is but one of several effects. The celestial 'portents,' such as comets and meteors, and the more manifest telluric 'portents,' such as earthquakes and volcanic eruptions, have been discarded; but the 'portents' derived from exceptional developments of insect life, from murrain, from unusual prevalence of certain diseases, and from remarkable perturbations of the weather, are retained under the denomination of 'precursors.' For example, the earlier epidemics of malignant cholera which visited Europe were believed to have been heralded by an unusual prevalence of 'fevers' and of diarrhœal affections. The epidemic of 1865-66 gave an excellent opportunity of studying the facts bearing on this question. Europe was taken by surprise when cholera appeared in 1865, on the south coast of the Mediterranean, at Alexandria, and began thence to extend rapidly to the southern and eastern shores of that sea. It was not, indeed, until the disease had effected a lodgment in several parts of the Continent that the attention of governments and the public with regard to it was fully aroused. No change of the public health in the several places visited by the epidemic had occurred of such a nature as to give rise to any, even the least, suspicion of impending pestilence. Moreover, notwithstanding the prevalence of steppe-murrain and of cerebro-spinal fever in Northern Europe at the beginning of 1865, there was nothing to suggest (indeed it was not suggested) that these phenomena were 'precursors' of the coming epidemic of cholera, or of the extension of yellow fever to the shores of England the same year. In fact, the different occurrences were parts of contemporaneous rather than successive phenomena. The condition of the public health preceding the appearance of cholera in England, and especially in respect to diarrhœa antecedents, was made the subject of careful study by the late Professor Parkes and others. These observers failed to obtain any inkling of a change in the public health which could be regarded as presaging pestilence—of the existence, in short, of any so-called 'epidemic constitution.' It is true that John Sutherland had described an increase of 'fevers' and diarrhœal disease in Malta, as preceding the appearance of cholera there, in June 1865, observing that: 'These facts are sufficient to show that, long before cholera began to come towards the north-west, there were indications of a changed condition of the public health in Malta;' and after giving an account of two groups of choleraic cases which occurred in May, he adds, 'It appears to me

scarcely possible to escape from the conclusion that, long before cholera appeared in Malta, possibly before the first outbreak in Arabia, the earliest wave of the coming epidemic had passed over those islands.' But while, according to Dr. Sutherland, the coming epidemic was thus clearly foreshadowed in Malta, no change in the public health presaged its appearance in Gibraltar in July. The absence of all foreshadowing of the epidemic at Gibraltar can hardly be reconciled with the suggested presence of such foreshadowing at Malta. The facts which have been interpreted as presaging the appearance of cholera in the latter island are, indeed, to be regarded as coincidental rather than related. It is true, also, that MM. Didiot and Gués have endeavoured to show that, prior to the appearance of cholera at Alexandria in 1865, choleraic disease existed in Marseilles, and that the outbreak of the epidemic in the latter town was preceded by 'une constitution médicale cholérique.' The evidence they advance in support of their conclusions simply shows that deaths from infantile cholera, which, as they properly remark, is, 'à la vérité, fréquente à Marseille,' and from so-called 'sporadic' cholera, are apt to occur in Marseilles during the months of May and June, as perhaps in every city and town of Southern and Central Europe.

Another illustration to the same effect as the above, in regard to the 'precursors' of epidemics, is furnished by the history of the small-pox epidemic of 1869-73, the greatest epidemic of this disease in recent times—a true pandemic (πάνδημος, from πᾶν, all; and δῆμος, the people) extension of the malady. The acutest observers were taken by surprise with the malignity and diffusibility of the disease during the epidemic—phenomena wholly unforeseen. Here, again, the study of the diseases preceding or accompanying the epidemic yields no facts of variation in their prevalence, from which the approach of a great epidemic might have been inferred, nor gives any hint that they were dependent with it on some common cause.

There were *concurrent* phenomena of disease during both epidemics, but they were concurrences of certain diseases existing at the same time among the population affected by the great epidemic, and some of them probably having certain secondary elements of causation in common. This is a different question to that of an assumed 'epidemic influence' or 'epidemic constitution.' The subject of the *concurrence of epidemics* is a new field of investigation, which has lately been opened by an arithmetical study of George Buchanan's, relating to epidemics in numerous extra-metropolitan registration sub-districts in England. This study was directed to ascertain—as necessarily introductory to a statistical investigation regarding community of causation or mutual antagonism of various

epidemics—the arithmetical probability of their concurring as a mere matter of chance. The data used were taken from a particular quarterly return of the Registrar-General, and they extended to 946 occurrences of epidemics (small-pox, measles, scarlet-fever, diphtheria, whooping-cough, fevers, and diarrhoea and simple cholera) in 1,490 districts. The result showed a good deal of general correspondence between the calculated number of concurrences and the actually observed number, but with departures, of more or less magnitude, of the actual from the calculated degree of concurrence, which deserve to be followed up by further investigation. The departures were shown in an excessive frequency of the concurrence of measles with the other epidemics, of diarrhoea with all other epidemics except small-pox, and of scarlet-fever with the 'fever' of the Registrar-General. On the other hand, it is of interest to observe that there was an absence of any notable excess of frequency of concurrence between scarlet-fever and diphtheria, or between scarlet-fever and whooping-cough.

In view of the extreme looseness with which the word 'epidemic' is used in medicine, sometimes simply as a descriptive term, sometimes as a technical or quasi-technical term involving various hypothetical and theoretical conceptions inconsistent with each other, and sometimes as implying an occult influence, it would be well if it could be discarded from medical literature and language. 'Epidemic,' in its present medical uses, is an instance of words which, as Bacon says, when writing of the effects exercised by a bad and inapt formation of words on the human mind, 'force the understanding, throw everything into confusion, and lead mankind into vain and innumerable controversies' (*Nov. Org. App.* 43). But the retention of the word, from its long and familiar usage, is practically a necessity in medicine; moreover, it would be difficult, if not impossible, to find a substitute which, if the word be used in its ordinary signification, would supply its place.

It is suggested here that the technical meaning of the word 'epidemic' should be assimilated to the common meaning; or, more accurately, that the technical meanings now attached to the word should be abandoned, and the word used in medicine in the same sense as in general literature and in ordinary converse; that is to say, as a merely quantitative term applicable to particular phenomena, whether pathological, mental, or social, in so far as they are 'common to a whole people, or to a great number in a community,' or, in a word, are 'prevalent' or 'general.' In this way not only would the confusion arising from the present medical uses of the word be got rid of, but the scientific study of epidemic phenomena would be facilitated in the only direction which gives promise of successful issue. As Léon Colin has aptly

written: 'It is the disease which constitutes the epidemic, not the epidemic the disease. The evil always remains the same, the number of affected alone being increased.' The medical study of epidemics is essentially a study of the individual diseases which are apt to become epidemic, and not, as has been too commonly the case hitherto, of some figment of the imagination (*epidemic constitution*, or *influence*, or *genius*), apart from the diseases. It is only in proportion as researches have been directed to particular diseases liable to become epidemic, and to the conditions under which they prevailed epidemically, that recent advances in our knowledge of epidemics have been made. In this country the two most important events which have occurred in this connexion of late years were the discrimination of *typhus* from *typhoid fever*, by A. P. Stewart and William Jenner, and the researches on the *typhus of horned cattle* (steppe-murrain, *cattle-plague*) promoted by the Royal Commission of 1865-66 on Cattle-Plague. The discrimination of typhus from typhoid fever proved that the voluminous speculations which to that time were current on the epidemiology of the continued fevers of this country, then regarded as but *one* disease presenting several varieties, were for the most part meaningless verbiage, by showing that the two most common forms of fever were distinct diseases, clinically, pathologically, and aetiologicaly. This discovery proved to be the inauguration of a true method of investigation concerning epidemics, by making evident that epidemic phenomena did not admit of accurate study, except in so far as it was based upon a just discrimination of the diseases manifesting them, and upon their clinical and pathological histories. The same lesson was taught, not less clearly, although in another fashion, by the researches promoted by the Royal Commission on Cattle-Plague, with which the names of Lionel Beale and Burdon Sanderson are especially connected. These researches demonstrated the essentially infectious nature of the malady, and that its prevalence was dependent upon the dissemination of the infection, directly or indirectly, from animals sick of the disease to the healthy. It was shown, indeed, in the pathological laboratory, that preconceived doctrines of some occult epidemic influence which had been submitted to the Commission as determining the prevalence of the cattle-plague had no existence in fact when the disease was subjected to experimental study, and that its conditions of prevalence were fully within human control. These researches proved the starting-point of those important investigations on the intimate pathology of contagion carried out by Burdon Sanderson and Klein, for the Privy Council, under the direction of John Simon, and to which William Roberts and others have independently contributed so

largely, and which have been followed by the investigations into the intimate pathology of scarlatina and diphtheria by Klein acting under the instructions of George Buchanan for the Local Government Board. See also PERIODICITY IN DISEASE.

J. NETTEN RADCLIFFE. SHIRLEY F. MURPHY.

**EPIDEMIC CEREBRO-SPINAL MENINGITIS.**—A synonym for cerebro-spinal fever. See CEREBRO-SPINAL FEVER.

**EPIDEMICS, Occurrence of.**—See PERIODICITY IN DISEASE.

**EPIDERMIS, Diseases of.**—See SKIN, Diseases of.

**EPIDERMOPHYTON** (*ἐπί*, upon; *δέρμα*, the skin; and *φυτόν*, a plant).—The name of the epiphyte, or parasitic fungus, of pityriasis versicolor; also called microsporon. See EPIPHYTA.

**EPIDIDYMITIS** (*ἐπί*, upon; and *δίδυμος*, a testicle).—Inflammation of the epididymis. See TESTES, Diseases of.

**EPIGASTRIC REGION.**—This region is situated at the upper and central part of the abdomen, just below the ensiform cartilage, and between the sloping margins of the thorax down to the level of the ninth cartilage, corresponding to what is popularly known as the 'pit of the stomach.' The structures within the abdominal cavity which normally occupy the epigastrium are the greater part of the stomach, a small portion of the liver, and more deeply a part of the pancreas, the aorta giving off the cœliac axis and superior mesenteric branch, the vena cava inferior, the veins forming the commencement of the portal vein, the receptaculum chyli, and the solar plexus.

**CLINICAL INVESTIGATION.**—Clinically, it will be found that patients frequently complain of abnormal sensations specially referred to the epigastrium. These are generally associated with the stomach, and may merely amount to a sense of discomfort, fulness, or tightness; or to actual pain of varying character, more or less severe according to the condition upon which it depends, and often much influenced by the ingestion of food. Sensations of trembling, throbbing, or sinking at the pit of the stomach are also of common occurrence, especially in females. These are often merely of a nervous character. In some cases there is evident tenderness, either over the entire epigastrium or in some limited spot, and it is important to recognise whether this is superficial or felt more or less deeply, for it may be connected with the superficial structures, the peritoneum, the liver, the stomach, or the pancreas. The sensation experienced in connexion with hunger is referred mainly to the epigastrium, and it may be of a painful character. Here may

also be noticed the epigastric pain termed *gastralgia* or *gastrodynia*, which is usually felt chiefly when the stomach is empty, being relieved by taking food. A most unpleasant sensation at the pit of the stomach accompanies nausea, which may be of a horrible but indescribable character; while violent vomiting or retching causes considerable pain or aching in this region, partly associated with the stomach, partly with the abdominal walls. Heartburn is another sensation which seems to start from the epigastrium. Sometimes the pain is situated deeply, or shoots towards the back. This may depend upon disease affecting the posterior wall of the stomach, pancreatic disease, aneurysm, or other causes. A sensation of tension or actual pain is sometimes experienced just below the ensiform cartilage in cases where the diaphragm is much pressed downwards, as from extreme emphysema, abundant accumulation of fluid or air in the pleura, or extensive pericardial effusion. A deep pain is not uncommonly referred to the epigastrium in cases of Addison's disease, and also in those of pernicious anæmia. Probably this is connected with the sympathetic plexuses.

*Physical examination* of the epigastric region is often of the greatest value, and important objective signs of various morbid conditions may thus be readily recognised. Of course organs which normally occupy other regions may enlarge so as to extend into the epigastrium, or may become very movable and consequently be felt in this region. It may, moreover, be occupied, along with other parts of the abdomen, by growths or accumulations of fluid. The objective signs and conditions which are more especially connected with the epigastrium may be indicated as follows:—

1. It is customary to apply the hand over the epigastrium for the purpose of counting the respirations, if these cannot be reckoned by merely watching the patient breathing.

2. Morbid conditions of the abdominal walls may be confined to the epigastric region, such as an abscess; and here it may be remarked that the recti muscles frequently become very hard and rigid when palpation is practised over this part, and so might be in danger of being mistaken for some serious lesion, unless care were exercised in the examination.

3. Growths connected with the peritoneum, especially the great omentum, may be felt chiefly or entirely in the epigastrium in some instances.

4. Abnormal states of the stomach are necessarily revealed mainly by corresponding signs in the epigastrium. Thus there may be evidence of dilatation of this organ; of carcinomatous infiltration or thickening of its anterior wall; of a localised tumour; or of some accumulation in its interior, whether solid or liquid. It must be remarked, however, that

the stomach, when diseased, frequently extends into other regions beyond the epigastrium, or is actually displaced.

5. When the liver is the seat of organic disease, this is often revealed in the epigastric region, usually along with other regions, but sometimes the abnormal physical signs are noticed specially in this part. Thus a cancerous mass may present here, or a hydatid tumour or hepatic abscess may tend in this direction. The gall-bladder has also occasionally been found, when the seat of some accumulation or of malignant disease, to have been displaced towards the epigastrium and become fixed there.

6. Pulsation is not uncommonly felt in the epigastrium. When situated at its upper part, just below the ensiform cartilage, it depends upon the heart, usually its right side, being due either to shortness of the sternum, displacement of the heart, or enlargement of its right cavities. Very often a pulsation is felt, and sometimes even seen, due to a pulsating aorta, especially in thin and anæmic subjects; and an impulse from this vessel may be transmitted through an enlarged pancreas, or through an abscess of the liver, of which the writer has seen a marked example. Occasionally an impulse in the epigastrium is connected with an aneurysm, either of the aorta or of one of its branches. A pulsation in this region has also been attributed to regurgitation of blood from the right auricle into the inferior vena cava and hepatic veins, in cases of tricuspid incompetency. A murmur may sometimes be heard in the epigastrium. Usually this is a conducted cardiac murmur, but occasionally it depends on an aneurysm.

FREDERICK T. ROBERTS.

**EPIGLOTTIS, Diseases of.**—See LARYNX, Diseases of.

**EPILEPSY** (*ἐπιλαμβάνω*, I seize upon).—**SYNON.**: *Morbus comitialis, sacer, major, &c.*; Fr. *Epilepsie, haut mal, &c.*; Ger. *Fallsucht*.

**DEFINITION.**—In its common form this apyretic affection is a neurosis, characterised by seizures of loss of consciousness, with tonic or clonic convulsions. When either of these manifestations recurs in fits, epilepsy exists, although incomplete, as shown by the following facts: first, that patients who generally have convulsions only are sometimes seized also with unconsciousness; secondly, that others who usually have seizures of loss of consciousness, without marked spasmodic action, sometimes have complete fits of epilepsy.

Some writers confine the name of *Epilepsy* to the well-defined form of that neurosis known as *idiopathic*. Others consider as belonging to epilepsy most of, if not all, the non-febrile affections recurring in fits, and of

which we sometimes have good instances in cases of paroxysmal aphasia, amaurosis, paralysis, or involuntary movements, either disordered or well co-ordinated, &c. There is no doubt that all apyretic nervous affections, appearing in seizures, have, on account of this special feature, a common link with epilepsy. In this article, however, the writer will only study the ordinary forms of the *morbus comitialis* and the epileptiform manifestations due to brain-disease.

**ETIOLOGY.**—Under this head we will study, first, what relates to the *causation of the morbid state* in which epilepsy essentially consists; secondly the *causes of attacks*.

**Predisposing Causes.**—*Heredity* deserves to be noticed first, not because it is the most frequent, but because it is a most undeniable cause. Pritchard has shown that all neuroses have the greatest relationship one to another. It is but natural, therefore, that epileptics are very often found to have had a father or a mother who suffered from some kind of nervous disorder. Moreau has shown that epileptics are often the offspring of insane parents. It is quite certain that people attacked either with organic disease of the brain, and having had no convulsions, or with any kind of neurosis or any form of insanity, very frequently have children who become epileptic. Important statistical data, given by Féré, show that, among the direct ascendants of 594 epileptics, 70 had suffered from epilepsy. 166 from insanity, 88 from paralysis, 21 from general paralysis, 72 from hysteria, 73 from senile dementia, 33 from puerperal eclampsia, 61 from chorea. The number of cases of nervous disorders among the direct ascendants of those 594 epileptics was 1,024. Inherited epilepsy, according to the writer's experience, which agrees with that of Féré, chiefly appears between the ages of 12 and 18; but its percentage remains high up to the age of 40, as, according to Dr. Gowers, out of 235 epileptics from 20 to 39 years old, 80 had inherited this affection, which gives 34 per cent. This shows how erroneous is the view of some able physicians, who maintain that after the age of 20 inherited epilepsy is not to be dreaded. It is certain, however, that this inheritance becomes relatively extremely rare in advanced life. But it can yet show itself at the ages of 70 (Poilroux) and 71 (Dr. Gowers). Still, we strongly advise that symptoms of an organic disease, and especially of a cerebral tumour, be most carefully sought for in all cases of supposed inherited idiopathic epilepsy in patients over 50.

The power of *alcoholism* in parents is so great that out of the 594 epileptics studied by Féré, 258 had parents addicted to habits of hard drinking.

As regards the influence of *sex*, there is a marked difference between men and women of somewhat advanced age, the proportion of

epileptic females being then larger than that of males; but for people under 25 years of age the reverse is true.

Independently of sex, *age* has certainly a decided influence on the appearance of epilepsy. The following table, borrowed from Hasse and Dr. Gowers, is important, as the results are grounded on most extensive statistical data. These results agree fully with the writer's own observations.

Age at commencement	No. attacked	
	Hasse	Gowers
Under 10 . . . . .	393	422
11 to 20 . . . . .	364	665
21 to 30 . . . . .	111	224
31 to 40 . . . . .	59	87
41 to 50 . . . . .	51	31
51 to 60 . . . . .	13	16
61 to 70 . . . . .	4	4
At 71 . . . . .	0	1
	995	1,450

The writer's experience seems to show that hereditary epilepsy makes its appearance chiefly between 10 and 12 in both sexes. At any rate it is certain that inheritance manifests itself rather before than during the period of change that puberty causes. Romberg, Dr. Russell Reynolds, and others have already pointed out that such is the general rule.

*Puberty* itself is a cause of epilepsy. This is clearly proved by the large number of epileptics who, as shown in the preceding table, have been attacked between the ages of 10 and 20 (364 out of 995, and 665 out of 1,450).

Of other causes, the most powerful are perhaps not those usually stated. In patients under 15 years of age the most frequent cause, after heredity, is some obscure alteration of nutrition of the brain, or congestion of that organ or of its membranes, lasting a more or less considerable time after typhoid fever or scarlatina. It is frequent, indeed, that in examining such patients (even when there are reasons to believe in heredity) there are a number of symptoms showing some deficiency in the action of the brain, as regards its motor, sensitive, or sensorial functions. It is most important to detect these symptoms, as the form of epilepsy due to or allied with the cerebral alteration then existing is often curable, or at least can be considerably benefited by treatment.

Alterations of *blood*, in quantity or quality, often contribute to the production of epilepsy. But, however true it is that a rapid and considerable loss of blood can cause an epileptiform attack, it has not been shown that such a cause has ever produced a lasting epilepsy. It is nevertheless well known that anæmia is a very frequent factor in the causation of that neurosis or of any other. A weak and slow action of the heart also is found in a number of cases to be among the causes of epilepsy (Sir W. Burnett, Blondeau). But

the reverse is sometimes observed; and, according to the writer's practice, cases of epilepsy are not very rare in which the pulse was strong and beating more than 90 or 100 times a minute, without any organic affection that could account for this phenomenon. In these cases the only morbid condition that could be looked upon as a cause of epilepsy was the abnormal circulation, which may be remediable.

There is no doubt that, when epilepsy appears in cases of Bright's disease, of affections of the liver or other viscera, it is due, at least in most cases, to the presence of some toxic agent in the blood; but other aetiological factors exist sometimes. Among them one especially deserves attention—it is an irritation of the nerves of the diseased viscus.

*Secondary syphilis* is a frequent cause of epilepsy. Undoubtedly syphilis then acts chiefly through the alterations it produces in the brain and meninges; but it often acts also on the cranial periosteum, and in disturbing nutrition and sanguification.

Of other dyscrasie *gout* has very little power; but *rheumatism*, although not frequently, can cause epilepsy by its morbid influences on the brain and its membranes, and by the production of diseases of the heart and the great blood-vessels.

*Diabetes* has been classed among affections sometimes causing epilepsy, but the question is then whether it is the presence of sugar in the blood or some affection of the medulla oblongata which brings on this affection.

Oxide of carbon, carbonic acid, lead, absinthe, alcohol, and a number of other *toxic agents* have sometimes produced a lasting epilepsy. Asphyxia by strangulation was in one case the cause of epilepsy (A. Beardley).

Purely *psychical* or emotional causes have much less power than has been supposed. But their influence is great in bringing on attacks when epilepsy exists.

*Determining Causes.*—The most frequent causes of epilepsy, however, are not the preceding, which generally are only predisposing circumstances. The really active efficient causes of epileptiform convulsions, and even sometimes of genuine and complete *morbus comitialis*, are to be found in the four following groups of organic morbid states:

(1) Lesions of some parts of the *brain*; (2) morbid conditions of the *meninges* or of the periosteum of the cranium; (3) lesions of the *spinal cord* or its membranes; (4) *peripheral* lesions anywhere. It is only for convenience sake that the writer establishes these divisions, as he looks upon all the four groups as quite alike. In neither of the four kinds of cases is the location of the lesion the seat of the neurosis, that place being only the seat of an irritative cause. Each of these kinds of causes will now be studied, the first one only with some development.

1. *Lesions of the brain considered as causes of epilepsy.*—This subject must be subdivided as follows: (a) parts of the brain that give rise to that affection; (b) kinds of brain-lesion which bring on that neurosis.

(a) What are the *parts* of the brain which can produce epilepsy? Facts answer that all parts have that power, as will be proved hereafter. On account of the importance of this subject as regards diagnosis and therapeutics of the nervous centres, the writer has noted down for more than twenty years all that relates to convulsions out of many thousands of cases of cerebral disease. Unfortunately, for a long time not having counted the cases without convulsions, he cannot say out of how many cases of brain disease come the 1,688 cases of convulsions he has collected.

The first important result of these researches is that partial or unilateral convulsions are more frequent than bilateral or general convulsions. Out of the 1,688 cases there are 887 in which the convulsions were altogether limited to one or two limbs, or began in one side before reaching the other, or were more violent in one side than in the other; while the number of general convulsions was 801.

The 887 cases of unilateral convulsions can be classified as follows:—

Side of Convulsions		Side of Lesion
Right . . .	102 . . .	Right
Left . . .	90 . . .	Left
Right . . .	294 . . .	Left
Left . . .	247 . . .	Right
Right . . .	73 . . .	Two sides
Left . . .	81 . . .	Two sides

As seen in this table, there were 192 cases of direct and 541 of crossed convulsions, with 154 cases of unilateral convulsions due to lesion in the two sides.

Of the 801 cases of convulsions in both sides of the body, there were 525 due to a unilateral lesion and 276 caused by a lesion in the two sides of the brain. In the 525 cases, the lesion in 235 was in the right side, and in 290 it was in the left.

It is remarkable that, contrary to what was long believed by the writer and other authors, the number of cases of convulsions (partial or general) due to a lesion in the right side of the brain is notably less frequent than that of cases of lesion in the left, as shown in this table:—

Side of Lesion	Convulsions	
Right	Left	
235	290 . . .	Two-sided
247	294 . . .	Crossed
102	90 . . .	Direct
—	—	
584	674	

It is very remarkable also that convulsions do not take place much more frequently on the right side of the body than on the left,

the numbers being for the right 469 cases, for the left 418 cases.

As regards the parts of the brain where a lesion is found, it is not possible to make use of all the facts, because, when a lesion is in the two sides, it very rarely occupies similar parts in both; and also in a rather large proportion of cases the lesion occupied two or more parts of the brain. The following table gives the number of cases for lesions in all the important parts of the encephalon:—

1. So-called motor centres . . . . .	196 cases
2. Cortex cerebri out of the motor zone	173 "
3. Surface or internal parts frontal lobe	144 "
4. " " " parietal "	140 "
5. Base of the brain . . . . .	91 "
6. Surface or internal parts of occipital lobe . . . . .	89 "
7. Cerebellum . . . . .	57 "
8. Surface or internal parts sph. temp. lobe . . . . .	54 "
9. Centrum ovale . . . . .	52 "
10. Optic thalamus . . . . .	42 "
11. Corp. str., opt. thal. & internal capsule	25 "
12. Corpus striatum . . . . .	24 "
13. Lateral ventricle . . . . .	21 "
14. Cornu Ammonis . . . . .	7 "
	1,115 "

It is essential to remark that the number of cases of lesion of the cortex cerebri in the so-called motor zone or out of it (369 out of 1,115 cases—*i.e.* about 33 per cent.) is possibly greater than it should have been, owing to the probable co-existence, in some cases, of a meningeal affection with the lesion of the brain-tissue. The writer must say that he has eliminated as completely as he could from the cases he has collected of disease of the cerebral surface those in which there was a complication of meningitis. If we add to these 369 cases one-half of those of disease of the four cerebral lobes (214 out of 427)—which ought to be done, because in more than one-half of these last cases the surface was partly the seat of the lesion—there are 583 cases of convulsions from disease of the cortex cerebri (583 out of 1,115—*i.e.* about 52 per cent.) The cerebral convulsions have more power than the rest of the brain to give rise to convulsions, but the cortex, outside of the so-called motor zone, has at least as much power as that zone.

Concerning the parts of the brain considered as not motor, we find that there are 89 cases of lesion of the occipital lobe and 54 of the spheno-temporal lobe, giving a total of 143 cases; to which we have to add the 173 cases of superficial lesion out of the motor zone; so that there are 316 cases of convulsion from a lesion in parts which, according to many authorities, ought not to have produced such a symptom. In more than one-half of the cases of disease of the cerebellum there was no pressure on the base of the brain, so that we could add a good many of the 57 cases of lesion of that nervous centre to the 316 cases above mentioned.

It would be wrong to imagine that, because the number of cases of convulsions was much larger when disease existed in the parietal lobe (140 cases), that that part has more power to originate epileptiform contractions than the occipital lobe (89 cases). The reverse is true, because, in opposition to all that is taught on this point, this last lobe has more power than the parietal in that respect (if we leave aside what relates to the ascending parietal). This can easily be proved. The number of cases of organic disease (especially hæmorrhage, as one can see in Gintrac's great work containing 127 cases of hæmorrhage in the parietal lobe and only 33 in the occipital) of the parietal lobe is more than treble that of cases of lesion of the occipital; while the number of cases of convulsions due to disease of the first of these parts not only is not three times as large as that of cases of convulsions due to the occipital lobe, but is even far from being the double. This last part, therefore, although supposed not to be motor, has more influence than the parietal lobe in giving rise to epileptiform convulsions.

Taking into account the frequency of disease in the four cerebral lobes, the number of cases of lesions which in each of them generate convulsions, shows that the frontal lobe is the one which has the greatest influence as regards the production of epileptiform convulsions. Next to it comes the occipital lobe, and, after these two lobes, we have to place almost on a similar level the parietal and the temporo-sphenoidal lobes.

Considering the frequency of disease, and especially of hæmorrhage, in the corpus striatum, which has been supposed to have a great influence in producing epileptiform convulsions, it is remarkable that that part of the brain has given rise to convulsions only 25 times (hardly more than 2 per cent.) The percentage given by the optic thalamus in that respect is higher (3.6 per cent.)

All the internal parts of the brain (centrum ovale, the lateral ventricles, and the central ganglia) give only 164 cases (15 per cent.), not much more than the frontal lobe (144 cases, almost 13 per cent.), although the central parts of the cerebrum are far more frequently diseased than the anterior lobe.

(b) There is certainly a very great difference between the various *kinds* of disease of or injury to the brain with regard to their power of giving rise to genuine epilepsy or to epileptiform convulsions. Still, it is very difficult to draw positive conclusions out of what has been published on this point, because the subject has not been studied as it should have been. Certain parts of the brain will give rise to convulsions, while others will not, under one and the same kind of lesion; but a part like the frontal lobe, for instance, which will so often generate genuine epilepsy or epileptiform convulsions under causes such as an injury, a tumour, an abscess, or an

inflammatory softening, will rarely produce convulsions under a cause which is an injury, but of a peculiar kind—*i.e.* a hæmorrhage. In other parts, on the contrary, a hæmorrhage will most frequently give origin to convulsions. The following table, from data furnished by Gintrac, is interesting in that respect; it shows what has been the frequency of convulsions in cases of hæmorrhage:—

Cases of hæmorrhage	Cases of convulsions
43 in cortex cerebri . . . . .	11
127 " parietal lobe . . . . .	25
33 " occipital " . . . . .	7
17 " frontal " . . . . .	2
38 " optic thalamus . . . . .	20
72 " corpus striatum . . . . .	0
48 " both central ganglia . . . . .	5
46 " lateral ventricles . . . . .	14
56 " cerebellum . . . . .	10
70 " pons Varolii . . . . .	25
5 " crus cerebri . . . . .	2
555	121

It is a most remarkable fact that out of 38 cases of hæmorrhage in the optic thalamus there were convulsions in 20—*i.e.* more than one-half of the cases; while there were none in any of the 72 cases of hæmorrhage in the corpus striatum. It is perhaps more remarkable that when both ganglia were the seat of the effusion of blood there were only 5 cases of convulsions out of 48 of hæmorrhage.

Tumour and tubercle are, with hæmorrhages and inflammatory softening, the lesions of the brain which give rise most frequently to partial or general convulsions. While tumours of the frontal lobe produce epilepsy or epileptiform convulsions in more than two-thirds of the cases, tumours of the temporo-sphenoidal lobe give rise to convulsions in about one-half, and tumours of the parietal and the occipital lobes in only about one-third of the cases. A remarkable fact which comes out of the study of cases of tumour of the brain published by Ladame, Bernhardt, Gintrac, and others, is that the base of the brain under the irritation of tumours gives rise to convulsions very much less frequently than the surface of the brain. Indeed, one part of the brain—the medulla oblongata—did not originate convulsions in one single instance out of 21 cases of tumour published by Bernhardt. This kind of disease of the medulla oblongata is not, however, incapable of giving rise to epileptiform attacks, as the writer knows of five cases of tumour of that organ causing convulsions.

There is no kind of lesion which may not give origin to convulsions. But there is one species of tumour, namely, aneurysm, which causes convulsions considerably less frequently than any other. Out of 86 cases of aneurysm of the various encephalic arteries, given by Gintrac, there were convulsions only in three, if we put aside, as ought to be done, the cases in which there

were alterations in brain-tissue or a rupture of the aneurysmal sac.

Abscesses of the brain have more influence in the causation of epileptiform convulsions than is generally admitted. Out of 87 cases of unilateral abscess collected by the writer, convulsions occurred in 43 cases.

Unilateral atrophy of the brain, of a very old date as regards its origin, is known to be allied not rarely to epilepsy (partial or general). Summing up 78 cases given by Cotard, Boyd, Turner, and Gintrac, the writer finds that epileptiform convulsions or genuine epilepsy existed in 20 cases. It is of great importance against the views of those who try to locate epilepsy in the so-called motor centres, that not only those parts were absolutely destroyed in more than one-half of those 20 cases, but that the so-called motor track in the brain and spinal cord was deeply altered if not destroyed, at least in certain parts of its length, and that nevertheless convulsions occurred, either anywhere, or chiefly or exclusively in the paralysed limbs.

Softening due to embolism causes convulsions almost as often as inflammatory softening, except when the seat of the lesion is the cortex cerebri. White softening of vessels of the occipital and of the frontal lobes produces convulsions more frequently than the same lesion in the parietal lobe. Embolism without any marked softening very rarely produces convulsions.

All kinds of entozoa in the brain very frequently give rise to convulsions, but none so often as cysticerci, if Kuchenmeister is right. He states that out of 30 cases of cysticerci in the encephalon there were 24 cases with convulsions. No other cause of irritation, whatever be its seat in the brain, originates convulsions so often as that.

The application of a ligature on one of the carotid arteries has brought on convulsions in a number of instances; and, what is remarkable, when the convulsions were unilateral they appeared on the corresponding side in more cases than on the opposite side.

2. *Lesions of the meninges considered as causes of epilepsy.*—The reader may refer to the articles on the Diseases of the Meninges and Diseases of the Cranium for what relates to convulsions due to these morbid states. Complete and persistent epilepsy due to chronic meningitis is very rare; but syphilitic affections either of the dura mater or of the periosteum of the cranium not very rarely will give rise to this affection. See MENINGES, CEREBRAL, Diseases of; and SKULL, Diseases and Deformities of.

3. *Lesions of the spinal cord considered as causes of epilepsy.*—In the writer's work on Epilepsy he has shown that this affection can arise from disease of the spinal cord. Since 1857, when that work appeared, many facts have come to his knowledge establishing that his conclusion was right. He only

maintained—and he does not now go beyond that assertion—that epilepsy sometimes has its cause in the spinal cord.

4. *Peripheral lesions*.—That genuine epilepsy can have its cause in any part of the body is now a recognised truth. But, besides that form of the *morbus comitialis*, what may be called the cerebral form, consisting in unilateral convulsions, can be originated by an irritation of either a nerve or a viscus. Among the parts of the body having the greatest power with regard to the production of epileptiform convulsions, we may name the bowels, the trigeminal and the sciatic nerves, the abdominal sympathetic nerve, and the sexual organs. Indigestible food may be the cause of an attack. In countries chiefly inhabited by the Anglo-Saxon race, children of the two sexes being less watched and less warned than elsewhere against the dangers of onanism, epilepsy due to this fatal habit is particularly frequent.

The great importance of a thorough knowledge of certain symptoms of cerebral epilepsy, especially as regards surgical treatment, makes it necessary that these symptoms should be carefully studied. It must be first stated that every kind of organic brain disease, wherever situated, may cause any form of epilepsy, and that, on the other hand, every form of this neurosis may exist without any cerebral organic affection. To this must be added that any part of the brain may be the seat of any kind of disease without convulsions appearing anywhere. In more than two thousand cases of brain disease analysed by the writer for other purposes, there were no convulsions. Out of that number there were more than one hundred cases of simply irritative lesion of the so-called motor centres, limited to them or invading the neighbouring parts, in which there were various symptoms, but no convulsions. In more than 160 cases of disease (destructive and irritative, or simply irritative) of the base of the brain there were no convulsions. Simply irritative lesions of the so-called motor tract, the internal capsule, between the great cerebral ganglia, did not cause convulsions in more than 45 cases. The part which gives the smallest number of cases without convulsions is the optic thalamus, and the part most able to bear any kind of irritation without producing convulsions is the corpus striatum.

These *negative* facts show as well as the *positive* ones—*i.e.* those in which convulsions take place—that that symptom appears or not according only to the excitability of certain parts of the nervous centres in different individuals. This result is borne out also by the study of unilateral convulsions, as is shown by an analysis of 887 cases collected by the writer.

The conclusions to be drawn from the study of these cases can be summed up as follows: (1) These convulsions can be produced by a

lesion in almost any part of the brain; (2) they can be caused by any kind of lesion; (3) they can begin in any muscle or group of muscles, or simultaneously in every part of one-half of the body (eye, face, neck, trunk, and limbs); (4) they can be preceded by any kind of aura, local or general; (5) they can be associated with any other symptom of brain disease, or be, for some time or till death, the only existing cerebral symptom; (6) they often precede general convulsions or appear at the end of such an attack; (7) they usually last longer than general convulsions even due to brain disease, but they especially last much longer than seizures of idiopathic epilepsy; (8) like other convulsions due to an encephalic affection, they often appear without loss of consciousness at the beginning or during the whole time they last; (9) they can take place either on the side of the brain-lesion that causes them or on the opposite side; (10) the proportion of cases of direct convulsions to cross ones, when the lesion is in the cerebrum, is about one to three, but the direct convulsions are more frequent than the cross ones, when that cause is in the base of the brain (crus cerebri, pons, and medulla); (11) the right limbs are more frequently attacked than the left (right 396 cases, left 337), and this rule is true for direct as well as for cross convulsions; (12) in the same individual, even when there is but one lesion, they can appear at first on the side of the lesion and afterwards on the opposite side, or *vice versa*; (13) they can occur on the side where exists a cross hemiplegia or on the opposite side; but they can also be direct, the paralysis also being direct; or they can be cross, the paralysis being direct; (14) Jacksonian convulsions (either when exclusively and persistently unilateral or only temporarily so) can appear on the side of the lesion, or from a lesion in parts of the brain belonging neither to the so-called motor zone nor to the first and second frontal convolutions, which Dr. Hughlings Jackson now supposes to be psycho-motor.

On account of the importance of Jacksonian convulsions for surgical treatment, it must be said that, as stated in the last of the above conclusions, they may appear, as shown by a number of cases, on the side of the lesion. In a case of that kind the surgeon, by pressing on the dura-mater on the *right* side, increased convulsions of the *right* limbs (Leroy).

Many cases show that surgeons led by the views of localisers might make great mistakes. For instance, in a typical case of Dr. Hughlings Jackson's, instead of the toes it was a finger which was first convulsed, although the lesion was in the paracentral lobe; in a case of Andral's, Jacksonian convulsions limited to the right arm were caused by a tumour between the left posterior lobe and the cerebellum; in a case of Prof. Virchow, Jacksonian convulsions beginning

in the left leg were due to a tumour of the right posterior lobe; in a celebrated case of Moreau (De Tours) there was a notable aura in the left hand and convulsions of the left arm due to a cyst in the anterior lobe near the right corpus striatum and the lateral ventricle. A good many facts show that Jacksonian convulsions, although caused generally by lesions of the first and second frontal or the two ascending convolutions, can be produced by lesions in many other parts of the brain very far from those convolutions.

Of the 887 cases of unilateral convulsions analysed by the writer, the arm or the leg alone, or one of these limbs at times, the other in other attacks, were the parts affected in 381 cases. The following table shows what parts of the brain were the seats of the lesion producing these partial convulsions.

	Arm	Leg	Leg or arm
Motor centres . . . . .	81	12	1
Surface frontal lobe . . . . .	17	1	1
„ parietal lobe . . . . .	13	1	2
„ sphen. temp. lobe . . . . .	8	1	0
„ occipital lobe . . . . .	12	1	0
„ several lobes . . . . .	22	2	2
Almost whole hemisph. . . . .	7	0	1
Frontal lobe . . . . .	16	1	0
Parietal lobe . . . . .	15	0	1
Sph. temp. lobe . . . . .	9	0	0
Occipital lobe . . . . .	10	1	1
Centrum ovale . . . . .	20	1	0
Lateral ventricle . . . . .	9	2	0
Two central ganglia . . . . .	5	2	0
Optic thalamus . . . . .	8	4	1
Corpus striatum . . . . .	4	2	1
Base of brain . . . . .	16	5	0
Cerebellum . . . . .	10	2	0
Several parts . . . . .	33	1	0
	<hr/>	<hr/>	<hr/>
	315	39	11

This table shows that the so-called motor zone and the rest of the cerebral cortex gave rise to a very large proportion of convulsions of one limb alone. There were 153 cases of convulsions of the arm, 18 of the leg, and 6 of leg and arm alternately. It will also be seen that the base of the brain, if it gave a small proportion of cases of convulsions of the arm, gave a large one of convulsions of the leg. But the most remarkable point is that *all parts of the brain can give rise to convulsions of the arm, and that most parts can produce convulsions in the leg.*

Most important conclusions can be drawn from the study of the percentage of cases of convulsions of the arm in the number of cases of unilateral convulsions. Thus it can be easily ascertained that the percentage is enormous—82 per cent.—for cases with a lesion of the sphenotemporal lobe; it is also very large for cases of lesion of the surface of the occipital lobe (50 per cent.); of the sphenotemporal lobe (47.6 per cent.); and of the frontal lobe (45.9 per cent.). Next come the so-called motor centres (45 per cent.), the mass of the occipital lobe (40 per cent.), the centrum ovale (37.7 per cent.), the cere-

bellum (37 per cent.), the parietal lobe (35 per cent.), and the frontal lobe (33.3 per cent.) A lesion of almost the whole brain (19 per cent.) and of the base of the brain (23.8 per cent.) give the smallest percentages.

As regards the leg, the largest percentage is furnished by the cerebral ganglia, next to which come the base of the brain, the cerebellum, the surface of the sphenotemporal and occipital lobes, and the so-called motor centres. It is evident from the facts relating to the leg, but still more from those concerning the arm, that great mistakes in the diagnosis of the localisation of disease might be made if convulsions of the leg or arm were looked upon as a proof that the cerebral lesion causing them is located in the motor zone, even if we include in it, according to Dr. Hughlings Jackson's views, the two first frontal convolutions.

Convulsions from brain disease may be alternate—*i.e.* they may affect the limbs on one side and the face on the other—and this owing to a lesion of either one or both sides of the cerebrum or the base of the brain.

It is important to know that unilateral convulsions are sometimes due to a peripheric irritation (in the bowels, the liver, the kidney, &c.), without any kind of disease existing in the brain.

There is no doubt that convulsions of one arm are often associated with spasmodic movements of the face; but there is no doubt also that the most heterogeneous associations of partial convulsions are sometimes witnessed: convulsions in one leg and in one eye, of the thoracic muscles and the face or of the neck and legs, and a great many other associations, from lesions which cannot give the key of these singular facts. The two arms or the two legs are not rarely affected together, even under the influence of lesions of only one side of the brain.

The kind of association between paralysis and convulsions is most interesting. French surgeons, till a somewhat recent period, gave it as a law that when convulsions from brain disease occur without paralysis they show themselves on the opposite side to that of the cerebral lesion; but that when there is paralysis, this symptom appears on the side opposite to that of the lesion, and convulsions occur on the corresponding side. This is the reverse of what is seen in the great majority of cases, in which paralysis and convulsions coexist on the same side. But there are two kinds of cases in which the convulsions are on one side and paralysis on the other. The writer has collected more than 200 cases in which the convulsions were on the side of the lesion, and paralysis on the opposite side; and 16 cases in which the contrary existed—*i.e.* convulsions on the opposite, and paralysis on the corresponding side. Here also we have a proof of the great variety of effects that can come from an irritation of the same or of various parts of the brain.

Unilateral convulsions have no essential connexion with psycho-motor centres, as is proved by cases of lesion of those centres on one side causing that kind of convulsions sometimes in one side of the body, sometimes in the other. The writer has seen two such cases and collected 42 other cases, some from most able observers, including Dr. Hughlings Jackson.

Many cases of epilepsy in man are on record, resembling the special kind seen in certain animals, particularly in this respect, that an attack can easily be produced by a peripheric irritation. The writer has collected more than sixty such cases. They form two principal groups—one in which an irritation of almost any part of the body was able to give rise to an attack; the other in which certain parts only had that power. Many of those cases belonged to the class of organic cerebral epilepsy; some depended on diseases of the pericranium, of the spine or its contents, of nerves or of one of the thoracic or abdominal viscera; while in other cases there was a genuine idiopathic epilepsy. The parts of the body which are most frequently found giving rise to an attack, are the pit of the stomach (under the influence of even a very slight pressure) and the skin of the neck laterally from the lower jaw to the collar-bone.

**SYMPTOMS.**—We shall consider this part of the subject under three heads—the *premonitory*, the *paroxysmal*, and the *inter-paroxysmal* symptoms.

**1. Premonitory.**—The frequency of premonitory symptoms, according to the writer's experience, is much greater than is generally known. It is extremely important to find out the existence of these warnings, as in many cases attacks may easily be prevented if we know when they are on the point of taking place. When we say that premonitory indications are extremely frequent, we do not mean that the classic or *Galenic aura* is often found. That vague, queer, and unexplained sensation, whether accompanied or not by a muscular contraction, is certainly more frequent than is admitted, but is considerably less frequent than the other kinds of warning. Among the premonitory symptoms there are four oftener observed than others: one is a change of temper (irascibility, appearing or increasing); another is a contraction of the vessels in the feet or hands, producing a diminution of temperature; and the two others are a spasmodic state of some muscle, or an optical illusion or hallucination. The following list mentions but a part of the various manifestations which indicate that an epileptic attack is threatening. Alterations of the various functions of the brain, sensations of any kind, headache or backache, vertigo, sensorial disturbances, sleeplessness or sleepiness, palpitation of the heart, dilatation or contraction of blood-vessels anywhere,

altered breathing, diminution or increase of the various secretions (of the skin, mucous membranes, or of the visceral glands), hæmorrhages from the nostrils or other parts, fever, more or less marked weakness (general or local), hunger or thirst, disgust for food or drinks, an increase of the sexual appetite or the diminution or loss of sexual desire or power, spontaneous erection, and spasm of the bladder—sometimes accompanied by involuntary evacuations of urine—involuntary expulsion of feces, cramps, trembling, choreic movements, tendency to run forwards, backwards, or round, rigidity or convulsions of a limb or other parts, paralysis of a limb or other parts—such is an abbreviated list of the forerunners noticed by a number of observers, and all of which the writer has seen or knows to have occurred in his patients.

Sometimes one or several of these symptoms will appear a day or two before the attack, but generally the warning shows itself a few hours, or a very much shorter time (even only a few seconds or a minute or two), before the seizure. Not rarely in cases of epilepsy due to an organic cerebral disease, or to cerebral congestion, a drawing of the head towards one shoulder will take place either before or during an attack. Premonitory symptoms unfortunately may be deceptive, as they can appear without being followed by the attack; and even in cases in which warnings usually exist there are sometimes attacks without any forerunner.

**2. Paroxysmal.**—The paroxysmal symptoms vary considerably according to the kind of attack. If the fit is one of *petit mal* (*epilepsia mitior*), there may be no other symptom than a loss of consciousness, with either a fixed state of the muscles of the eye or a slight contraction of one or more muscles of the face or neck, or a movement of the lips, tongue, and throat, as in the act of swallowing. According to his personal observation, the writer is inclined to affirm that an attack of *petit mal* never consists in a loss of consciousness only—*i.e.* without the least trace of any other trouble whatsoever. Dr. Russell Reynolds, however, states that he has seen such attacks, and therefore we must admit that sometimes a pure and simple loss of perception and volition is all that exists in a seizure of *epilepsia mitior*. In such an attack the patient may, if walking, continue to walk; but, if talking, he stops—generally for so short a time, however, that the trouble may pass absolutely unnoticed by listeners. Such an attack usually lasts only from one to four or a few more seconds.

**Haut Mal.**—In a complete attack of epilepsy, an irritation proceeding from a moral or physical source is the cause of an act of inhibition of the mental activity, and of a reflex contraction of blood-vessels in the cerebral lobes. Loss of consciousness is, of

course, the consequence of that inhibition. Together with Kussmaul and Tenner, the writer had long looked upon spasm of cerebral vessels as the cause of the loss of consciousness. He had a confirmation of that supposition in his epileptic guinea-pigs, in which he found that the blood-vessels of the brain contracted when consciousness disappeared. Since then, however, he has ascertained that consciousness was lost in the seizure, even after the excision of the upper cervical ganglia of the two sympathetic nerves—when, of course, the cerebral vessels remained dilated before, during, and after the fit—just as it is lost in animals having contraction of these vessels. Dr. W. Alexander has observed that in man, loss of consciousness in fits occurs after the excision of these ganglia, the same as before that operation. It is evident, therefore, that a spasm of the vessels of the brain is not an essential element in the causation of the loss of mental faculty. Still, when that spasm exists, and so long as it lasts, the lack of blood in the cerebral hemispheres must necessarily contribute to the cessation of the mental activity. The reasons for admitting that an act of inhibition is the first or principal cause of the loss of consciousness will be found in the writer's published papers on this subject.

Muscular spasms of a tonic character begin at the same moment that consciousness disappears. They may begin first in any part of the body, whether the attack is to be general, or to remain in one half of the body, or in one limb. Rather frequently the muscles of the face, the eyes, and the neck are seized before those of other parts, but the time is so very short between the appearance of spasm in these muscles and the general stiffness, that it is almost always very difficult to detect that these phenomena are not simultaneous. Together with the first appearance of a spasmodic condition in the face and neck there is also in a great many cases a contraction of some muscles of the larynx and of the expiratory muscles of the thorax and abdomen, producing the so-called epileptic *cry*, the character of which is so special that, when once heard, it can always be recognised even when the patient is not seen. When an epileptic is standing, at the onset of a complete attack, he falls. This is not due solely to loss of consciousness, as we know that in fits of *petit mal* this accident may not occur. The fall is sometimes an *active* one: the patient is precipitated more or less violently forward, backward, or laterally by muscular contractions. Then the fall may be either a *paralytic* or a *convulsive* one.

Generally a period of rigidity exists before the clonic convulsions appear. This stage of tonic spasm almost always lasts only a few seconds; but it may constitute the whole attack, and then it may last longer. On the contrary, clonic convulsions may appear

at the onset of the seizure. In almost all cases, however, they follow a general stiffness, and form a second stage or period of the fit of which by their violence and duration they are the principal part. The clonic spasmodic contractions as a rule last hardly more than from one to three minutes in idiopathic cases.

When a fit lasts five, six, eight, or ten minutes, even if there be no sign of an organic disease of the brain, there is some reason to suspect that the attack is brought on by such a cause. Generally, in complete seizures, the limbs on the two sides of the body have similar convulsions, but in cases of brain-disease there may be clonic convulsions on the one side and tonic rigidity on the other. In the face, the eyes, the neck, differences between the sides are frequent, or at least not rare, even in idiopathic epilepsy. In most cases, the eyes are drawn up under the upper eyelid, and they roll from side to side. It is not rare that the eyes, face, and head turn convulsively towards the left or the right side, sometimes jerking, however, alternately from one to the other side. When the head is carried sideways—to the right, for instance—it is chiefly owing to a spasm of the left sternocleido-mastoidens, but also to the group of muscles of the top of the neck on the right side.

Breathing is considerably interfered with during the whole attack. After the spasmodic action of the expiratory muscles contributing to the shriek, there is almost always for a time (not longer usually than one or two seconds) a stoppage of respiration from the persistence of that initial spasm. During the whole fit respiration is convulsive, irregular, jerky, or gasping, and always incomplete. A real asphyxia, going as far as cyanosis, soon shows itself. The face, which was pale at the onset, becomes red, and promptly assumes a livid, dark, purple or bluish-violet hue. A frothy saliva, frequently of a reddish tinge, from effused blood coming from the biting of the tongue or lips, issues, little by little, from the mouth. It is extremely rare that the movements of the heart are modified at the onset; but palpitation, or a diminution (and even a complete inhibition) of the cardiac movements has been noticed by the writer in a few instances. Soon, however, the heart becomes very much excited and beats quickly, as in ordinary asphyxia. The temperature rises ( $1^{\circ}$  or  $2^{\circ}$  F. and sometimes a little more); and the whole trunk, the face, and limbs, are covered with perspiration. The pupils always dilate considerably, sometimes after an evident contraction; the eyes are bloodshot; and hæmorrhage may occur there or in other parts, such as the nostrils, the ears, the bronchi, or even the capillaries of the skin or of the brain. Urine may be expelled from a spasm of the bladder at the onset, or from paralysis of the sphincter at the end of the fit.

As a rule, at the end of the convulsions the patient passes, without recovering consciousness, into a heavy sleep, with more or less violent stertorous breathing. A real comatose state is not rare. There is no doubt that these symptoms are more intense if no care be taken as regards the tongue and head. The patient should not be left on his back; and the tongue, which falls and covers the laryngeal opening, should be drawn forward. Some alleviation is almost always obtained when this is done for the tongue, and when the body, and especially the head, are placed on one side. Generally the sleep following a paroxysm does not last much more than an hour, sometimes not as long as that; but it is exceedingly rare that there is no sleep at all. The more violent the convulsions have been, the more there is relaxation of the muscles after they have ceased. When lifted up, the limbs may fall as if they were completely paralysed.

On waking from the sleep following a seizure most patients are very tired, the limbs and trunk aching, as well as the head. Usually there is some mental alteration, often consisting of confusion or stupor, and sometimes delirium. The mind, however, may be quite clear, even after a violent attack, and the head free from pain, the only effect of the fit being general lassitude. On the contrary, after an apparently slight seizure there is sometimes considerable mental disorder. The degree and duration of stupor after an attack have no relation to the duration of the convulsive period. Stupor is chiefly an effect of asphyxia, and is therefore in direct relation with laryngismus, trachelism, and the spasms of the thoracic muscles, of the diaphragm, and perhaps of the bronchial tubes also. It has been stated, and denied, that the urine passed after an attack of epilepsy sometimes contains albumen in patients free from kidney-disease. In at least five cases the writer has ascertained that there was a notable amount of albumen in the first issue of urine after attacks in which there had been violent spasmodic contractions of the abdominal and thoracic muscles. There was no disease of the kidneys or of the heart in those patients.

Attacks of epilepsy are sometimes very slight, consisting only, besides the loss of consciousness, of an extremely short tonic spasm of muscles of the trunk, the neck, the head, and the limbs. But even in the shortest and slightest attack of that kind the epileptic cry may be uttered, the tongue may be bitten, and there may be a somewhat comatose sleep after the fit.

It is now very well known that frequently there is after a fit a real paralysis, which sometimes persists for a long time. This loss of power is supposed to be the result of exhaustion from discharge of nerve-force, while it is evidently the effect of an act of inhibi-

tion associated with the convulsions—inhibition striking other motor elements than the excitation which causes convulsions. If it were an exhaustion from over-action the following facts could not exist: (1) the paralysis may appear after attacks which are not violent, and not show itself after most violent and prolonged convulsions; (2) there may be a paralysis in one or two limbs after an attack of *petit mal*, in which no convulsion has taken place in those parts; (3) after equal convulsions on the two sides of the body there may be a paralysis of one side only; (4) after unilateral convulsions there may be a paralysis of one limb only (especially the arm), while both have been convulsed; (5) the writer has seen a case in which a patient alternately had attacks of only paralysis or convulsions, or of convulsion followed by paralysis, always in the same limb—the left arm; (6) the writer has seen three cases of hemiplegia from brain-disease, in which the paralysis slightly but evidently *diminished* after each attack of convulsions. Several similar facts are on record; (7) Féré has found a loss of power after convulsions in 21 per cent. of his cases in the right arm, and in 23 per cent. in the left arm, while in cases in which, without convulsions, there was vertigo with or without loss of consciousness, the loss of power was 30 per cent. in the right arm and 27 per cent. in the left; (8) Dr. Hughlings Jackson has seen in those cases in which an abortion of the attack can be produced, that paralysis not only may appear, but can be greater than if convulsions had occurred. All these facts are in harmony with the idea that paralysis occurring in seizures either after or without convulsions is due to inhibition and not to exhaustion.

There are many reasons also to consider inhibition and not exhaustion as the cause of all the other losses of power which are sometimes observed after an attack of epilepsy, such as aphasia, amnesia, anæsthesia, loss of the muscular sense, amaurosis, deafness, anosmia, loss of taste, loss of knee-jerk. This is so true, that certain of these kinds of loss of function are sometimes, or even very frequently, as is the case for knee-jerk (Beevor), replaced by an increase of power.

*Petit Mal.*—The symptoms of a seizure of *epilepsia mitior* are very different from those of a violently convulsive attack of epilepsy (*haut mal*). The loss of consciousness occurs only for one or a few seconds, and the spasmodic contractions take place in a few muscles only—in the face, tongue, throat, eyes, and neck. If seized while standing up the patient rarely falls, and, on the contrary, if walking he may continue his movement as regularly as before the fit. If attacked while speaking, he generally stops while the consciousness is lost, and on recovering it he may complete the unfinished sentence, so that the bystanders may know

nothing of what has occurred. Sometimes, however, the patient's mind is deeply altered, even when the attack of *petit mal* has been as short and slight as possible.

*Nocturnal attacks* of epilepsy may occur without any knowledge of their having taken place. Indeed, the writer has sometimes been consulted by persons who only asked for advice on account of headaches, and who had no pain in the head except after nocturnal attacks of epilepsy occurring without their knowledge. In such cases, the patient, after a seizure during sleep, wakes up tired, as if he had walked considerably; he has pains in the limbs, the back, and the head. He finds his mind confused, and his memory affected; he feels disinclined to get out of bed or to exert himself in any way; and often he is excitable or depressed. Sometimes his tongue or his lips are sore, and if the pillow is examined it shows bloody spots. More rarely it is found that an involuntary evacuation of urine has soiled the bed. Anyone sleeping within hearing distance of the patient may be wakened by the piercing epileptic cry, and then hear the noise of the shaking of the bed, caused by the convulsive movements. Such attacks, although very frequent and also very violent, may remain altogether unknown and unsuspected by the patient and his friends.

3. *Inter-paroxysmal*.—The general health of epileptics is usually very poor. Besides the alterations of their mental powers, and especially of their memory, they show a great deal of excitability and often depression of spirits. Their circulation and their digestion are often affected. There is nothing special, however, either in the morbid state of their mind and of their feelings, or in the disturbances of their physical health. Certainly the mental aberrations observed in the inter-paroxysmal state are not essential elements in the symptomatology of epilepsy. These aberrations can exist without epilepsy, and in a great many cases they are missing.

The *frequency of attacks* varies immensely in epilepsy. In one case the writer learned that for more than seventeen years the patient had passed no night without a fit, and for more than ten years the average nightly number of fits had been about twelve, which gives a total of more than forty thousand attacks in ten years. On the other hand, he was once consulted by a patient, sixty-two years old, who had had but seven attacks, the first of which occurred when he was nineteen years old, the interval between that and the second fit having been thirteen years, and the interval between the two last seizures having been seven years. Between extremely different cases like the two just mentioned we find the greatest variety as regards the frequency of attacks. Usually, however, there are a number of fits every

month. If there are attacks with extremely violent convulsions, the frequency is generally much less than when the convulsions are slight. Seizures of *petit mal* are usually very frequent. A perfect periodicity is extremely rare, but an approximation to periodicity is not rare, especially in women. There may be singular and inexplicable periods; the writer knows of a number of cases with a weekly periodicity, and of a case in which for years attacks recurred every forty-nine days.

*Status Epilepticus*.—Bourneville, Féré, and Dr. Gowers have studied carefully this dangerous condition, which is often fatal to epileptics. As the writer will show about the treatment of *morbus comitialis*, it is important to know that this recurrence of a large number of fits, within a short period of time, with no return of consciousness between the attacks, may be the only kind of epileptic manifestations in some patients, in whom, after variable but sometimes almost perfectly periodical intervals, this so-called status epilepticus reappears. Usually, however, this frightful and very rapid succession of violent fits occurs in the worst cases of organic epilepsy due to disease of the brain, kidneys, or liver, and frequently then destroys the life of the patient.

COMPLICATIONS AND SEQUELÆ.—Epilepsy has no necessary or usual complication. The diseases which often accompany it are frequently its causes and not complications. Bright's disease and other organic affections of the kidneys, diseases of the liver, the womb, and other viscera, when allied with epilepsy, if they have not been the first and only cause of it, are powerful additional causes. As regards sequelæ, the important researches of Dr. Hughlings Jackson, Dr. Hughes Bennett, and Dr. Féré have established that difficult speech, aphasia, amnesia, trembling, general physical and mental weakening, as well as any mental disorder, paresis or paralysis, anæsthesia, or great disturbances in the various organic functions, may not only appear temporarily as post-paroxysmal phenomena in epilepsy, but may persist and increase after each fit. The most common of those effects of seizures of this neurosis, and especially of *petit mal*, is amnesia, which may last, even if the epileptiform affection is cured.

*PATHOLOGY*.—The writer does not pretend to know what is the nature of epilepsy, so that when he tries to explain how that affection is produced and how its manifestations are generated, he will only state facts, and the conclusions they forcibly lead to.

I. As early as 1857 he endeavoured to show that one of the most essential features of epilepsy is an increased excitability of the reflecto-motor parts of the nervous system, which may exist not only in many parts of the centres, but also in some peripheric parts

of the nervous system. The analysis of the phenomena which appear, after certain injuries, in guinea-pigs or cats in which epilepsy can be gradually produced, leads forcibly to this view. In these animals, a few days after a lesion which causes epilepsy (especially a section either of the sciatic nerve or of a lateral half of the spinal cord, in the dorsal region) the first morbid change to be detected consists in an increase of the reflex power of certain parts of the skin of the face and neck, while a greater excitability supervenes in the medulla oblongata, in the upper part of the cervical region of the spinal cord, and in some parts of the trigeminal and of the two or three first cervical nerves. Gradually the reflex excitability of the nerve-cells in direct communication with the fibres of the nerves we have named increases, so that irritation of the skin of the face and neck, instead of producing only, as at first, a reflex contraction of the neighbouring muscles, causes tonic or clonic spasms of all the muscles of the trunk, neck, and head, on the side of both the lesion and the irritation of the skin. Later on, the reflex spasmodic action extends to the other side, and at last (a month or later after the traumatic injury from which proceeds this series of effects) a complete attack of epilepsy takes place, characterised, as in man, by loss of consciousness, with tonic and clonic convulsions.

In cases of injury to certain parts of the base of the brain in some animals, the same changes may occur, but with two differences: 1st, the rapidity of increase in the reflex excitability of the parts above-named is very much greater than after an injury to the spinal cord or to the sciatic nerve; 2nd, the zone of skin that acquires the power, when irritated, to give rise to an attack is on the side opposite to that of the brain-injury, while it is on the side of the lesion of a nerve or of the spinal cord.

The writer cannot give here the many reasons showing that what takes place in animals rendered epileptic by the lesions mentioned, may be applied in a great measure to the production of epilepsy in man. He will only say that cases identical with those of his guinea-pigs have been published by several clinical writers.

After the important discovery of Fritsch and Hitzig, concerning the galvanic excitability of a certain zone of the cortex cerebri, it has been found that epileptiform convulsions can easily be produced by an irritation of that zone in monkeys, dogs, and cats. It is clear, therefore, that perfectly healthy animals can be rapidly rendered epileptic. Man also can be seized almost at once after a hæmorrhage in the brain or a fracture of the cranium.

II. In cases in which for the first time these spasmodic phenomena take place, the

irritation which brings them on does this *only after having given rise to a morbid change constituting epilepsy* or an epileptiform affection. A longer time is necessary for the appearance of these convulsive movements than for any kind of natural reflex contractions.

The facts relating to the production of an attack of epilepsy by the galvanisation or certain parts of the cortex cerebri in monkeys, dogs, and cats, also show that a certain time is necessary for the production of the morbid state in which epilepsy consists. In that case also, as hereafter shown, the spinal cord is partly the seat of the genuine epilepsy generated. In other words, two distinct effects result from the irritative lesion—the one the genesis of the disease, *i.e.* of the morbid condition allied with which is the power of giving rise to epileptic or epileptiform attacks; the other, the production of a seizure.

III. Experiments in sudden crushing of the head in animals show that epilepsy can appear, although nothing remains of any part of the brain, *not even a portion of the medulla oblongata*, the connexion between the encephalon and the spinal cord having been altogether severed. Epilepsy, in the case of the crushing of the head, has its seat in the spinal cord, whatever be the share of the brain in giving origin to it. The writer, although recognising that fact about the location of this affection in such special circumstances, is not, and has never been, ready to admit that the spinal cord in all cases is the seat of this neurosis.

IV. The writer believes that the supposed absolute distinctions between the various kinds of convulsions (eclampsia and other forms of epilepsy due either to peripheric causes or to the state of the blood, &c.) are purely artificial and based only on the knowledge of the cause. All kinds of epilepsy may cause attacks, exhibiting the same essential features. In idiopathic, as well as in any other kind of epilepsy, an irritation which may come from any of the reflecto-motor parts of the nervous system, goes to a great many other parts of the nervous system, producing in some an inhibition of action or of power of action, and in others a simple expenditure of the normal or morbidly accumulated nerve-force, or, together with an expenditure, an increased generation of force (dynamogeny<sup>1</sup>).

Two facts forcibly show how these various effects of an irritation are produced.

<sup>1</sup> The writer designates by that name just the contrary to inhibition. That action is the effect of a transformation of force due to an irritation, and in virtue of which there is a sudden or rapid production of force. A great many facts establish the dynamogenetic power of certain parts of the nervous system over other parts of that system and over contractile tissues. See his article 'Dynamogénie,' in the *Diction. Encyclop. des Sciences Médicales*.

(1) Under the influence of a very slight irritation—that of a superficial prick of the medulla oblongata, close by the *nib of the calamus*—the brain may be suddenly and (so far as the writer knows) irrevocably inhibited, so that all the powers and functions of that organ disappear, while, on the contrary, the spinal cord, the nerves, and the muscles gain immensely in power: they are *dynamogenised*. The loss of cerebral activity is not due to a diminution or a cessation of circulation in the brain, as, on the one hand, the heart, for a time, shows no great weakness, although breathing is stopped; and, on the other hand, the experiment succeeds although the cerebral blood-vessels are dilated owing to the previous extirpation of the two superior cervical ganglia.

(2) The other *fact* shows the simultaneous production of convulsions with inhibition and dynamogeny. Instead of pricking the medulla oblongata, that organ is suddenly crushed, and then it is found, first, that the powers of the cervical and of a part of the dorsal spinal cord are inhibited, sometimes so completely that they can be galvanised without producing any muscular action; secondly, that, for a longer time than in death by asphyxia, either tonic or clonic convulsions alone, or tonic followed by clonic muscular contractions, take place in the hind limbs; and, thirdly, that the power of that part of the spinal cord giving rise to convulsions is considerably increased, as shown not only by their violence and duration, notwithstanding the cessation of circulation, but also by the fact that reflex actions can continue to exist after convulsions have ceased.

The first of these facts clearly proves that one and the same cause—a very slight irritation in a certain part of the nervous centres—can change at once and everywhere the dynamical condition of the nervous and muscular tissues, inhibiting all the powers of the brain and increasing considerably the powers of the spinal cord, of its nerves, and of all muscles.

The second fact positively shows that an irritation of a certain part of the nervous centres can give rise simultaneously to three radically different effects—an inhibition; a production of nerve-force; convulsions due to an expenditure of nerve-force.

V. These facts throw a flood of light on the phenomena of epilepsy. First, they clearly show that it is by an act of inhibition that the loss of consciousness takes place. Secondly, they lead to the conclusion that convulsions do not require the pre-existence of a great quantity of nerve-force in any part of the nervous centres, and that they can take place, with great violence and last a long time, from irritations which need not be very great. The supposition that epileptiform fits depend on a sudden discharge of

previously accumulated nerve-force is no longer necessary.

VI. The lesions found in many parts of the brain in epileptic patients do not show the *seat* of epilepsy—they only have one or the other of the three following significations: First, they are places of irritation generating that neurosis in the same way as peripheric lesions in the skin, in mucous membranes, in nerves, &c., produce it. Second, they may be mere co-existing alterations, having no influence over the epileptic affection. Third, they may be, and very often are, effects of the changes in circulation, in the state of the blood, and in cerebral nutrition, during fits. The alterations of the medulla oblongata, described by Schröder van der Kolk, and more completely by Ch. Bouchard, are certainly, as pointed out by this last observer, effects of fits, but able, however, at certain stages of their development to have a share in some of the convulsive phenomena. The last effort recently made by Dr. Chaslin to establish that in idiopathic epilepsy there is a constant lesion, has failed like all others. That lesion, consisting in the existence of a diffuse sclerosis located chiefly in the grey matter, is so little the essential organic cerebral cause of that form of *morbus comitialis*, that Féré, in a case of cure of that affection, found that the brain was the seat of the cortical sclerosis, besides having an induration of the two cornua Ammonis.

VII. One only of the supposed seats of epilepsy in the brain deserves some attention. It is that part which is the most frequently found diseased in cases of unilateral epileptiform convulsions, whether Jacksonian in character or not—the so-called motor zone. That part is also very often diseased in cases of general epileptiform convulsions, and, not rarely, besides, in cases of apparently pure idiopathic epilepsy. This frequency of lesion of the so-called motor centres, in epileptic cases, may as well be due to the fact that an irritation starting from that place has given rise in other parts of the nervous centres to the morbid state in which epilepsy consists, as due to the fact that this peculiar morbid condition is really located there. In other words, a lesion of the cortex cerebri along the Rolandic fissure, like that of a nerve anywhere, may be only the location of an irritative cause and not the seat of this neurosis.

Many facts observed in animals and in man show that this supposition is the correct one. Experiments on dogs have shown to the writer that every part of the motor tract in the encephalon, after the production of an epileptic attack by the galvanisation of the so-called motor centres, can, on galvanic irritation, give rise to an identical convulsive seizure. They have also established the fact that it is on the motor cells of the base of the brain and of the spinal cord that the

irritation of the cortex cerebri acts, in producing, first, the morbid state constituting epilepsy, and afterwards genuine epileptic attacks.

VIII. What is proved for dogs is, at least, extremely probable for man. Clinical facts show that most frequently any kind of lesion of the so-called motor centres or of the neighbouring frontal convolutions<sup>1</sup> has given origin either to genuine and complete epileptic fits, or to the greatest variety of epileptiform manifestations. This has led to the erroneous conclusion that epilepsy has its seat in certain cortical parts of the brain. It can be shown that all parts of the cerebrum, of the cerebellum, of the base of the brain, and also the cerebral meninges, the spinal cord, and the centripetal nerves, can produce epilepsy. On the other hand, irritative lesions are known to have existed, in many cases, in one or another part of the frontal or parietal lobes without the appearance of epilepsy.

When any kind or form of that affection appears under the influence of a disease or of an injury to the cortex cerebri in the parts looked upon as its real location, the altered tissues, as the following facts well show, may be only the seat of an irritation acting on other parts of the nervous centres, so as to give rise to epilepsy and to its manifestations. This view is proved by the facts—1st, that when this neurosis is due to a worm in the bowels, it can be cured by the expulsion of the irritating cause; 2nd, that when it depends on a neuroma, or neuritis, it can be cured by the section of a nerve between the diseased peripheric part and the nervous centres; 3rd, that when caused by some pathological alteration of a cerebral convulsion, it can be cured by the excision of the diseased part. In all those cases there is a cure, not because the seat of this convulsive affection is separated from the nervous centres, but because the irritating cause is suppressed.

Sometimes, as is well known, epilepsy will continue after the expulsion of a worm, the section of a nerve, and the excision of a diseased convulsion, or the opening of a cerebral abscess. In the first place, the irritative cause may be only partly suppressed by those means; in the second place, as said already, the morbid state in which epilepsy consists may exist in other parts of the nervous system than those where the irritative causes are located. That morbid condition may persist, remaining able, under the influence of other irritations than those

<sup>1</sup> Dr. J. Hughlings Jackson, in his late remarkable 'Lumleian Lectures,' has put forward the supposition that the seat of 'complete or genuine' epilepsy is in the prefrontal lobes. Against this supposition the writer will only say that all the characteristics of this affection have existed when these lobes were either absolutely normal or completely destroyed.

which acted previously, to give rise to seizures.

IX. Together with the conclusions flowing from the facts and arguments above discussed, the writer will give some others, the grounds for which lack of space does not permit him to mention:—

1. Epilepsy is a reflex neurosis even when positively allied with an organic lesion of the brain. Its only constant feature is an increase of the reflex excitability of a number of nerve-cells located in various parts of the cerebro-spinal centres. That hyper-excitability is generally, but not necessarily, greater at the time of an impending fit.

2. No distinction can be made between the kinds of epilepsy, with predominances either of considerable disturbance of the mental functions or of the higher senses, or of convulsive movements, or of any symptom. The greatest variety of epileptiform manifestations can arise from a similar lesion in any part of the brain or elsewhere.

3. Some parts of the nervous centres are always ready to cause, rapidly but not immediately, all or some of the phenomena of epilepsy, under the influence of an irritation, and especially a sudden one, proceeding from any central or peripheric part of the incito-motor nervous system. Two successive and absolutely distinct actions take place then: the first is a morbid change in the properties of a number of cells of the nervous centres—change according to which epilepsy or an epileptiform affection is generated; the second is a reaction from those cells bringing outwardly the manifestations of the special kind of the neurosis produced.

4. All the manifestations of genuine epilepsy, even in its worst and inherited form, in certain animals, can take place after the destruction of the whole brain, including every part of the medulla oblongata.

5. The seat of epilepsy is not where an organic lesion or a traumatic injury is found, either in the brain proper, the cerebellum, or any other part of the nervous centres, nor in any nerve or viscus. All organic lesions are only foci of irritation, seats of causes. The true location of the morbid state constituting epilepsy or an epileptiform affection is in reflecto-motor nerve-cells in parts of the central nervous system varying extremely.

6. Attacks of epilepsy are not essentially due to an overflow of nerve-force accumulated anywhere. The same irritating cause which brings on an attack can produce nerve-force at the same time that it determines convulsions by discharges of that force.

DIAGNOSIS.—In most cases it is easy to distinguish epilepsy from the few affections which resemble it. Sometimes, however, difficulties exist. There is no essential difference between the attacks of eclampsia in women and children and attacks of epilepsy, except the

existence in eclampsia of a peripheric cause of irritation, which is likely to disappear. But those purely reflex epileptiform attacks in women and children are sometimes succeeded by genuine attacks of epilepsy. Changes in the nervous centres occur during the eclamptic attacks, and lay the foundation of persistent epilepsy, although the peripheric irritation, which was the first cause, has ceased to exist. It is sometimes, therefore, almost impossible, as regards children especially, to say if we have to deal with eclampsia or epilepsy. The same may be said of all kinds of attacks of loss of consciousness and convulsions due to a peripheric cause, whether we call the affection eclampsia or reflex epilepsy (*see* CONVULSIONS). The first cause in these cases may cease to exist without our discovering positively that it has disappeared, and still attacks may continue. A mistake is not dangerous, as the treatment will be very much the same, except that in purely reflex epilepsy we have to fight against the peripheric cause, besides making use of means against epilepsy itself.

As regards the known facts concerning the ætiology of epilepsy and hysteroid affections, it must be remembered: (1) That, instead of epilepsy, hysteria may appear in the descendants of epileptics; (2) that an organic disease of the brain can give rise to genuine hysteria or to mixed forms of hysteria and epilepsy; (3) that diseases of the womb or even of the ovaries can give origin to epilepsy, and diseases of the testicles to hysteria; (4) that traumatic causes, and especially injuries to nerves, can sometimes produce hysteria; (5) that syphilis, uræmia, worms in the bowels, lead poisoning, can give rise to hysteroid affections. Ætiology alone cannot, therefore, be decisive for the diagnosis between epilepsy and hysteria.

The aura hysterica and the epileptic aura are generally very different one from the other, but they may be quite similar. The writer has seen a case of idiopathic epilepsy in an adult male, in whom the *globus hystericus* was the warning of almost every attack. As regards other means of diagnosis, we will refer to the articles HYSTERIA and HYSTERO-EPILEPSY.

Epilepsia mitior (*petit mal*) sometimes cannot be recognised easily from syncope. In *petit mal*, however, the loss of consciousness is absolutely sudden; the eyes are fixed; almost always some muscles of the face are contracted; sometimes other muscles, in the neck and elsewhere, are also contracted; if the patient is standing he rarely falls, and if he is walking he may continue to do it. The attack is very short, and there may be no appearance of its having occurred, except that some mental disorder may follow it; the pulse may be diminished, but it never disappears altogether. Syncope differs from *petit mal* in all those characteristics.

It is not necessary to draw here the lines of diagnosis between idiopathic epilepsy and cerebral epilepsy. The characteristic features of this last affection have been described at length in several parts of this article.

PROGNOSIS.—Inherited epilepsy is very rarely cured. The writer can positively state, however, that it may be cured. Among other good cases of persistent cure, he has seen two extremely remarkable. The patients were first cousins, and had inherited the disease from a grandmother: one of them died from a fall while intoxicated, five years after his last attack of epilepsy; the other died from typhoid fever, seven years after his last attack. They had both been treated for about two years, in 1852 and 1853.

There is a very great difference as regards prognosis between pure idiopathic epilepsy and other forms of that neurosis. The possibility of cure is greater when some curable or amendable organic alteration exists as a cause of epilepsy. One form of this nervous affection—that which is due to some congestion or even a more serious alteration of the brain, consecutive to typhoid fever, scarlatina, or measles—is often much benefited by treatment, if not cured. Already, before the great progress in the treatment of epilepsy through surgical means, the writer looked upon epilepsy caused by disease of the brain—syphilitic or not—as much more curable than any of the forms of this neurosis, excepting, perhaps, that which is due to an easily removable peripheric irritation. Epilepsy beginning in childhood, from teething or a bowel complaint, and having lasted many years, is almost incurable.

TREATMENT.—*Abortion of the attack*.—A most important fact ought at first to be pointed out under this head: it is that, as every attack causes in the nervous centres changes which prepare other attacks, it is essential to produce, if possible, the abortion of attacks whenever warnings occur. The treatment to obtain such an abortion varies with the kind of warning. In cases in which a real aura exists many means can be employed with the greatest benefit. The writer long since showed that the old-fashioned mode of prevention of attacks, consisting in the application of a ligature round a limb, acts not as the Galenic doctrine supposed that it did—that is, by barring the way to something going up to the brain; but, on the contrary, in doing just the reverse—that is, by sending an irritation towards or rather to the nervous centres. The writer has also shown that the ligature need not be left applied, and that a greater success is obtained by tying suddenly a handkerchief or a band, and repeating this tying several times in succession, than by applying the ligature even very tightly and leaving it so. Besides, he has found: (1) that the

ligature can do good even when applied on another limb than that where the aura is felt, although it is more efficacious on the latter; and (2) that pinching or striking the skin, or irritating its nerves by heat, by cold, by galvanism, or by repeated pricks with a needle, will generally do as much good as the ligature.

In those cases in which an involuntary muscular contraction takes place before an attack—that is, before the loss of consciousness—one of the most efficient means to produce an abortion of the fit is to draw forcibly on the contracted muscles, so as to elongate them. For instance, in those cases in which the unconsciousness is preceded by a contraction of the muscles of the neck, which draws the chin towards one shoulder, turning forcibly and rapidly the head towards the opposite shoulder gives in most cases a very good chance of checking completely the tendency to the fit. In case of contraction of the flexor muscles of the forearm, forcible extension of the hand over the forearm may succeed in preventing the attack. A blow, pressure, or friction on parts where some muscles become rigid, may have the same favourable effect.

If there are disturbances of breathing among the premonitory symptoms, the inhalation of ether or chloroform may prove successful. In cases of laryngismus simular means, or the cauterisation of the fauces by a strong solution of nitrate of silver, has been found most useful by the writer. The use of anæsthetics as a means of warding off an attack is too much neglected. By the help of ether in inhalations the writer succeeded, with Mr. R. Dunn, in preventing an attack in a patient who had had a fracture of the arm in a previous seizure, and who used to have a fit regularly every week. A whole week was gained in that way. In some cases the recurrence of attacks has been warded off by giving chloroform or ether (by inhalation) to patients who had had the first of what would have been otherwise a series of many fits. There is, in cases in which a fit is expected, a considerable chance of preventing it by anæsthetics.

According to the kind of warning and to its seat, one means or another out of a very large number (some of which only shall be mentioned here) ought to be used. An emetic, a purgative, a stimulant, the immersion of the two hands in hot water, the application of a lump of ice to the back of the neck or between the shoulder-blades, the subcutaneous injection of a solution of  $\frac{1}{100}$  of a grain of sulphate of atropine with  $\frac{1}{2}$  of a grain of acetate of morphine, powdered *asarum* taken as snuff, a dose of 25 grains of hydrate of chloral, the inhalation of a small dose of nitrite of amyl, extremely rapid and ample voluntary respiratory movements for five or six minutes, jumping, running for at least

ten minutes, reading very loud and fast—such are some of the means which the writer has found to be the most successful.

In a number of cases, patients under the writer's care obtained abortion of an impending fit by making energetic movements. Sometimes, however, even when the warning allowed the performance of very powerful general movements for a long time, the fit came with its usual violence. These facts are in decided opposition to the view that attacks of epilepsy are the effects of discharges of accumulated nerve-force by the so-called motor centres, or by other parts of the brain. Just as the tension of vapour diminishes in a boiler when steam is let off, the amount of nerve-force should diminish as a necessary consequence of the expenditure taking place in voluntary movement.

The second point of importance about treatment is to try to discover a part of the body which can by irritation give rise to a premonitory symptom of an attack, or even to an attack itself. If such a part is discovered, counter-irritation of some kind is to be applied there. Our remarks must be confined to saying that hard pressure on certain parts of the head, the spine, the breast, the abdomen, or the limbs, has in a number of cases produced an attack or some symptoms of it. We have seen the passage of a galvanic current produce the same effect. In such cases a blister or other local application has done good in diminishing the violence or frequency of attacks, and even, in a few instances, helped notably to a cure.

It is a remarkable fact that, whether a sensory or muscular aura exists from some affection or injury in the neighbourhood of its seat in the periphery, or from an organic affection or a traumatic injury of the nervous centres, the best place of application of any means of abortion of an attack, or of cure of epilepsy, is the same—*i.e.* the skin near the seat of the aura, or along the passage of nerves going to that seat. If a place, which is not connected with an aura, is found to be capable under an irritation of giving rise to a fit, there also is the best situation for the application of counter-irritants and other means of treatment against the epileptic affection. One of the most powerful means of counter-irritation, in all cases like the above, is a circular blister to be applied all round a finger, a toe, or an arm or leg. This means, used also with benefit by Dr. Buzzard, has led him to find that sometimes the aura, under the influence of a circular blister, disappears from the limb so treated, and is transferred to homologous places of the other side. In a case of epilepsy of Récamier's, a sensory aura changed place a great many times, and each time after an application of a small blister. The aura

so pursued disappeared, and the patient was cured.

*Treatment during the attack.*—The best plan in most cases of epilepsy is simply to save the patient from the danger of striking anything which might cause a bruise; to loosen his clothing, cravat, &c., so as to give freedom to respiratory movements; to wash the face with cold water if it become covered with perspiration; and at last to turn the head and face at the end of the fit, so as to prevent the falling back of the tongue on the glottis, and even to draw forward the tongue if there is deep stertorous or comatose breathing. There is, however, one means of treatment which has certainly proved sometimes successful in curtailing an attack even in so-called idiopathic epilepsy, although it is more powerful in partial epilepsy due to brain disease or to some peripheric irritation: this is pressure on the part of the neck where lie the carotid arteries. The idea was to lessen the amount of blood going to the brain. Whether obtaining that effect is really of service or not the writer cannot say, but, as often stated by him, it is not possible to press strongly over the carotid arteries without pressing also on the cervical sympathetic and on the nervi vagi. It is known that Augustus Waller and Czermak have ascertained that in man, as in the mammalians, pressure on the carotid artery acts sufficiently on that nerve to inhibit the heart more or less completely. This effect is certainly quite capable of curtailing a fit. The writer has shown also that such pressure can act on the nerve so as to modify favourably the condition of the respiratory centres, and he has proposed to employ pressure on both carotids and neighbouring nerves in those cases of epilepsy (and also of apoplexy) in which the heart beats very violently, while the respiratory muscles partake of the general convulsions.

*Treatment of epilepsy itself.*—The modes of treatment of the various forms of epilepsy which chiefly deserve to be noticed, must be divided into two classes, the internal and the external and surgical. We will first speak of internal means.

Against idiopathic epilepsy the most powerful means consists in the simultaneous use of some tonic remedy (such as strychnine or arsenic) be taken after meals; and of a mixture composed more or less like the following:—

R. Potassii iodidi ℥ij.  
Potassii bromidi ℥i.  
Ammonii bromidi ℥iij.  
Potassii bicarbonatis ℥j.  
Tinct. calumbæ ℥j.  
Aquæ destillatæ ℥v.

Of this solution four doses a day may be given to adults, three of one teaspoonful each before meals, and the fourth of three teaspoonfuls at bedtime, with as much water as desired. According to many circum-

stances the dose of one or another of the ingredients is to be changed. For example, if the *petit mal* exists alone, or co-exists with the complete epilepsy, the dose of the bromide of ammonium must be larger, and that of the other bromide diminished. If there is a weak pulse, carbonate of ammonium is to be substituted for the bicarbonate of potassium. In the writer's work on functional nervous affections all the rules relating to this mixture are given, and he will only say now that its use is considerably more beneficial than that of any of its ingredients alone, or any two of them. Two essential rules are to be followed when bromides are employed against epilepsy. The first is, that there ought to be no cessation whatever in the use of such remedies, as the whole benefit that may have been obtained may be lost at once after an interruption of even only a few days; the second is, that the treatment must be persevered with for at least two years after the date of the last attack. There is no evident harm in the great majority of cases from a prolonged use of a mixture like the above; a large number of patients have taken it for many years without any marked bad effect.

The writer has tried many other bromides than the two of this mixture. None has answered so well as these two. In a great many cases he has made use simultaneously of this mixture and of one or another of the many remedies which have been proposed for epilepsy. None of them has proved very useful except when there was some local affection which could be benefited by the added remedy. Such has been the case with atropine, ergotin, cannabis indica, digitalis, aconitine, cod-liver oil, &c.

In those cases in which the above mixture either did not produce a sufficiently good effect, or, still more, allowed of an increase of the convulsive neurosis or of *petit mal*, the use of atropine, of the ammoniated sulphate of copper, of borax, of the oxide or other salt of zinc, has frequently proved useful. It is not possible to say in what kind of cases one of these remedies is to be used rather than the others, except that zinc seems to be of better use when hysteria is more or less mixed up with epilepsy.

The writer has obtained a permanent cure of two cases of idiopathic epilepsy through the agency of nitrate of silver, without any discoloration of the skin. He employed a small dose ( $\frac{1}{8}$  of a grain three times a day), and stopped giving the medicine for eight or ten days after having had it used for three weeks. It required nearly a year for the complete cessation of the fits, and the use of the remedy was continued a few months after the last attack.

*Petit mal*, as well as complete epilepsy, can be treated advantageously with the above-mentioned mixture. Much benefit will some-

times be obtained in the treatment of this special neurosis by the addition of arsenic or of atropine.

It is hardly necessary to say that, when syphilis is the cause of epilepsy, the dose of the iodide of potassium in the prescription is to be notably increased (it must be at least a drachm a day), and also that frictions with the mercurial ointment are often to be resorted to. If epilepsy depend on some visceral affection, it is clear that the treatment should be directed against that affection. But if the liver is diseased from some influence of malaria, the sulphate of quinine should not be given, as it is almost always a bad remedy to employ in epilepsy, often more hurtful than useful. Arsenic should then be the remedy used for the sequelæ of fever and ague. If quinine must be employed in cases of clearly periodical epilepsy, the valerianate should be given.

The writer's experience shows that in most cases of epilepsy iron is harmful rather than useful. It is only in cases allied with or caused by chlorosis or considerable anæmia that the good effects of iron or manganese are often very marked. Still, the citrate of iron, although less powerful against a deficiency of blood-globules, may be used, because it is less apt to give rise to attacks than are most ferruginous preparations. Writers who disagree with these views (which are accepted by Dr. Hughlings Jackson) have lately tried to show that iron can always do some, if not much, good, and can do no harm; but they have, in support of their statements, given cases belonging rather to hysteroid affections than to epilepsy.

The writer has obtained no decided good effects from nitrite of amyl, antipyrin, picrotoxine, curara, or simulo.

It is important to state that, in cases of status epilepticus, the prompt and continued use of anæsthetics (ether, chloroform) may stop the tendency to the recurrence of fits. The condition of the tongue must then be watched carefully. If, as is usual, the fits are very violent, this organ falls over the larynx and increases the state of asphyxia, which contributes to the return of convulsions. The tongue is to be drawn forward, and any accumulation of frothy or bloody mucus in the throat must be wiped out. Anæsthetics have some power of curtailing, if not stopping at once, the tendency to a return of fits.

*External and surgical means.*—The notion that, if epilepsy depends on a peripheral cause which can be removed, it is right to employ surgical means, has often been acted upon, and with decided benefit. The number of cases is large in which a cause has been profitably suppressed by an operation or some other means. In a good many such cases, the section of a nerve or the extirpation of a foreign body or a tumour

has proved successful. Lately some cases of stretching of nerves, followed by cure or amelioration, have been published. A great deal is to be expected from counter-irritation. Ice applied on the bare skin, the actual cautery, or sedative applications on sores, may do much good.

As regards cases of idiopathic or cerebral epilepsy, all kinds of counter-irritation on the nape of the neck or at the level of the first and second dorsal vertebræ may prove useful, especially if there is considerable pain or heat in the head. When attacks are very violent and frequent, a decided amelioration may be obtained from croton-oil applications on a great part of the shaved scalp. Setons and issues very rarely do any good, and often weaken and irritate.

Among surgical means against epilepsy, two bold and somewhat strange operations have successively been proposed by an able surgeon, Dr. W. Alexander, of Liverpool: the one was supposed to act in producing anæmia in the base of the brain; the other, on the contrary, was to cause a vascular fulness. The first of these means consisted in the ligature of both vertebral arteries. Out of 36 cases there was a temporary cure or a persistent amelioration in 19 cases—certainly a very large proportion. Having given up this therapeutic means, Dr. Alexander formed the idea of taking away on the two sides the superior cervical ganglion of the sympathetic nerve, and obtained this remarkable result—that there was a cure or an improvement in 25 per cent. of the cases, while it is generally admitted that medical treatment gives about 7 per cent. of cure or improvement. The writer, notwithstanding the apparent superiority of this kind of treatment, would be quite indisposed to recommend it except after complete failure of all the best internal remedies.

Operations on the genital organs of epileptics of the two sexes are only to be resorted to in desperate cases.

Trephining against epilepsy, during a great part of this century, although employed indiscriminately, has nevertheless frequently proved useful. It is now, in appearance, used according to rational scientific principles—in reality sometimes quite faulty. Statistics show that, with the addition of very bold operations, it is not so frequently useful as it previously was. In other parts of this article (*Ætiology*; *Symptoms*) there are facts showing that recognition of the seat of a tumour or of some other organic disease of the brain is not always possible, and must in many cases be extremely difficult. Be that as it may, it is quite certain that not rarely after the laying bare of the dura mater or the cerebral convolutions, surgeons have been disappointed in not detecting an expected morbid state to which they attributed an existing epileptic affection. On the other

hand, however, they were, sometimes, agreeably surprised in finding that in such circumstances the affection disappeared. In 1857, after having given all the details of a case of the kind, furnished to him by Dr. van Buren, the writer in his work on Epilepsy put forward a view, very tersely expressed a few years ago by Dr. Gowers in the following sentence: 'It is possible that the measure (trephining) is sometimes beneficial as an energetic form of counter-irritation, but it is doubtful whether for this object its performance is justifiable.'

Knowing how powerful counter-irritation may be against epilepsy caused by a blow on the head or allied with some tenderness of a point of the scalp, the writer long ago proposed that, before resorting to trephining, the most energetic means of counter-irritation should be employed. Trials should be made of ice applied on the shaved and bare skin of the head twice a day for an hour each time, of the actual cautery, of blistering, of croton oil, and of a means which the writer (imitating Pouteau) used successfully in two cases, consisting in a deep crucial incision of the scalp and the pericranium (the cuts being of one inch and a quarter). It is possible that it was a more or less similar operation, and not the trephining, which was the means of success in the cases above mentioned.

When we read of cases of cure after the extirpation of bony spiculæ or of a mass of cicatricial tissue, of a tumour, or of a certain amount of the convolutions of the brain, it is impossible not to be struck with the resemblance of these facts to the cases of cure of epilepsy by the expulsion of worms from the bowels, or the removal of a neuroma or a foreign body pressing on a nerve, or of other causes of peripheric irritation. The analogy between the cerebral and the peripheric cases is very great: both are cases of reflex epilepsy. In both kinds of cases there may be a failure of the means used, owing, no doubt, to changes having occurred in the nervous centres which are able to give rise to attacks of epilepsy. In such cases the excision of the supposed centre of certain muscles may not be followed by a cessation of convulsions of those parts. So it was in the case of a boy spoken of by Dr. Ferrier, and who had been operated upon by Mr. V. Horsley.

Notwithstanding the diminution of risks due to antiseptics, great is still the proportion of deaths following, if not caused by, the operation of trephining, with the ablation of morbid parts of the brain, and sometimes, alas! of perfectly healthy-looking parts.

There is no doubt that surgeons are entitled, in certain cases, to employ trephining against epilepsy; but, for the reasons above briefly given, the writer thinks that,

before resorting to this operation, when there is no clear evidence that some morbid parts are to be removed or an abscess to be opened, or some necessity arising from a fracture, it would be wise to first exhaust all means of counter-irritation.

C. E. BROWN-SÉQUARD.

**EPILEPTIC INSANITY.**—SYNON.: Fr. *Folie épileptique*; Ger. *Epileptisches Irresein*.

**DEFINITION.**—Mental disorder which is the outcome of the epileptic neurosis, and which is generally, though not invariably, accompanied by motor epilepsy.

**ÆTIOLOGY.**—The statements in the article EPILEPSY may be taken generally as applicable to epileptic insanity. But an exception must be made in regard to what is said concerning sympathetic epilepsy, the insanity described here being associated only with epilepsy of cerebral origin.

**PATHOLOGY.**—The structural changes associated with epileptic insanity have their seat in the cerebral cortex, and involve an altered nutrition of certain of the nerve-cells. This altered nutrition is usually due to idiopathic degeneration; but it may also be due to morbid growths or other irritative lesions in neighbouring parts of the brain. It seems probable that functional disturbance of the cells in the higher centres or psychic area may be excited by radiation from the motor area in which an epileptic discharge originates; but in most cases there can be little doubt that the cells in the psychic area are themselves the subject of structural change. In some cases, indeed, the psychic centres are the first affected, and the discharges may be at least for a time limited to these centres. In such cases the discharges are manifested by loss of consciousness, hallucinations, or outbursts of mania unaccompanied by convulsions. The structural change chiefly occurs in the small irregularly shaped nerve-cells which are found in the second cortical layer. Bevan Lewis regards as of a highly characteristic nature a fatty degeneration of the nuclei of these cells, which goes on to vacuolation, and ends in complete disintegration of the cells. He describes similar changes as usually affecting to some extent the other layers of the cortex. This form of degeneration is not, however, as Bevan Lewis himself shows, peculiar to epilepsy; and other observers, though using Bevan Lewis's own method of examination, have failed to find it in some cases of epilepsy. When a patient dies in a fit, there is usually found intense venous engorgement with sometimes punctiform hæmorrhages in all the viscera, including the brain. This condition has important pathological bearings; but it is post-epileptic in its nature, and is properly regarded, not as the cause, but as a result of the epileptic seizure.

DESCRIPTION.—It is necessary to consider separately three aspects of the unsoundness of mind which is associated with epilepsy, according as it appears (1) as arrested or impeded mental development—*epileptic idiocy* and *epileptic imbecility*; (2) as progressive mental degeneration—*chronic epileptic insanity*; and (3) as transient mental disorder—*acute epileptic insanity* or *epileptic mania*. These three conditions are to be regarded as different phases and not as different kinds of epileptic insanity. They are intimately connected with one another, and occasionally they may all be illustrated in the progress of a single case. Thus epilepsy supervening during infancy or youth may so retard mental development as to produce imbecility; the subsequent periodical recurrence of epileptic fits during adult life may gradually pervert and deprave the already imperfect intelligence, causing it to pass through a phase characterised by emotional disorder, hallucinations, and delusions, and ending in the hebetude of complete dementia; this progress may occupy many years, and it may be interrupted by intercurrent outbursts of acute maniacal excitement. Though it is necessary, therefore, for the purpose of description to deal with each of the phases separately, their interdependence must not be overlooked.

1. **Epileptic Idiocy and Epileptic Imbecility.**—These terms are used to indicate different degrees of the same affection—a more or less complete arrest of mental development due to epilepsy. Those individuals in whom epilepsy supervenes in infancy, where there is no development of mind, or very little, are called idiots. Those in whom epilepsy commences after substantial progress has been made in mental growth, or in whom the epileptic seizures, though appearing early, do not recur frequently, and where there is considerable though defective mental development, are called imbeciles. The effect of the fits upon the mental condition of children differs greatly in different cases. The fits are usually followed by more or less mental retrogression. In many cases after a fit, and especially after a series of fits, the patients fall into a state of prostration and stupor of some duration. The educational training of the epileptic imbecile is seriously embarrassed by the occurrence of such attacks. A great part of what has previously been learnt seems to be wiped out, and requires to be learnt over again. In cases where the fits cease the evolution of the intellect may resume its course, but the mental organisation always remains damaged. It is estimated that about one-third of the total number of cases of idiocy are due to epilepsy.

2. **Chronic Epileptic Insanity.**—In a large number of epileptics, estimated by Russell Reynolds at about one-third of the total number, the mental condition is not affected to such a degree as to be recognised

as abnormal; and in a small number of cases the disorder is limited to a few brief attacks, sometimes to a single attack, of maniacal excitement. In about two-thirds of the cases there is an appreciable mental enfeeblement which gradually increases so long as the fits continue to recur, in some cases never going farther than a peculiar emotional mobility and weakness of memory, and in others passing on to profound dementia. This progressive mental degeneration, though varying greatly in degree, is very uniform in type, and it is important to be able to recognise it. In the mildest cases there are usually to be detected lapses of memory, fits of absent-mindedness, and transient confusions of thought; and there is always more or less instability of mood and morbid irritability. The instability is the most characteristic mental feature of the epileptic. At one time he will be gloomy, despairing, conscious of his mental enfeeblement, and lamenting its hopeless nature; at another time he will exhibit a surprising self-satisfaction, hopefulness, and confidence. Many epileptics are liable to fits of intensely passionate anger, occurring without apparent cause. The changes of mood often take place with a startling suddenness which is important as a diagnostic indication. In the fully developed chronic insanity of the epileptic the variability of the mental condition may show itself in the intellectual as well as in the emotional state. A patient who usually exhibits confusion of thought, weak memory, and little power of attention may suddenly awake to a state of great mental activity, and show remarkable clearness and rapidity of thought, vivid memory, and great power of concentration. A common condition is to be habitually quarrelsome, argumentative, fond of teasing, insolent in demeanour, and given to capricious cruelty; yet such patients will sometimes be found marvellously sweet in disposition, benevolent, affectionate, and considerate. In the advanced stages of the disorder the moral nature is generally deeply degraded, as shown in repulsive obsequiousness of demeanour, low cunning, treachery, and cruelty. The intellectual condition also passes through stages of degradation. Delusions of being injured by other persons fill the mind, and visual and auditory hallucinations occur. When these are present the patient, with his tendency to impulsive passion, is dangerous to persons near him. The last stage is marked by complete loss of moral sense and mental capacity. Nothing remains but the lower instincts in their most degraded form; and this is accompanied by a physical debasement which accords only too well with the mental condition.

3. **Acute Epileptic Insanity—Epileptic Mania.**—The term 'epileptic mania' is used to denote a peculiar automatic form of acute mania which sometimes follows epileptic seizures, and more rarely occurs in

persons with the epileptic neurosis who have never had a convulsive attack—or at least who have not been known to have one. There is generally some transient mental perturbation preceding or following a convulsive seizure, which does not attain the intensity of mania, but which may be fitly alluded to here. The pre-paroxysmal phase of these perturbations may last for a few hours or a few days. They consist in a development of conditions which have been already described as characteristic of the early stage of chronic epileptic insanity. Sometimes there is morbid irritability, depression, or exaltation, sometimes confusion of thought or loss of memory. Immediately before the occurrence of a fit there is often some special idea which rivets the attention, the recollection of some particular scene, or a special hallucination such as a flame, a red colour, a spectre, or a peculiar odour. When there is a particular idea, or an hallucination, it is usually the same which recurs at each seizure, and it generally has some relation to the circumstances in which the first fit occurred. After the convulsive fit has passed off, there is frequently a short period of confusion of thought, and lethargy. The condition known as epileptic mania is an outburst of maniacal excitement lasting from a few hours to several days. The general features which are distinctive of these attacks are the suddenness of their onset, the violence and abrupt rapidity of the acts of the patients, the painful and appalling nature of the ideas and hallucinations from which they suffer, the suddenness as well as the completeness of the return to sanity, and the entire, or nearly entire, forgetfulness of what has happened during the course of the attacks. During an attack the impulsive acts are often of a homicidal or suicidal character, and this makes epileptic mania one of the most dangerous of all kinds of insanity. Sometimes the attack is characterised by furious maniacal excitement during its whole course, but there are also cases in which there is an apparent calmness of conduct. In a typical case of this kind the patient is irritable, suffers from a vague terror, seems impelled by a force, which he feels irresistible, to commit acts without conscious purpose. In this state the patients will walk long distances without object, steal or destroy articles in an unaccountable way, and will commit homicide or suicide with apparent deliberation. They seem insensible to everything which does not fall in with their dominant idea or impulse. This automatic action is a special characteristic of epileptic mania, both in the cases where there is violent excitement, and in those where there is an appearance of deliberate co-ordination of thought and act. The patients are as if in a waking dream; and when they awake suddenly to rational consciousness, they feel as if they had passed through a dream which they dimly conceive

to have been of a distressing and frightful nature, but of whose details they have no recollection, or only a very vague one.

The relation of the mental disorder to the convulsive seizures is concisely given in the following sentences. When the fits are frequent, and continue through a course of years, they are generally associated with chronic epileptic insanity, though there are exceptional cases in which the mind is not seriously affected. Where the fits are frequent, and are of the character of *petit mal*, the mental degeneration is usually more rapid and complete than where the motor symptoms are more severe, as in the *grand mal*. The calmer type of epileptic mania, called by Falret *petit mal intellectuel*, is most frequently associated with fits of the character of *petit mal* and with nocturnal fits; while the violent excitement, called by Falret *grand mal intellectuel*, is most frequently associated with the severe motor fits. Epileptic mania is most frequent after a fit or a series of fits, is less frequent before a fit, and least frequent in the intervening periods; it is specially apt to occur after a recrudescence of fits following a long period of freedom from them. It is also apt to occur after a rapid succession of abortive fits. It is rare in the first stages of epilepsy, most frequent in the middle stage, and rare after dementia has begun.

TREATMENT.—In the treatment of epileptic insanity everything which is useful in the treatment of epilepsy is to be included (*see EPILEPSY*). The bromides are the most important remedies, especially bromide of potassium. It should be given persistently in doses just short of producing bromism, combined with small doses of arsenic. It may be given for years if necessary. When syphilis is present, the bromide should be combined with the iodide. When pre-paroxysmal symptoms are observed, 20 grains of chloral hydrate should be given and the patient put to bed. The same treatment is useful after a fit; and it may also arrest a fit, or series of fits, or ward off an attack of epileptic mania. Constipation and overloading of the stomach with food should be carefully avoided. There should be complete abstinence from alcohol; it is apt to bring on fits and also mania.

MEDICO-LEGAL RELATIONS.—Where responsibility for acts committed in what seems to have been an attack of epileptic mania is in question, the whole character of the mental symptoms and the evidence of other epileptic conditions must be kept in view. The difficult cases are those in which there has been no violent excitement, and when the conduct has had the character of the calmer type of epileptic mania. Here it is important to bring clearly out the automatic character of the conduct. But the most important point is the existence of the fits. It may happen that the fits have not been recognised though

they were really present. Nocturnal fits are the most likely to escape recognition. It must not be forgotten that they may have occurred without the knowledge of the patient himself. Careful inquiry should therefore be made for signs of their occurrence, such as biting of the tongue, of the lips, or of the cheek; spots of blood on the pillow; gathering of froth about the mouth on waking; unconscious emission of urine; peculiar fits of snoring; or the occurrence of numerous small ecchymoses under the skin of the neck or forehead, the last being a sign the importance of which is strongly insisted on by Trousseau. When no convulsive fits can be ascertained, evidence of vertigo, or of brief losses of consciousness occurring periodically or in series, should be looked for. If in the opinion of a medical witness an act of violence has been the result of epileptic insanity, it is his duty to show that not merely the act itself, but also the conduct and condition of the accused otherwise, are symptomatic of the epileptic condition.

JOHN SIBBALD.

**EPILEPTIFORM }.—Partaking of the**  
**EPILEPTOID }**  
characters of epilepsy; terms generally applied to convulsions. See CONVULSIONS.

**EPIPHORA** (*ἐπί*, upon; and *φέρω*, I carry).—A flow of tears so persistent that they run down the cheek, due either to obstruction of the lacrymal duct or to excessive secretion. See LACRYMAL APPARATUS, Diseases of.

**EPIPHYTA** (*ἐπί*, upon; and *φυτόν*, a plant).—These are the plant-like organisms found on the skin and its appendages, or on mucous surfaces, the so-called *vegetable-parasites*, originating certain diseases, such as the various forms of tinea and thrush. The more important of them are the achorion, trichophyton, and microsporon. The achorion Schönleinii is the vegetable fungus which constitutes the mass of the crusts of favus, and belongs to the group of oidia. The trichophyton is the dermatophyte of tinea and sycosis, and is found in the substance of the hair as well as in the epidermis. The microsporon, termed epidermophyton by Bazin, is the parasitic fungus of pityriasis versicolor. Both the latter are members of the group of torulacæ. See EPIPHYTIC SKIN-DISEASES; and APHTHÆ.

ERASMUS WILSON.

**EPIPHYTIC SKIN-DISEASES.**—**SYNON.:** Tineæ; Dermato-mycoses or Dermato-phytoses; Ringworms; Fr. *Trichophytes* (Besnier); *Teignes*; Ger. *Flechte*.

**DEFINITION.**—An *epiphyte* is a living vegetable organism which grows in the superficial parts of the skin or mucous membrane of another organism, and is parasitic—i.e. lives at the expense of its host.

**CLASSIFICATION.**—The vegetable parasites are all fungi and belong to the subdivisions Schizomycetes (bacteria), Blastomycetes (yeasts), and Hyphomycetes (moulds). In addition to the pathogenic microbes of anthrax, glanders, erysipelas, leprosy, tuberculosis, &c., which may occur in the skin in association with these diseases, there exists under normal conditions a number of micro-organisms amongst the scales of the skin and its appendages, in nature between saprophytes and genuine parasites. There is a growing tendency to assume that, under certain conditions, this 'dermatological flora' may possess pathogenic properties. There may also be found from time to time accidental organisms of various kinds, and these are the pyogenic microbes. A parasitic origin has been ascribed to alopecia areata, but not proved; and the ætiological connexion of certain organisms found in furuncles, oriental sore, and impetigo contagiosa, &c., is still unsettled. Lastly, there are the bacteria of bromidrosis and red sweat, and the organisms associated with certain hair conditions, such as tinea nodosa, piedra, and forms of sycosis.

Apart from all such, however, there is the distinct group of Hyphomycetes, achlorophyllous thallophytes, living only on organic carbon compounds and needing oxygen, which constitutes the long-established class of true epiphytic diseases. The group is composed thus:—

1. *Tinea favosa* or *favus*, caused by the *Achorion*, discovered by Schönlein. The phases occurring in the scalp, beard, non-hairy parts, and nails may be specially noted.
  2. *Tinea trichophytina* or *ringworm*, caused by the *Trichophyton tonsurans* of Gruby and Malmsten. Here also the phases occurring in the scalp, beard, non-hairy parts, and nails have special characteristics.
  3. *Tinea imbricata* or *Tokelau ringworm*, a special (?) form found in Polynesia, and the Malay and Indian Archipelagos.
  4. *Tinea versicolor* or *chromophytosis*, caused by the *Microsporon furfur* of Eichstedt.
  5. *Erythrasma*, caused by the *Microsporon minutissimum* of Burckhardt.
- To this list are usually added *mycetoma* or the fungus-disease of India, due to the *Chionophye Carteri*; and an obscure disease of Mexico and parts of Central America called *pinta*.

This group of the Hyphomycetes, whose natural history is even yet not fully known, stands apart by itself, but its members are most nearly allied to the Mucorinæ. The difficulty of determining their affinities and classification has given rise to much controversy, now almost subsided. Increased knowledge of their morphology, direct experiment, and successful pure cultures have established that these dermatomycoses are

not due to polymorphic forms of some mould, such as *Penicillium*, *Aspergillus*, *Mucor*, &c., but are distinct species, with different vegetations, growing in a constant and determined form, with different behaviour under attempted cultivation, and always giving rise to similar objective symptoms. *Tinea trichophytina*, *favosa*, and *versicolor* cannot give rise to one another, but breed true. These dermatomycoses are contagious, and are inoculated from person to person and from animals. Their fungi have no sporangia, but consist of spores and simple or branched, jointed or unjointed, filaments or tubes, which are either mycelial (vegetative) or spore-bearing. The spores become isolated, or are arranged in moniliform threads or in masses. Balzer says, 'Everything is derived from the spore, which by elongation forms a tube or filament of mycelium; in the interior of this tube the substance of the nucleus (*protoplasm*) buds, sends out lateral processes and segments, so as to form the *spore-bearing tube*; when, later on, segmentation of the sheath occurs in its turn and imprisons the segments of the central substance, new spores are thus formed and will evolve, in their turn, in a similar manner.'

**DESCRIPTION.**—To examine the fungus, some epidermic scales should be scraped, or a hair placed on a slide, and soaked for a time in 20 to 40 per cent. soda, potash, or ammonia solution or in acetic acid. Then the specimen should be gently pressed out by the cover-glass to ensure proper focussing, and examined by a microscopic power of 300 or more diameters. If there is much fat present, the material must be previously soaked in ether. For more accurate and delicate examination, coloration of the specimen is necessary with methyl violet, eosin, or iodine. The erythrasma fungus needs high powers and staining. It is often impossible to name with certainty isolated spores or even filaments of these epiphytes, but when seen in bulk and in connexion with the clinical symptoms, there is rarely any difficulty. Unfortunately there are no elective stains to differentiate them. The abundant *Microsporon minutissimum* is so minute and slender as to be unmistakable. *M. furfur* also is very characteristic, with its roe-like masses of large uniform spores, and its short, much curved pieces of mycelium. The *Achorion* and *Trichophyton* are much more difficult to distinguish apart from cultures, and they differ from the other two epiphytes in attacking the hairs, though *achorion* does so to a much less extent than *trichophyton*. Kaposi says *achorion* displays a predominance of conidia of very variable size and shape, a comparatively short and remarkably jointed mycelium, a scarcity of smooth-bordered tubes, and a facility for breaking up into single cells; *trichophyton* less numerous, smaller and more uniform conidia, and more

tenacious, branched, and smooth-bordered mycelium. In the hair and nails, however, *trichophyton* forms conidia with the greatest freedom.

**ANATOMICAL SEAT.**—These dermatomycoses flourish in the epidermic tissue—*i.e.* the epidermis, hairs, and nails. We know little of many of the conditions of the soil favourable or otherwise to their growth. *Achorion* and *trichophyton* prefer the skin of the young and such as grows fine hair. These fungi are more likely to attack and flourish in uncared-for children, and hence the prevalent idea that ill-health or dirt are essential factors. A certain degree and combination of moisture and temperature are favourable, as exemplified by the affection of calves after being shut up in winter quarters, and by tropical ringworm.

**EFFECTS.**—The effects which epiphytes produce are, firstly, to mechanically dissociate and thus destroy the epidermic cells, on the nitrogenous products of which they live; and, secondly, to cause local inflammation, chiefly in all probability by the formation of irritating chemical substances. The degree of inflammation excited varies for each fungus and with different individuals; and it is difficult to determine the relative parts played by individual susceptibility, and the powers of irritation possessed by the fungus under the different conditions of its source of origin, the character of the nutrient medium, &c. The inflammation may be evidenced only by hyperæmia and desquamation, or by exudation and the formation of papules, vesicles, pustules, &c. As the fungus tends to grow equally in all directions, a rounded area of inflammation is formed, which subsides in the older central parts, so that a *ringed eruption* is commonly produced.

The general health is not perceptibly affected by any absorption of the products of fungus-growth, nor is there evidence of any auto-vaccination preserving from a second attack.

These dermatomycoses are inoculable from human beings and some lower animals directly, and by means of infected caps, towels, brushes, razors, clothes, &c. Their course is usually very chronic, but the soil may become exhausted and unfavourable.

**DIAGNOSIS.**—The diagnosis, when the clinical symptoms are well-marked, is clear; but scientifically, and even practically at certain stages, it is necessary to search microscopically for the fungus.

**PROGNOSIS AND TREATMENT.**—The *prognosis* depends on the site affected and the nature of the fungus; but the dermatomycoses are all amenable to *treatment* by parasiticides and the mechanical removal of the fungus. Great inconvenience and annoyance, however, are occasioned, and sometimes disfigurement. See *TINEA TONSURANS*; *TINEA VERSICOLOR*; and *FAVUS*.  
T. COLCOTT FOX.

**EPIPLOITIS** (*ἐπίπλοον*, the omentum). Inflammation of the epiploon or great omentum. See PERITONEUM, Diseases of.

**EPISPADIAS** (*ἐπί*, upon; and *σπάω*, I tear).—A malformation of the penis, in which the urethra opens on its upper surface. See PENIS, Diseases of.

**EPISPASTICS** (*ἐπί*, upon; and *σπάω*, I draw).—Substances which, when applied to the skin, are capable of producing a blister. See COUNTER-IRRITANTS.

**EPISTAXIS** (*ἐπί*, upon; and *στάζω*, I drop).—SYNON.: Fr. *Épistaxis*; Ger. *Nasenbluten*.

DEFINITION.—Epistaxis signifies a bleeding from the nose.

ÆTIOLGY.—Epistaxis is either *traumatic* or *idiopathic*.

The *traumatic* form may be occasioned by a blow, by violent sneezing, by snuffing up irritating substances, or by direct violence; but in these cases there frequently appear to be general or local *predisposing* causes to account for the readiness with which it occurs, such as a hæmorrhagic diathesis, an inflammatory or congestive hyperæmia, or some ulceration of the mucous membrane.

The *idiopathic* form of epistaxis frequently occurs in children, particularly boys, just before or about the age of puberty, and in girls as a form of vicarious menstruation. Epistaxis may be one of the forms of bleeding in persons of hæmorrhagic diathesis, in which case it is a source of anxiety and difficulty. Occurring in advanced life, it may be indicative of over-tension or obstruction of the cerebral venous system from chronic Bright's or cardiac disease; and the blood which flows is then often venous in appearance. Occasionally it occurs as a spontaneous relief to determination of blood to the head, in which form the blood generally proceeds from one nostril only. In other instances epistaxis is connected with serious disorder of the blood, as in the specific fevers. Thus it is often associated from the outset with remittent, enteric, typhus, or scarlet fever, and is indeed regarded in some degree as pathognomonic of enteric fever. It may also attend scurvy, purpura hæmorrhagica, splenic disease, pyæmia, and erysipelas, being a consequence of the septic condition.

In its passive form, epistaxis may be associated with organic disease of the heart, pleurisy, emphysema; or with ascites or ovarian dropsy, on account of pressure on the diaphragm inducing a stasis of the venous circulation. It may occur spontaneously from exposure either to great cold or great heat, or a sudden change from cold to heat, or from the diminution of atmospheric pressure, as in going up high mountains.

SYMPTOMS.—Hæmorrhage from the nose is too familiar to demand description in this

place. The flow of blood may be either continuous or drop by drop. As a rule, the escape of blood is from one nostril, bleeding from both being rare. It may last a very short time, or for some hours, and in severe instances for days, causing syncope, or even being attended with fatal results. It is at times met with as occurring periodically.

DIAGNOSIS.—Epistaxis must not be confounded with hæmoptysis, as may happen if the epistaxis takes place posteriorly, and the blood passes into the mouth.

TREATMENT.—When epistaxis is obviously a salutary process, as it undoubtedly is in a good many instances, it subsides spontaneously; where it occurs frequently and severely, recourse must be had to mechanical, cutaneous, or internal remedies. The local application of cold, in the form of cold-water or iced compresses to the nose, neck, or forehead is most useful, acting as these agents do either directly or by inducing a reflex effect on the vaso-motor nerves. Simple pressure upon the nostril, or upon the septum nasi, by compressing the bleeding nostril with the finger of the opposite hand, while the arm of the affected side is raised above the head, is the most readily practicable and effectual of all measures. The application of mustard over the stomach or upon the ankles is sometimes successful. When simple means fail, astringents, either in the form of solution or powders, may be injected into the cavities, or applied on plugs of lint or cotton-wool; such as alum, acetate of lead, the salts of iron, or gallic or tannic acid.

Internally, the frequent administration of saline purgatives, tincture of perchloride of iron, turpentine, bromide of potassium, belladonna and quinine, ergotin (subcutaneously), sulphuric acid, or acetate of lead, may be necessary in cases of periodic attacks.

In very severe cases the operation of plugging the nares, and thereby favouring the formation of a clot, must be resorted to; and the most ready and easiest method is by the employment of Bellocoq's sound, or, more properly, cannula. If, however, this instrument be not at hand, a catheter or an eyed probe should be threaded with a stout silk or hemp ligature, and pushed along the floor of the nose until it protrudes beyond the velum palati; one end of the thread should now be pulled out of the mouth by the fingers or forceps, and a roll of lint or a piece of sponge tied to it, and then pushed up behind the velum. The catheter and the attached thread being now withdrawn through the nostril, the plug is pulled forcibly against the posterior nares, and by the pressure exerted the hæmorrhage can generally be arrested. The intra-nasal plug is invaluable. It consists of a thin indiarubber bag, connected with a tube provided with a stopcock. The bag is passed empty along the floor of the nose into the nasal fossæ, and then blown up or filled

with water. Transfusion is indicated in very severe cases, where there is danger of delay. The strictest antiseptic precautions should be used in 'plugging'—such as puffing with iodoform, or syringing with the various antiseptics, as it not infrequently happens that the condition known as *ozæna* commences after severe cases of epistaxis.

EDWARD BELLAMY.

**EPISTHOTONOS** (*ἐπισθεν*, forwards; and *τείνω*, I extend).—A synonym for *emprosthotos*. See *EMPROSTHOTONOS*.

**EPITHELIOMA** (*ἐπί*, upon; and *θηλή*, the nipple (the word 'epithelium' being at first used for the tissue covering the nipple); and *oma*, a termination indicating a tumour).—**SYNON.**: Epithelial Cancer; Canceroid; Fr. *Epithélioma*; Ger. *Epithelkrebs*.

**DEFINITION.**—Cancerous disease of the skin and mucous membranes, caused by growth inwards of the epithelial cells of the rete mucosum and of the mucous membrane, leading to destruction of connective tissue and progressive ulceration.

**ÆTIOLOGY.**—Epithelioma is a disease of advanced age, occurring mostly after forty, although it is found occasionally in young, and, rarely, in very young, individuals. It is more common in men than in women, and is usually considered hereditary.

The frequency with which it begins in a wart, shows that certain histological conditions of the skin predispose to its development. The exciting cause is often to be found in local irritation, of which a striking example is seen in the frequency with which cancer of the lower lip in men is caused by the tobacco-pipe. Cancer caused by soot gives rise to chimney-sweep's cancer. It may develop in chronic ulcers, in lupus, in syphilitic scars, and in cicatrices. The irritation of bad teeth may cause it on the cheek. In Cashmere, where braziers are frequently applied to the abdomen and thigh, epithelioma occasionally results. Leukoplakia of the mouth in certain cases ends in cancer. Three cases have been reported in which it developed in warts growing on the hands of persons employed in tar and petroleum works. It may be set up by the irritation of old-standing eczema. In Kaposi's disease a great number of cancerous epitheliomatous ulcers develop in the skin, following the deposition of patches of pigment. Four cases have been reported in which epithelioma developed in old-standing psoriatic patches. It is, however, abundantly proved that chronic irritation of the most various kinds may, in certain persons, result in producing epithelioma; but in what the predisposition consists, or in what way the irritation produces the epitheliomatous growth, are questions upon which no light has yet been thrown.

**DESCRIPTION.**—In the early stage of epithelioma of the skin the affected surface is found

to be hard, more or less nodular, and characterised by small, closely-set, irregularly defined papules which are covered with epithelial scales. These scales are constantly being shed and re-formed; and if the detached epidermis is removed, a red granular surface is exposed. The thickened and elevated area gradually enlarges, forming a superficial hard tumour, which is raised above the level of the surrounding skin, with an abrupt margin. For some time the tumour moves with the skin in which it has grown; but, as it extends, it becomes fixed to the underlying tissues, and a growth is formed, which may either be of very limited extent, or may involve an area of considerable size. Eventually the growth ulcerates; or ulceration may occur so early that the initial indurated stage may not have been noticed, and the first morbid appearance that attracts attention is a small, dry, yellow or blackish crust covering a fissure in the epidermis.

Not infrequently the first development of epithelioma is in a warty growth which becomes fissured after having remained of a simple nature for a long period of time. The wart finally indurates, then ulcerates, and a typical epithelioma is the result.

The *epitheliomatous ulcer* has usually an uneven surface, and is irregularly bounded by hard, livid, everted edges. It is covered by minute granulations or by a greyish crust, which after removal is quickly renewed. It discharges a thin, scanty, sanious ichor, which is usually inoffensive; and it bleeds easily when touched. In some cases there is very little or no pain. In other cases the pain is severe and lancinating, or there may be stinging or pricking sensations, which cause frequent scratching and consequent irritation. The ulcer is usually round, oval, or elongated. Its essential character is the hardness of its borders and base, which is produced by the infiltration which is an invariable feature of the disease. The indurated boundary can be felt as a distinct mass adherent to the subjacent tissues. The borders are generally raised, sinuous, or nodulated, frequently everted, and sometimes undermined. The surface of the ulcer is concave, unequal, nodular, or warty. As the disease progresses, the ulceration extends in depth, and causes destruction of the subcutaneous tissues, muscles, and bones. Whilst ulceration and destruction are proceeding in the centre, the small papillary nodules with a scaly surface, which mark the early stage, are found on the circumference of the sore, and they may increase till they form small tumours before being finally involved in the extension of the ulceration.

In course of time the lymphatic glands corresponding to the affected part become enlarged, being felt at first as small, hard, movable, isolated tumours producing little inflammatory reaction. As they increase in size they

become incorporated in a single, somewhat lobulated mass, which eventually softens. From the infected glands the disease may extend further towards the trunk, but more frequently the proximate glands alone become cancerous. The affected glands may inflame, suppurate, and lead to cancerous ulcers, in which the peculiarities of the disease are reproduced. Secondary epitheliomatous growths are occasionally, but not frequently, found in the internal organs.

In the earlier stages of an epitheliomatous growth the destruction may take place chiefly in the corium, in which case the growth is not much elevated above the normal level of the skin. If, however, development takes place chiefly in the papillary layer, we have a permanent and warty growth, whilst if it grows freely in the subcutaneous tissue we have a deep-seated, flat, or rounded mass. These different varieties may more or less intermingle, and thus in the earlier stages of an epithelioma we may have a variety of appearances. This depends to some extent on the anatomical peculiarities of the part in which the disease develops. In all cases before ulceration takes place the affected part is unusually hard and enlarged. The lip, for example, pouts and projects like one overgrown.

The chief varieties of epithelioma are the *superficial* and the *deep-seated* forms, which respectively depend on the greater or less resistance opposed by the *pars reticularis corii* to the extension inwards of the epithelial growth. If the morbid epithelium be for a long time unable to penetrate the strong, firm bundles of the corium, and contents itself with 'cropping off,' as Erasmus Wilson has expressed it, the highly organised *corpus papillare*, we have a typical superficial form which, whilst it extends on the surface, makes an effort at healing in the centre. The disease presents the appearance of an extending cicatrix somewhat like that of a burn, the centre appearing to be healed whilst the advanced border which surrounds the cicatrix is in a state of slow ulceration. But when, either from the greater destructive power of the cancerous epithelium or from weakness of the fibrous tissues of the corium, the growing epithelium pierces the *pars reticularis*, the clinical aspects are much more serious, and are very different. The growth having once penetrated into the subcutaneous tissue, extends, usually with comparative rapidity, producing the destruction which is characteristic of deep-seated epithelioma. When the chief feature of the epithelioma is a projecting papillary growth, we have the so-called *papillary epithelioma* or *malignant papilloma*. This form is not infrequent when the disease develops in a wart. It may have begun as a papilloma or a papillary growth, or may develop subsequently on a superficial or more frequently on a nodulating,

ulcerating epithelioma. It may project considerably above the level of the surrounding skin, and may be seated either on a broad base or have a constricted neck, assuming a fungating form. The surface is formed of cicatrices and enlarged papillæ, which are cylindrical, pyriform, or conical, and single or in clusters. They are very vascular, the surface chiefly covered with an opaque, white cuticle, of a pink, vermilion, or brightly florid hue. When the loose epidermic scales are washed off, we have the granulated and lobulated surface known as 'cauliflower-like excrescence.' The epithelial cells may contain melanotic matter, which is also found between the cells, and we have then *melanotic epithelioma*. In *discoid epithelioma* there is a sharply bordered circular or oval disc, the margins being bounded by healthy integument, which is raised and slightly everted by the growth.

An epithelial cancer may grow up in the form of a cone, although such cases are rare. Sir James Paget removed one from the lower lip half an inch high, and nearly as much in diameter at its base, and another in a chimney-sweep's neck of a similar kind. In both cases the growth was covered with a thick laminated scab, not easy at first to distinguish from syphilitic rupia. Mr. Henry Morris has seen one in the outer dorsum of the foot in a man aged fifty-six. Or the growths may even be pendulous. Sir James Paget refers to growths on the lower lip and anus consisting of masses of very firm granulations far overhanging the adjacent healthy skin.

The tendency in all these forms is to produce continued destruction of healthy tissue, unless the morbid growth is removed by operation. In the later stages of the malady exhaustion supervenes, and the patient eventually succumbs, worn out by pain and increasing discharge from the ulcerating surface.

**SEATS.**—Epithelioma may develop at any part of the integument, but is most frequent on the *face*. In 102 cases 78 were on the face, 48 of these being in the lip, 4 were on the extremities, and 10 on the mucous membrane of the mouth. The local ravages may be very extensive before they cause death, and in the face may produce hideous deformity, laying bare the bones, teeth, and fauces, destroying portions of the skull until the *dura mater* is exposed.

The *lower lip* is a very common seat of epithelioma, which commences there either as a warty growth, or as a fissure from the edges of which the disease spreads. The whole lip becomes thickened and hard, projecting outwards, and allowing the saliva to flow from the mouth. The surface ulcerates progressively; and if the affected tissue is not removed, the disease extends to the mucous membrane of the cheeks, giving rise to

ulceration, and eventually the throat and bones become affected.

In the *penis* epithelioma may commence as a nodular swelling of the prepuce, or as a hard scirrhous infiltration of the glans, or between the prepuce and glans. In the latter case a discharge may be the first symptom observed. A tight prepuce and phimotic condition of the parts favour the development of the disease. In epithelioma of the glans and sulcus there early occurs thickening of the dorsal lymphatic vessel of the penis, which can be felt as a thick, hard, nodulated tract extending towards the pubes, to be continued towards the inguinal glands, which enlarge into hard, nodulated masses. Abscess and ulceration eventually occur.

Primitive epithelioma of the *vulva* frequently develops as a small warty tumour; at other times it is preceded by pruritus or a long-standing leukoplakia. The point of origin is not infrequently on the mucous surface of the labium majus, or at the junction of the skin and mucous membrane. There is usually a discharge of bloody, ill-smelling serum. A tumour may form as large as a nut or hen's egg, with a hard, shining, papillated surface. It may or may not be pedunculated. The labium minus may be the seat of infiltration of a hard consistence and reddish colour, and there may be a tumour, more or less pedunculated, of the clitoris. After the first symptom of pruritus there is a sensation of cold and fulness of the pelvis, especially in walking or standing. The progress is usually slow. At the beginning, epithelioma of the vulva may be mistaken for chronic granular vaginitis, or non-malignant tumour, and in the ulcerative period for a primary syphilitic sore or a gumma. The severe lancinating pains and extension of the ulceration remove the doubts which may have existed during an earlier period.

Epithelioma of the *anus* is rare. It occurs as a notched and protuberant growth at the anal orifice, and extends into the gut.

In the *limbs*, usually the leg, epithelioma mostly develops in cicatrices, varicose ulcers, syphilis, and lupus.

Epithelioma of the *mucous membrane* of the nose, conjunctiva, vagina, and rectum may appear either primarily, or secondarily as an extension from the adjacent skin. Cancer of the tongue and mucous membrane of the cheek is not uncommon, in which situations it may develop on the seat of syphilitic change, or on a patch of leukoplakia. On the tongue it begins as a raw granulated surface, which is sometimes thickly set with white points, tender to pressure; and it may be the seat of pain, nodular infiltration developing under the sore. Sometimes the nodular infiltration precedes the ulceration. On the mucous membrane of the cheek epithelioma is rarer, and is generally flat but sometimes fungoid when it occurs.

#### ANATOMICAL CHARACTERS AND PATHOLOGY.

The pathology of epithelioma may be summed up briefly in describing it as an epithelial neoplasm, which leads to the destruction of the vascular tissues amongst which it grows. From the rete mucosum there occur offshoots of epithelial growth, which travel along the papillary layer, pierce the corium, and (wherever they grow) produce, in the first instance, well-marked developments of inflammatory action. Wherever the epithelioma comes in contact with the vascular tissue, it acts like a poison, and evidences of destruction are found. The essential feature is not that there is a new growth of epithelium, but that there is a growth of a new kind of epithelium, which is, in effect, a connective-tissue poison. Like all poisons, this epithelial poison exerts an irritating influence on the adjacent blood-vessels, as is witnessed by abundant small cell infiltration, serous effusion, redness, and other evidences of engorged blood-vessels. Cancerous epithelium has then two abnormal qualities: one in that it grows inwards into the connective tissue; the second, that wherever it grows it produces inflammatory action. When a vertical section is made through the edge of an epitheliomatous ulcer, we find on the outer border which is adjacent to the healthy tissue prolongations inwards of the rete mucosum, whilst in the connective tissue of the papillary layer and cutis we find sections of apparently isolated masses of epithelial growth. These isolated masses, which are mostly in a cylindrical form, were really in unbroken continuity with growths from the rete; but as the growths, after they have penetrated downwards, follow to a certain extent the lines of least resistance, they cease to be in a direct line with the interpapillary cone from which they have come, and therefore, in a thin section, appear to be detached. It is this apparent isolation that led to the erroneous interpretation that epithelial cancer was caused by a heterologous growth of connective-tissue corpuscles. That in cancerous tissue the epithelium takes its origin in the rete mucosum is a fact that was recognised so long ago as 1852 by Hannover, but was lost sight of for many years after Virchow's views on cellular pathology became generally accepted. It was again clearly shown by Thiersch in 1865.

There is a special epithelial physiognomy peculiar to the morbid growth in each individual case. In many instances the cell columns undergo an extreme degree of horny change, and we find in the sections groups of very large horny cells clustering concentrically, giving rise to the so-called laminated capsules or 'nests.' In some cases the cell-columns do not take on the extreme horny metamorphosis, all the cells retaining the ordinary apparent characters of rete cells.

The large horny cells frequently break down, and in their cavities numbers of nuclei are found, an appearance which has suggested the theory of so-called 'mother' and 'daughter' cells. The appearance is, however, equally in harmony with the probable theory that leucocytes have found their way into the interior of the broken-down cells.

The new cells are generally supposed to be formed by cell-fission—a process, however, which is not found when sections are examined. Whatever the exact method may be by which this epithelial growth takes place, there is no reason to believe that in principle it differs from that which is characteristic of increase of epithelium in healthy conditions.

When the changes in the connective tissue which lead eventually to ulceration are carefully scrutinised, it is found that under the influence of the epithelial poison there is an extensive emigration of white and red corpuscles, a splitting up of the bundles of connective tissue into the primary or elementary bundles of which they are composed, and eventually disintegration of the bundles altogether, a granular detritus taking their place. Immediately under and bounding the ulceration, masses of small cells are found, numbers of engorged and dilated blood-vessels, and many free blood-corpuscles.

The diseased epithelium not only possesses the power of destroying the connective tissue, but finds its way into the lymphatic vessels, and by them into the lymphatic glands. Occasionally it is conveyed into the viscera, leading to cancerous deposits in the internal organs.

Attempts to associate cancerous developments with the presence of micro-organisms have not yet been confirmed. Dr. Yakimoff has stated that in cancer the blood-vessels, not only near the tumour but throughout the body, acquire thickened walls with diminished lumen. Mr. Ballance and Mr. Shattock have described in cancerous tissue, after sterile incubation, a projection of nuclear granules into the cell protoplasm, and they suggest that the stimulus which excites the epithelial growth may be due to these particles.

**DIAGNOSIS.**—Before ulceration has taken place it is possible in the early stage to confound epithelioma with sebaceous *acne*, with *warts*, and with certain forms of *lupus*. If in *acne sebacea* the crusts on the affected part are removed, by rubbing with soft soap or other means, a healthy skin with distended sebaceous follicles is laid bare, the disease not extending deeply into the true skin. In distinguishing between epithelioma and *lupus*, it is to be borne in mind that in *lupus* the patient is, as a rule, either young or has a history of having suffered from the disease in early youth. In *lupus* the characteristic infiltration will be found on or near the borders of the affected part. In an ulcerat-

ing *lupus* the edges of the ulcer are soft and regular and not everted. The ulceration extends more on the surface than in depth. It must not be forgotten that epithelioma may develop in the seat of *lupus*.

A *tubercular syphilide* is distinguished by the colour and grouping of the tubercles, which are multiple; whilst epithelioma begins usually by a single tubercle, runs a slower course and lasts longer, and is not affected by a course of iodide of potassium. The diagnosis between epithelioma and syphilis is sometimes difficult, and without microscopical examination is occasionally impossible. Syphilomata presenting this difficulty are not infrequent on the tongue and lower lip, the diagnosis being rendered difficult by their late appearance, and often by their long period of latency. In such cases it is of great importance that a small portion of the affected surface should be excised and subjected to microscopical examination. In syphilitic ulcers the ulceration progresses usually more rapidly than in epithelioma. The ulcers are multiple, whilst in epithelioma the ulcer is usually single. Whilst the tissues surrounding the epitheliomatous ulcer are infiltrated and hard, in syphilis the diseased surface is usually bounded by apparently healthy skin. The pain which is frequently present in epithelioma is not usually present in syphilis, and when it is present it yields to palliatives.

It may be difficult to distinguish between a *simple wart* and an epithelioma which begins in a warty growth. If a tumour of this kind develops in an elderly person, shows a tendency to spread, and is the seat of frequent desquamation with excessive formation of epidermis, the probability is in favour of epithelioma.

It is sometimes not easy to distinguish epithelioma on clinical grounds alone from *rodent ulcer*, which is a form of superficial cancer depending on a specific epithelial cell. A typical epidermic epithelioma and a typical rodent ulcer can be distinguished; but there are cases in which, without microscopical examination, the diagnosis for a time is impossible. Rodent ulcer produces less inflammation in the surrounding tissues, and usually after ulceration occurs; it takes place at first slowly and superficially. If the glands become affected we may feel confident that we are dealing with an epithelioma. Either of these forms may occur on any part of the body, but in the majority of cases epithelioma occurs on the lip, whilst rodent ulcer is found on the upper part of the face. The rolled-over edge, which is frequent but not constant in rodent ulcer, is very characteristic. See **RODENT ULCER**.

**PROGNOSIS.**—The prognosis in epithelioma of the skin is not so grave as that in other forms of cancer, because its course is slow, and because it is frequently well within reach of radical treatment. If removed sufficiently

early and completely it will not return at all. Metastasis is rare. The gravity in any special case depends upon whether the ulceration remains localised on the surface or penetrates deeply. In the deep-seated form the danger to life is considerable. The disease may last a long time before the health becomes seriously affected. Unless, however, relieved by successful treatment, the patient's strength eventually fails, the characteristic pain of cancer increases, and death usually takes place from exhaustion; sometimes from hæmorrhage caused by blood-vessels being exposed in the progress of the disease.

**TREATMENT.**—No medicines administered internally have any effect on this disease; but so long as the new-growth remains single and is within the reach of surgical treatment, we may hope for a successful result. As soon as the nature of the growth is ascertained, the whole of the diseased tissue should be removed, where this is possible, either by the knife or with caustics. The surgeon will naturally, when it is possible, have recourse to a cutting operation; and where the ulcer has attained a certain size and there is considerable infiltration present, complete excision of the whole of the diseased tissue is a very effectual method of dealing with the disease. In the early stages of the affection there is no doubt that it is within the reach of caustics. An epithelioma in a limited warty condition may, when the affected surface is small, be successfully treated in this way after scraping with a curette. In severe cases, too advanced to be reached by the knife, the progress of the disease may sometimes, to a certain extent, be arrested by cauterisation, under which the condition of the ulcerated surface improves, pain is diminished, the discomforts of the patient's condition lessened, and life prolonged. If the knife is used, it is necessary to cut well beyond the affected tissue, as there is little doubt that relapses, when they do occur, are due to the fact that a nidus of infected cells has been left behind, although they had not developed sufficiently to enable the surgeon to recognise the extent to which the disease had spread. We have found by microscopical examination of excised epitheliomata, that sections from the part next the line of incision have contained cancerous epithelium between the connective-tissue bundles.

The caustics generally used are caustic potash, chloride of zinc, pyrogallic acid, and arsenic. Caustic potash may be used either in stick or in solution. It has the advantage of being thorough in its action, and of not leaving much pain after it has been used. The growth, of course, should be thoroughly cauterised. Pyrogallic acid is used as a 10 per cent. ointment, which is spread on linen and kept continuously applied for from three to six days. It is less painful than chloride of zinc. Chloride of zinc is used as a paste in

the proportion of one part to three parts of gum mixed with a little water. The paste is laid over the diseased surface, which must be blistered if the skin is entire, the surrounding healthy skin being protected by adhesive plaster. A strip of plaster cover should be placed over the whole. The paste will destroy structures equal to three or four times its own thickness. The zinc paste which is used at the Middlesex Hospital is composed of equal parts of chloride of zinc and liquor opii, with sufficient flour to make it into a paste. Resorcin has been recently recommended for epitheliomata. There are cases in which it answers well; in other cases its action does not seem sufficiently energetic. The writer has used it as incorporated in Beiersdorf's (Hamburg) gutta-percha plasters, aiding its action by painting the surface with a solution in alcohol. The treatment is comparatively painless, and interferes little with the comfort of the patient. Lactic acid has also been recommended for epithelioma. It is supposed to have a destructive effect on the diseased tissue, whilst it spares the healthy structures; but the alleged advantages have not yet been sufficiently confirmed.

Amongst the various caustic pastes which have been used in epithelioma arsenical paste, as regards efficiency, occupies a high place, although, on account of the danger attending absorption, it requires special care. The writer cannot speak from his own experience; but he has the testimony to the merits of the paste recommended by Dr. Marsden of his friend Dr. MacLaurin of Sydney, who has used it extensively in hospital and private practice, and is satisfied that for a considerable number of cases it possesses very great advantages.

G. THIN.

**EPITHELIUM, Diseases of.**—The different kinds of epithelium must be separately considered, as they differ in their pathological relations—namely, *squamous* and *cylindrical* epithelium of mucous surfaces; *serous* epithelium, or *endothelium*; and finally *spheroidal* or *glandular* epithelium. The last kind will be better treated of under the heads of the several glands.

**A. DISEASES OF SQUAMOUS AND CYLINDRICAL EPITHELIUM.**—1. **Catarrhal Inflammation.**—Both varieties of epithelium occurring on mucous surfaces are subject to inflammation, which usually takes what is called the catarrhal form.

Catarrhal inflammation is characterised by hyperæmia, swelling of the tissue, rapid production and casting-off of cells, and increased production of the normal mucous secretion of the parts, without the production of any coagulable exudation, or any layer of new material. The cells thrown off are partly epithelial, partly leucocytes or pus-cells. The

secretion contains mucin. The proportion of the various factors of catarrhal inflammation varies greatly, sometimes hyperæmia with swelling, sometimes cell-production, sometimes fluid secretion predominating; and these differences sometimes mark different degrees or stages of the inflammatory process. When the number of leucocytes thrown off is very large, the catarrh becomes purulent, which differs only from the other in degree. Catarrhal inflammation is the ordinary result of irritation applied to a mucous surface; but it persists after the irritation has ceased, and has a marked tendency to become chronic. While the chief share in producing and maintaining the phenomena of catarrh must be referred to the condition of the blood-vessels which keeps up the congestion, and allows increased transudation and copious emigration of leucocytes, the part played by the epithelial cells in these processes is a point of great interest and importance, though as yet imperfectly determined. These cells, whether squamous or cylindrical, enlarge and alter in shape, sometimes containing vacuoles or mucous drops; while there must be (since so many are shed) a rapid new formation of them; but the seat of this new formation, whether in the normal or the pathological condition, is still obscure. All that can be clearly seen is that new epithelial cells grow up from the basement membrane by the side of the old ones. Further, it is not unusual to find swollen epithelial cells which show division of the nuclei or partial division of the cell itself, and others which show within their substance several smaller, roundish bodies, with the general character of young cells. These appearances have been regarded as indicating (1) multiplication of cells by fission, (2) endogenous cell-formation within the mother-cells, and (3) the origin of the numerous pus-corpuscles seen on the inflamed surface. There is, however, no reason for thinking that new epithelial cells, or still less that pus-corpuscles, are formed in this way. The presence of pus-corpuscles or young cells within the epithelial cells is probably an accidental complication, the young cells which possess the power of migration being absorbed into the protoplasm of the epithelial cells, so as to appear as if originally formed there. The production of vacuoles or mucous droplets in the epithelia appears to show an abnormally rapid production of mucus, and indicates also the death of the cell producing it. It is, therefore, an evidence of excessive mucous formation, and explains also the rapid desquamation of the mucous epithelium.

**2. Croupous Inflammation.** — Croupous inflammation is distinguished by showing, in addition to hyperæmia and swelling, the production of a layer of new material, or false membrane, easily detached from the surface. This form is usually, if not exclusively, seen on surfaces covered with cylindri-

cal epithelium, as in the air-passages. The false membrane, composed of fibrin cementing together layers of detached epithelium and leucocytes, does not owe its origin to any alteration of the epithelium itself.

Croupous inflammation of epithelial surfaces has been regarded as always indicating some specific form of inflammation. It now appears, however, that it may be produced by simple irritation, such as that which produces the catarrhal form, provided the irritation be sufficiently intense and destroys the superficial epithelium.

**3. Diphtheritic Inflammation.** — This is a name used with much variation of meaning, but generally to signify a process in which there is production of a false membrane closely adherent to the epithelial surface, and which is accompanied by some degree of necrosis or gangrene. In the production of the diphtheritic false membrane an important part has been assigned to the epithelium, it being held that the new material, which appears like exuded fibrin, is really produced by a metamorphosis (the so-called *fibrinous transformation*) of the pavement epithelium. It is pretty clear that when this condition occurs on surfaces covered with this variety of epithelium some such change in the epithelium does take place, but not that the membrane is made up wholly or even in any large degree of such altered cells. Moreover, even this cannot be clearly traced on surfaces covered with cylindrical epithelium. The membranes consist in large measure of cast-off epithelium, and also (as the writer holds) partly of fibrin, though the presence of the latter constituent is denied by some authors. It should be noted that the terms croupous and diphtheritic inflammation, as here used, are not synonymous with the diseases named croup and diphtheria. Micro-organisms, either micrococci or bacilli, especially the former, are always found in the diphtheritic false membrane. Some may be regarded as mere saprophytes, living in the dead tissues; but there is apparently at least one species, possibly more than one, capable of originating the morbid process, and causing necrosis of the epithelium. See DIPHTHERIA.

**4. Fatty Degeneration.** — It is very common to find the protoplasm of both squamous and cylindrical epithelial cells dotted with oil-globules, so much so that this must be considered normal, to a certain extent, in some glandular epithelium (as kidney), and does not appear to interfere with the function of the cells. When the fatty change, however, is extensive, and more especially when the whole body of the cell is opaque, the condition must be regarded as one of fatty degeneration (see FATTY DEGENERATION). This is seen in the stomach in cases of alcoholism, in poisoning with metals or phosphorus, and in cases which are probably nothing more than chronic catarrh. It is also seen in the alve-

ular epithelium in pulmonary emphysema. Fatty degeneration in epithelium appears to be a process leading to atrophy or destruction.

**5. Mucous or Colloid Degeneration.** Epithelial cells, especially in parts which are naturally adapted to the production of mucus, particularly cylindrical epithelium, are liable to undergo a form of degeneration in which a portion of their protoplasm becomes converted into mucous substance, and thus liquefied. Cells having this character are often seen in catarrhal conditions of the mucous membrane of the air-passages, but the change is probably only an exaggeration of the physiological production of mucus in the interior of the cell. It has not been traced in squamous epithelium.

**6. Albuminoid (Waxy or Amyloid) Degeneration.**—This form comparatively rarely affects epithelial cells, but still in cases of albuminoid disease we may sometimes find that the mucous surface of the intestines is stained with iodine in the characteristic manner. In one or two cases the writer has observed a similar change in the surface of the pelvis of the kidney, and even the bladder, where there has been albuminoid disease of the kidney.

**B. DISEASES OF ENDOTHELIUM.**—The single layer of flat epithelium found on serous surfaces, now generally known as *endothelium*, differs also in its pathological relations.

**1. Catarrhal Inflammation.**—This is unknown on serous membranes, their characteristic form of inflammation being exudative and fibrinous, corresponding thus to the

**2. Croupous Inflammation of mucous surfaces.** In this inflammation the endothelium does not necessarily take any part; though when the inflammation is once established, the endothelium, in part, is simply shed, in part shows changes of a proliferative kind; cell-division, multiplication, and endogenous cell-formation being observed here with less ambiguity (as it appears to the writer) than in the epithelium of mucous surfaces. Similar changes appear to occur in chronic inflammation of serous surfaces, and to play an important part in the production of fibrous adhesions between opposing surfaces.

**3. Fatty Degeneration.**—Endothelial cells are also subject to fatty degeneration, which may be very clearly seen in surfaces macerated by a collection of fluid, as in serous effusions of the peritoneum or pleura.

The other pathological changes of endothelium have not been studied.

The epithelium (or endothelium) lining the inner surfaces of the walls of arteries and veins closely resembles the endothelium of serous surfaces. It is very subject to fatty degeneration, as may be seen on examining atheromatous arteries. A distinct fatty degeneration of this tissue also occurs quite independently of atheroma, or of any in-

flammatory process. Proliferative changes have also been traced by some observers in the process of occlusion of ligatured arteries or veins obstructed by thrombosis; but they do not appear to play any important part in idiopathic disease.

**C. DISEASES OF GLANDULAR EPITHELIUM.**—The diseases of glandular epithelium are best spoken of under the head of diseases of the several glands. See BREAST, Diseases of; SALIVARY GLANDS, Diseases of; &c.

J. F. PAYNE.

**EPITHEM** (ἐπί, upon; and τίθημι, I place). A general term for a class of external applications which are soft and moist, such as poultices and fomentations. See FOMENTATIONS; and POULTICE.

**EPIZOA.**—See ENTOZOA.

**EPULIS** (ἐπί, upon; and ὄδον, the gum). See MOUTH, Diseases of.

**EQUINIA** (*equus*, a horse).—A synonym for glanders. See GLANDERS.

**ERETHISM** (ἐρεθίζω, I irritate).—A condition of excitement or irritation, affecting either the whole system or a particular organ or tissue. The word has been especially applied to the condition of the body in the early stage of acute diseases, and also to that induced by the too free use of mercury (*mercurial erethism*). See MERCURY, Poisoning by.

**ERGOTISM.**—SYNON.: *Morbus cerealis*; Fr. *Ergotisme*; Ger. *Ergotismus*; *Kriebelkrankheit*.

**DEFINITION.**—A disease due to the action of ergot.

**ÆTIOLOGY.**—This disease derives its name from the fact that it is the result of the ingestion of ergot—the sclerotium of a fungus called *Claviceps purpurea*, which grows parasitically in the ear of the rye. In some seasons this form of blight affects the grain so extensively that ten per cent. of the meal may consist of ergot. The phenomena to be described as symptoms of ergotism have been regularly and exclusively traced to the use of articles of food made from rye-meal thus contaminated. The appearance and severity of the disease vary with the amount of ergot consumed. Children at the breast are never attacked. Ergotism has frequently broken out in well-marked epidemics, after unfavourable harvests. In ruder times it constituted a severe form of scourge; but now it usually occurs sporadically, or is limited to families or small communities.

**ANATOMICAL CHARACTERS.**—Ergotism is characterised by definite anatomical changes, mainly in connexion with the vessels and the spinal cord. The arterioles are found *post mortem* to be contracted, and to be the seat of a peculiar hyaline degeneration of the

intima, with thrombosis and diminution, or even complete disappearance or occlusion, of their lumen. Gangrene is the ultimate result of these morbid changes. The posterior columns of the spinal cord are the seat of a degenerative or sclerosing process, somewhat resembling the lesion so familiar to us in locomotor ataxy.

**SYMPTOMS.**—Within a few days of the first ingestion of rye-meal contaminated with ergot, the ordinary phenomena of irritant poisoning are developed, namely, vomiting, diarrhoea, severe abdominal pains and cramps, and general depression—giddiness and headache being specially marked.

Along with the preceding, certain specific symptoms gradually make their appearance. The first and most characteristic of these is formication (a sensation of insects running along the skin), attended with severe itching of the extremities. The other special senses, such as vision and hearing, may also become disordered. Occasional tearing pains occur in the limbs, and clonic spasms of the muscles. Ravenous hunger is said to be a striking symptom in some instances. The pulse is infrequent and small. Respiration is not markedly disturbed.

The remaining phenomena peculiar to ergotism are usually described as belonging to two forms, the *gangrenous* and the *spasmodic*, according as the circulation or the nervous system is chiefly affected.

(a) *Gangrenous ergotism.*—The gangrene produced by ergot is peculiar only in respect of its cause. The toes, fingers, feet, ears, and nose are the parts most commonly attacked. The incipient discoloration, pain, and swelling are observed within a period of two days to three weeks from the commencement of the other toxic symptoms. The necrotic process passes through the ordinary stages of development; may be either 'wet' or 'dry,' and advances to complete separation of the part, if this have not been previously removed by operation.

(b) *Spasmodic ergotism.*—The leading symptom of this form of the disease is the occurrence of severe intermittent cramps or painful spasms, specially affecting the lower extremities. These develop into tonic contraction of the muscles, with fixation of the limbs; and end perhaps in general convulsions, prostration, various kinds of mental derangement, unconsciousness, and death. Abortion does not appear to be of frequent occurrence.

**COURSE, DURATION, AND TERMINATIONS.**—Many cases of ergotism are acute rather than chronic; but when gangrene appears, the course may be very protracted and variable. Spasmodic ergotism may last from two weeks to as many months. The mortality is said to have fallen from sixty to ten per cent. In a few cases, resolution occurs in affected extremities.

**PATHOLOGY.**—Beyond its effect as an irritant poison, the specific influence of ergot is mainly exerted upon the organs of circulation, the central nervous system, and the uterus. The different effects have now been satisfactorily traced to the actions of the different organic constituents of the diseased grain.

Cornutine, an alkaloid, one of the four important organic constituents of ergot, produces slowing of the cardiac rhythm by stimulation of the vagus at its central extremity. The contraction and hyaline change of the walls of the arteries are due to the action of a second constituent, sphacelinic acid. The result of these effects on the heart and arteries is that the blood-pressure rises. The gangrene is readily accounted for by the vascular changes, and is thus definitely referable to the sphacelinic acid.

The painful spasms, as well as the formication and other sensory disturbances, are the direct result of the action of the cornutine upon the spinal cord.

The action of ergot upon the uterus is explained by some authorities as due to stimulation by cornutine of the uterine centre in the cord. Other authorities maintain that it is set up by the local anæmia produced by the vascular contraction; whilst others, again, consider that ergot acts directly upon the muscular fibres of the organ.

**DIAGNOSIS.**—The occurrence of gangrene in a number of young and previously healthy persons, in countries where rye-meal is an article of food, should remove all difficulty from the diagnosis of ergotism. The spasmodic form of the disease can usually be distinguished from epidemic cerebro-spinal fever by the absence of pyrexia.

**PROGNOSIS.**—The prognosis depends chiefly upon the early recognition and removal of the cause. The probability of the escape of affected extremities may be estimated by the degree to which the gangrenous process has advanced.

**TREATMENT.**—The treatment of ergotism must commence with the removal of the cause. An attempt must then be made to hasten the elimination of the poison by the cautious administration of emetics and purgatives; to allay the symptoms of gastro-enteritis; and to support the strength of the patient by internal and external stimulants, such as alcohol, warmth, and friction. The treatment of less acute ergotism, including spasmodic cases, is symptomatic. Gangrene must be averted by careful local stimulation—by means, for example, of warm fomentations; or treated surgically if it make its appearance. See GANGRENE.

J. MITCHELL BRUCE.

**EROSION** (*erodo*, I gnaw).—A superficial destruction of tissue, caused especially by friction, pressure, corrosion, or certain forms of ulceration.

**EROTOMANIA** (ἔρως, love; and *μαυία*, madness).—**SYNON.**: Love-melancholy; Satyriasis (in men); Nymphomania (in women); Fr. *Monomanie érotique*; Ger. *Liebeswuth*.—Insanity characterised by excessive sexual excitement; sometimes symptomatic of cerebral lesion, sometimes of disorder in the reproductive organs. See **INSANITY**.

**ERRATIC** (*erro*, I wander).—Wandering, shifting, or irregular. Applied to pains, eruptions on the skin, and other morbid phenomena, when they shift or move from place to place.

**ERUCTATION** (*eructo*, I belch).—**DEFINITION.**—The sudden escape or expulsion of gas from the stomach upwards, with or without an admixture of portions of liquid or solid food, or of gastric juice or other liquids.

**DESCRIPTION.**—The act of eructation may be voluntary or involuntary. In the former case a small portion of air is first swallowed, and by the over-distension thus produced the escape of a portion of the gaseous contents of the stomach is favoured. When involuntary, we must suppose that the cardiac orifice, which is closed in the normal state of digestion, is relaxed, and thus permits the rejection of portions of undigested matter. From the violence with which eructations often take place, we may also assume that the muscular coat of the stomach contracts spasmodically at the same moment that the relaxation of the cardiac opening occurs. The nature of the material rejected varies greatly. Sometimes it is tasteless, at other times acid, and in rarer instances alkaline.

**ÆTIOLOGY.**—Eructations occur in all gastric diseases attended with an undue formation of gas. They are constantly complained of in atonic dyspepsia, more especially in that form which occurs in elderly people, and are probably the result of an imperfect contraction of the stomach, preventing the due expulsion of the digested food into the intestine. They form a prominent and distressing symptom in dilatation of the stomach. In such cases the patient often complains of a sense of fermentation in his abdomen, and immense quantities of gas are expelled, generally mixed with an acid or acrid fluid.

**TREATMENT.**—The indications in the treatment of eructations are to prevent the decomposition of food, and the formation of gases and other products; to restore tone to the stomach, and remove any morbid condition of this organ; and to give remedies with the view of absorbing gases, or assisting the act of eructation.<sup>1</sup> See **STOMACH, Diseases of**.

SAMUEL FENWICK.

<sup>1</sup> Hepatic disorder frequently requires attention, with remedies directed to the liver. Lavage may be called for in the severe type of eructation which attends dilatation.—Ed.

**ERUPTION** (*eruptio*, a bursting forth).—This term is commonly applied to a pathological manifestation in the skin, more or less general; sometimes marked by colour, sometimes by prominence, but more frequently by both. When sudden and hyperæmic, a term derived from the efflorescence of a plant, namely, *exanthema*, is employed, as in the instance of the exanthematous eruptions—scarlatina, rubeola, roseola, and variola. The term is equally applicable to less acute forms of dermatosis, such as urticaria, eczema, impetigo, ethyma, acne, and furunculus; and is also used for still more chronic forms of disease, for example, psoriasis vulgaris; and for outgrowths of the skin due to aberration of nutrition, as in the instance of warts and molluscous tumours. ERASMUS WILSON.

**ERYSIPELAS** (ἐρυθρός, red; and πῆλλα, skin).—**SYNON.**:—Lat. *Erysipelas*; Fr. *Erysipèle*; Ger. *Erysipelas*. Popular names:—St. Anthony's Fire (English); the Rose (Scotch); *der Rothlauf*, and *die Rose* (Ger.)

**DEFINITION.**—Inflammation of the integument tending to spread indefinitely (*Royal College of Physicians' Nomenclature of Diseases*). The vagueness of this definition indicates the looseness with which the term is employed. The features common to all inflammations usually spoken of as erysipelatous are fever, sometimes apparently preceding the local phenomena; and an inflammation tending to spread indefinitely by means of the lymph-spaces and lymphatic vessels of the affected part.

**SUMMARY OF VARIETIES.**—Erysipelas is usually divided into: (a) *simple cutaneous*; (b) *phlegmonous or cellulocutaneous*; and (c) *cellular or diffuse cellulitis* (Nunneley). In the first of these the inflammation is seated in the cutis vera, the subcutaneous tissue being often œdematous, but not actually implicated in the morbid process. There is practically no tendency to supuration in simple cutaneous erysipelas. In phlegmonous or cellulocutaneous erysipelas the subcutaneous areolar tissue is primarily affected, but the skin covering the inflamed area is speedily implicated. If not relieved by treatment, the inflammation tends inevitably to diffuse supuration, often accompanied by extensive sloughing of the cutaneous and subcutaneous tissues. In cellular erysipelas or diffuse cellulitis, deep-seated planes of areolar tissue are affected, such as those of the pelvis. The course of the disease is the same as in cellulocutaneous erysipelas, tending to terminate in diffuse supuration and sloughing. In addition to these three chief varieties, erysipelatous inflammation of the lymphatic vessels and veins, and of serous and mucous membranes, is also described.

**ÆTIOLOGY AND PATHOLOGY.**—All these affections grouped together as erysipelatous belong to the class of infective inflammations;

that is to say, they are dependent upon a virus which increases in quantity in the affected area, thus causing the characteristic spreading of the inflammation. The virus is present in the fluids of the inflamed area, and the disease can be communicated from one individual to another by inoculation of the inflammatory products; and in the case of cutaneous erysipelas, at least, there seems reason to believe that the poison may also be carried by the air. In all erysipelatous inflammations the virus diffuses itself by the lymph-spaces and lymphatic vessels of the affected part, showing little or no tendency to invade the blood-vessels, and to be disseminated by the blood-stream to distant parts. It is probable that there is more than one virus which may thus give rise to a spreading lymphatic inflammation; but in the vast majority of cases clinically described as erysipelas, which have been submitted to examination, a micro-organism of a definite form has been found, which is believed to be the actual virus. In 1869 Hueter suggested that the virus of erysipelas was probably a micro-organism, and thought that he had found it in the discharges of the inflamed part. In 1880 Lukomsky, Tillmanns, and others discovered the presence of micrococci in the lymph-spaces of the skin at the advancing edge of the rash in cutaneous erysipelas; but in 1882 the relation of these organisms to the disease was clearly demonstrated by Fehleisen. A small piece of the skin from the advancing edge of the rash was removed with proper precautions to avoid contamination, and planted upon some nutrient gelatine. From this a film soon spread over the gelatine, which on microscopic examination proved to be a pure cultivation of a micrococcus. The organism belongs to the genus streptococcus—that is to say, it shows a tendency to grow in chains or pairs. It does not liquefy the gelatine in its growth. This organism, after cultivation for several generations upon gelatine, gave rise to typical erysipelas when inoculated on the ear of a rabbit. Subsequently inoculations were made on the human subject with the intention of setting up erysipelas for the relief of lupus or incurable cancer. Seven cases were thus treated and genuine cutaneous erysipelas followed the inoculation in six; the seventh patient had recovered from an attack of erysipelas a few months before. Other observations seemed to show that one attack of erysipelas confers upon the patient a short period of immunity to the disease.

In all forms of cellulo-cutaneous erysipelas and in diffuse cellulitis a streptococcus exactly resembling that described by Fehleisen is found in the fluid filling the lymph-spaces of the affected part, whether it be purulent or serous. It cannot be distinguished from that found in cutaneous erysipelas by its reaction to aniline dyes, by its mode of growth, or

microscopic appearances. To this micrococcus Rosenbach gave the name of streptococcus pyogenes. Cultivations of this organism cause acute suppuration when inoculated on animals; but it has lately been asserted that when inoculated superficially on the ear of a rabbit typical cutaneous erysipelas is set up. It has been stated that cultivations of the streptococcus erysipelatosus from the human subject injected into the subcutaneous tissue of the rabbit have given rise to deep-seated phlegmonous inflammation and suppuration. This is, however, denied by Fehleisen, who asserts that he has repeatedly injected erysipelas micrococci into the subcutaneous tissue and muscles of rabbits without causing inflammation or suppuration. The identity of the organisms must, therefore, be considered an open question at the present time. Clinically the simple cutaneous variety of erysipelas differs so widely from the subcutaneous forms that it is difficult to believe they are due to the same virus. In cutaneous erysipelas the streptococci are found in the lymph-spaces of the cutis at the advancing margin of the rash. Their presence excites the ordinary phenomena of inflammation—the blood-vessels are injected, and abundant migration of colourless corpuscles takes place. These seem to destroy the micro-organisms, which are not to be found in the more central parts of the rash. The persistence of the phenomena of inflammation in the parts in which no streptococci can be demonstrated must be supposed to be due to the damage done to the tissues by the chemical products formed during the growth of the organisms. The organisms never enter the blood-vessels locally, and though very probably some may find their way into the blood-stream by the lymphatics, there is no reason to believe that they multiply in the blood. The constitutional symptoms are therefore due to the entrance of the chemical products of the local process into the blood-stream, and not to any general infective process. In phlegmonous erysipelas and in cellulitis the streptococci can be easily demonstrated in the inflammatory exudation filling the lymph-spaces in the whole affected area, and not only at the advancing margin; but, as in the cutaneous form, the organisms have not been proved to infect the blood. Further evidence of the absence of blood infection is furnished by Fehleisen's experiment on the rabbit's ear. Having induced typical cutaneous erysipelas by the inoculation of a pure cultivation of the streptococcus, he amputated the ear with a Paquelin's cautery, with the effect of immediately relieving the elevation of temperature and all other constitutional symptoms. It is not proved, however, that the virus may not enter the circulation from the alimentary canal or the air-passages, and be carried by the blood, without actually infecting it, to some part

which, from local injury or other cause, may form a suitable point for its development. On the assumption that the streptococcus is the actual virus, this seems the only possible explanation of many cases of erysipelas, especially of the cellulose-cutaneous or cutaneous variety, in which no evidence of direct inoculation by a wound or otherwise can be obtained.

The streptococcus grows readily at ordinary temperatures on gelatine, agar-agar, and potatoes, and probably on almost any damp organic matter. It has been said to have been found on the floor of an affected ward. This explains the well-known fact that hospital wards are apt to become infected, case after case breaking out until the ward is closed and disinfected. In military practice, erysipelas is very rare except in cases treated in civil hospitals. In this respect it differs from hospital gangrene and pyæmia, which can always be generated by crowding the wounded in buildings of any kind. It seems probable therefore that the virus is really specific, like that of scarlet fever or smallpox. That it can be carried by the air seems also to be proved, as the streptococcus has been detected on potato cultivations made from the air of infected wards. The cultivations thus obtained gave rise to erysipelas when inoculated on a rabbit. There is little doubt, however, that it is more commonly conveyed by the hands or clothes of the attendants. Erysipelas certainly does not show the same degree of infectiousness as most of the acute specific diseases.

The micro-organism is killed in 45 seconds by a 1 in 30 solution of carbolic acid, and in 15 seconds by a 1 in 1,000 solution of perchloride of mercury.

All the evidence above given tends therefore to show that erysipelas is a local disease, dependent on the invasion of the tissues by a specific micro-organism, and that the constitutional disturbance is secondary to the local. The opposite view has been held by many authorities, who have classed erysipelas with the acute specific fevers, regarding the rash as the local manifestation of a general condition. The arguments in favour of the latter are briefly as follows: (a) In many cases there is a distinct period of invasion in which the constitutional symptoms are present without the local; (b) there is frequently no evident point of inoculation; (c) the constitutional symptoms in cutaneous erysipelas are often much more severe than the local inflammation seems sufficient to account for.

Against these it is urged by those who support the local theory: (a) that even during the apparent period of invasion without local phenomena, a careful examination will detect some swelling of the lymphatic glands or other evidence of local mischief; (b) that the minutest abrasion merely being necessary for inoculation, it is very easily overlooked; and (c) that when we consider the intensely

poisonous nature of the products of the growth of micro-organisms in albuminous fluids, the constitutional symptoms are not greater than might be expected. The results of the inoculations, and Fehleisen's experiment of amputation of the rabbit's ear above mentioned, and the fact that inoculation of the blood has never communicated the disease, nor have organisms been satisfactorily demonstrated in it, are further proofs of the local nature of the disease. Moreover the disease does not run a definite course like an acute specific fever. The fever continues as long as the rash is extending, and ceases as soon as that comes to an end. It must be remembered, however, that it is not proved that the blood may not serve as a carrier of the virus, though not as a medium for its growth, as in many other infective diseases—tubercle, for example. Granting therefore that erysipelas is an infective disease, and that the micro-organism above described is the actual virus, the predisposing causes may be divided into (1) *those favouring the growth and dissemination of the organism out of the body*; and (2) *those predisposing the patient to receive it*.

1. (a) *Accumulations of decaying animal or vegetable matter*, upon which the organism can develop, will predispose to the disease. Thus the presence of a dust-heap near a window of a ward, has been known to cause repeated outbreaks of erysipelas in the nearest bed. Dampness of the floors, from repeated washing, has also been said to predispose to erysipelas. Decomposing discharges from wounds, and want of cleanliness in dressings, are also important causes. (b) Certain meteorological conditions are said to predispose to erysipelas. East winds, low temperature, excessive moisture, cold and heat, have all been considered causes, but the evidence on these points is very unsatisfactory. It is certainly least common in the hottest and driest part of the year, and more common in spring and late autumn. (c) Bad ventilation leads to concentration of the virus and increases the chances of infection. (d) The virus can undoubtedly be carried by the medical and other attendants, but risk of this happening is greatly diminished by the proper use of antiseptics.

2. (a) A constitutional predisposition to erysipelas is occasionally met with, which is sometimes hereditary. The patient usually suffers from repeated attacks of the disease. One attack of erysipelas is therefore said to predispose the patient to another. (b) Exposure to the impure atmosphere of badly ventilated or overcrowded wards increases the liability to erysipelas. (c) Imperfect hygienic conditions generally, such as bad or insufficient food, impure water, &c., predispose the patient to the disease. (d) Diseases of the liver or kidneys make a patient especially liable to it. (e) The abuse of alcohol is one of the most important of all predisposing

causes. The great majority of the cases of fatal phlegmonous erysipelas occur in hard drinkers. (f) Age has no influence. (g) The disease is said to be more common in women, especially at the menstrual period. (h) The presence of a wound or abrasion to serve as the seat of inoculation is the only important local predisposing cause of erysipelas.

Erysipelas undoubtedly occurs in *epidemics*, and the type of the disease varies in different outbreaks. We know nothing of the causes which render the disease epidemic.

We shall now proceed to discuss in detail the several varieties of erysipelas summarised above.

**I. Simple Cutaneous Erysipelas.**—This is the most typical form of the disease.

**ANATOMICAL CHARACTERS.**—The *post-mortem* appearances of erysipelas are by no means characteristic. The redness of the inflamed area of course fades after death, leaving a faint yellowish tint. The skin feels hard and inelastic, and the subcutaneous tissue contains an excess of serous fluid. In very acute cases there may be the usual signs of blood-change seen in all malignant fevers: early *post-mortem* staining, imperfect coagulation of the blood, subserous petechiæ, swollen and soft spleen, and cloudy swelling of the liver and kidneys. Hiller states that microscopic examination of the blood before death shows many of the white corpuscles to have undergone degenerative changes and become converted into masses of highly refracting granules. Busk has described plugs of such altered corpuscles in the small vessels of the lung, and Bastian has observed a similar condition in the vessels of the brain. Microscopic examination of the affected part of the skin shows large numbers of migrating leucocytes, lying in the spaces of the fibrous tissue, amongst the fat-cells, and in the lumen of the lymphatic vessels. They are especially abundant round the small vessels. The micro-organisms found in the lymph-spaces of the skin at the advancing margin of the rash have been already described.

**SYMPTOMS.**—In simple erysipelas the constitutional symptoms often appear to precede the local. The invasion is marked by chilliness, seldom by an actual rigor; loss of appetite; general malaise; nausea, but seldom actual vomiting; headache; pain in the limbs; and the usual signs of pyrexia. The invasion is tolerably sudden. The temperature rapidly rises, to about 103° F., or higher. The rarer symptoms at this stage are epistaxis in adults, and convulsions in children. Usually within twenty-four hours of the invasion the characteristic cutaneous inflammation becomes evident. It may, however, commence simultaneously with the febrile disturbance, or be delayed even for two or three days. Frequently the lymphatic glands nearest to the part are swollen before the cutaneous eruption appears; afterwards

they are invariably enlarged and tender. The local inflammation usually, if not always, starts from some wound, scratch, or abrasion. It commences indifferently in a fresh wound or a granulating sore. When no wound can be recognised as its starting-point, it usually starts from the junction of mucous membrane and skin, most commonly from the corner of the eye, causing a swelling across the bridge of the nose. It may also start from the angle of the mouth, the external auditory meatus, or the anus. It may commence in the nasal fossæ or pharynx, and extend outward to the skin of the face. Possibly in all cases it starts from some slight abrasion which is scarcely to be detected (Trousseau). The inflamed skin is bright red in colour, with sometimes a yellowish tinge; the redness advances in all directions, but usually most rapidly in that of the lymph-stream. The advancing margin is irregular, sharply defined, and very slightly raised. The cutis is œdematous, and pressure with the finger-nail leaves a deep and abiding mark. Where the subcutaneous areolar tissue is lax, as in the eyelids or scrotum, it also becomes greatly swollen. In the limbs the subcutaneous swelling is great only in severe cases. In many cases small vesicles rise, which may coalesce, forming blebs of considerable size. These ordinarily contain clear yellow serum, which, in bad cases, may be stained with blood-pigment. As these bullæ burst they dry up, forming scabs on the surface, but no ulceration takes place beneath these scabs. The inflammation has but little tendency to end in suppuration; when this does occur it is in those parts in which the œdema has been greatest, as the eyelids. There is heat, tension, and pain in the affected part, and a peculiar sensation of stiffness, which may even precede the appearance of the redness.

The febrile symptoms which usher in the attack remain unrelieved so long as the redness continues to spread. The pulse is at first quick and full, but it soon loses force, and in bad cases becomes extremely rapid and feeble. It is by the pulse, more than anything else, that the gravity of the case is marked. The temperature seldom rises above 106° F., though 107.5° F. has been recorded. The daily variations are not great, there being merely the usual slight morning fall and evening rise. Delirium is not uncommon at night, even in mild cases. In erysipelas of the head it may be a prominent symptom. It is usually due to the blood-condition, and not, as was formerly supposed, to extension of the inflammation to the membranes of the brain. This, however, does occur in rare cases, especially in erysipelas of the orbit, or in that following a compound fracture of the skull. The tongue is always foul, and usually dry; in bad cases becoming cracked and brown, with sordes on

the lips and teeth. In erysipelas of the head the fauces are always red and congested, even when the inflammation has not actually extended to that part. The bowels are sometimes confined, but diarrhœa with offensive motions is liable to occur. There is nothing characteristic about the urine. As in other acute febrile diseases, it frequently contains a small quantity of albumen. In erysipelas of the head, when the disease reaches its height, the appearance is often hideous in the extreme, the features being completely obliterated by the swelling of the lax subcutaneous tissue, and the face further disfigured by the scabs formed by the dried blebs.

The duration of simple erysipelas is very uncertain. The cessation of the disease is marked by the inflammation ceasing to extend, and by a simultaneous fall of temperature, often very sudden. This may occur as early as the fifth day, or be delayed till the end of the second or middle of the third week. As the rash fades its margin loses its distinct outline, and the redness shades off insensibly. It is not uncommon to see the inflammation spreading at one part, fading at another. After the subsidence of the inflammation there is desquamation of the cuticle, and in erysipelas of the head often complete loss of hair, which is, however, never permanent. Even after a mild attack the patient's strength is much reduced, and he often remains weak and anæmic for a considerable time. Relapses are by no means uncommon. When death occurs from simple cutaneous erysipelas, it arises most frequently from exhaustion. It may also be due to the gravity of the blood-change. Occasionally the fatal termination is preceded by violent delirium ending in coma. Sometimes it is due to complications, as pleurisy or pneumonia, or in very rare cases meningitis. When sloughing of the skin or suppuration occurs, death may take place from septicæmia or pyæmia.

**VARIETIES.**—Some writers have divided simple erysipelas into *medical* and *surgical*—or *idiopathic* and *traumatic*, and have described these varieties as distinct diseases. They are, however, probably identical, for the following reasons: they closely resemble each other in mode of invasion, course, and pathological changes; infection from so-called idiopathic erysipelas will give rise to the traumatic form in patients suffering from an open wound; and during an outbreak of erysipelas in a surgical ward, patients without open wounds are occasionally attacked by the idiopathic form. Erysipelas has also been subdivided according to the part it attacks, as erysipelas faciei, capitis, scroti, &c.

Erysipelas occasionally affects the mucous membrane of the pharynx and upper part of the larynx. This form presents some peculiarities, and is spoken of as *erysipelatosus pharyngitis* and *laryngitis*. The invasion and constitutional symptoms are similar to

those of simple cutaneous erysipelas. There is a bright redness of the back of the pharynx and the fauces, always accompanied by considerable œdema of the soft palate and some swelling of the tonsil. The glands at the angle of the jaw are swollen and tender. The danger of this affection arises from extension to the glottis, causing œdema glottidis, with intense dyspnoea, expiration being more easy than inspiration, and both liable to obstruction by spasm. In such cases tracheotomy or laryngotomy may be required at any moment, to prevent death from asphyxia. In other cases the inflammation may extend forwards and appear on the face, either at the nostril or mouth, and afterwards extend as ordinary facial erysipelas.

Erysipelas occasionally attacks new-born infants, starting from the navel or genitals. This form has been spoken of as *E. neonatorum*. Serous membranes, especially the peritoneum, are said sometimes to be affected by erysipelas following wounds. In lying-in women the poison of erysipelas seems, in common with that of many other unhealthy inflammations, to be capable of causing puerperal fever.

Dermatologists have, according to their wont, invented a name for every possible variation. Thus when the inflammation spreads at one part while fading at another it has been called *E. ambulans* or *erraticum*; when spreading in a winding course, *E. serpens*; when causing small vesicles, *E. vesiculare* or *miliare*; when blebs form, *E. bullosum*; when there is much swelling, *E. œdematosum*, &c. Such names are useless, and might be multiplied *ad infinitum*.

**DIAGNOSIS.**—When erysipelas is fully developed it is scarcely possible to mistake the disease. During the stage of invasion, before the appearance of the rash, diagnosis is impossible. Simple diffuse inflammation around a wound or abscess is distinguished from erysipelas by the absence of the characteristic invasion, and of the sharply defined border. Simple erythema differs from erysipelas in the absence of fever, and in the eruption being composed of numerous isolated patches. Occasionally, in malignant smallpox, there may be much redness and swelling of the face before the appearance of the vesicles, but the symptoms of invasion are much more severe than those of erysipelas.

**PROGNOSIS.**—The prognosis depends chiefly upon the gravity of the general symptoms. The following are bad signs: high fever, violent delirium, excessive diarrhœa, early prostration, and very dry tongue with sordes. The extent of the inflammation is of less importance. Old age, disease of the kidneys or liver, and especially chronic alcoholism, add greatly to the gravity of the case. When erysipelas affects the pharynx there is always danger from œdema glottidis. In uncomplicated cases the death-rate is not high. Of

25 patients treated in the medical wards of University College Hospital for facial erysipelas from 1872 to 1876, only one died, and he was suffering from chronic Bright's disease.

**TREATMENT.**—1. *Constitutional.*—Erysipelas being a most exhausting and depressing disease, no antiphlogistic treatment is ever justifiable. Clear the bowels at the commencement of the attack, but avoid violent purgation. Only two drugs have any reputation in the treatment of erysipelas. The tincture of perchloride of iron, in large and repeated doses, has been strongly recommended by Dr. Reynolds and others, and is stated by some to act as a specific. To be of any use it must be given in doses of forty minims every four hours. Aconite, if administered as soon as the temperature begins to rise, is said to cut the attack short. It may be given in half-minim or minim doses of the tincture, at first every quarter of an hour for one or two hours, and afterwards hourly, till the skin becomes moist and the temperature falls, but its effects must be very carefully watched, to avoid dangerous depression. The *diet* must be as nourishing as possible—beef-tea, eggs and milk, &c. Solid food can never be taken during the advance of the disease. Stimulants are usually required, and the amount must be regulated by the pulse. Large quantities are often necessary.

2. *Local.*—Local treatment is very various. Warmth and avoidance of variations of temperature are essential. Cold is utterly inadmissible; it aggravates the inflammation, and tends to cause suppuration or even sloughing. Hot fomentations or hot baths may be employed when the part affected renders them admissible. In other cases dry warmth must be used; it is best obtained by covering the affected part with a thick layer of cotton-wool. Poultices should be avoided, as they needlessly irritate the skin, and are dirty and apt to get cold. With the application of warmth innumerable varieties of local applications have been recommended. These may be divided thus: (a) *Indifferent applications.* These are intended only to exclude the air, but they have the disadvantage of shutting in the secretion of the skin. The most common of these are collodion, oil, and a thick layer of flour or starch under cotton-wool. (b) *Sedative applications.* The most important remedy of this class is belladonna. It is best applied as a paint composed of equal parts of the extract and glycerine. It is especially useful when there is much inflammation of the lymphatic vessels and glands. (c) *Powerful astringents.* Valette, of Lyons, recommends a 30 per cent. solution of perchloride of iron; Higginbottom a solution of the 'brittle stick of nitrate of silver'—20 grains to one drachm of water. Before applying either of these, the skin must be carefully washed with soap and water to free

it from grease. The perchloride of iron must be rubbed in with a glove. (d) *Antiseptic applications.* Marshall recommends creasote made into a paste with kaoline; Dewar, equal parts of sulphurous acid (B.P.) and glycerine. Tincture of iodine is a common application. Koch recommends an ointment composed of creolin 1 part, iodoform 4 parts, and lanolin 10 parts. Hueter practised the subcutaneous injection of a 1 in 30 solution of carbolic acid. He stated that this causes an immediate arrest of the inflammation for a small distance round the puncture; if, therefore, the treatment be adopted at so early a stage that the area of inflammation can be surrounded by four or five punctures, the disease may be checked. Beyond this there would be danger of carbolic-acid poisoning. Kraske and Riedel recommend scarification of the advancing margin with a special instrument, and the subsequent application of lint soaked in a 1 in 500 solution of corrosive sublimate. (e) *Drawing a limiting line* in front of the advancing rash. This has been done with solid nitrate of silver and with blistering fluid. It is utterly useless. Wölfler recommends the application of pressure immediately in front of the advancing line of redness by means of adhesive plaster. He believes this prevents the invasion of the lymph-spaces by the micrococci.

Erysipelas of the fauces is best treated by the local application of a strong solution of perchloride of iron. If there be œdema glottidis the swollen parts must be scarified, and if this fails to give relief, tracheotomy may be necessary.

**II. Phlegmonous or Cellulo-cutaneous Erysipelas.**—This was described by Dupuytren under the name of 'diffuse phlegmon.'

**ANATOMICAL CHARACTERS.**—Incisions made into the inflamed part in the early stages show the spaces of the areolar tissue distended with a semi-solid inflammatory exudation; a little later this is found to be a turbid and puriform fluid; later still the subcutaneous cellular tissue is represented by masses of shreddy sloughs soaked in pus. Unless exposed to the air by incisions or by sloughing of the skin, these sloughs are free from any odour of decomposition, and contain no gas. The streptococcus already described can invariably be demonstrated in the exudation fluids of the affected part. There is nothing characteristic in the *post-mortem* appearances of the internal organs.

**SYMPTOMS.**—The invasion is usually marked by chilliness or a rigor, elevation of temperature, nausea, headache, and general malaise. The local inflammation may commence in some wound or abrasion, but it may also arise spontaneously. From the beginning there is marked œdema of the subcutaneous tissue. The skin is reddened, but the margin of the redness is not sharply

defined, and swelling and tenderness of the lymphatic glands are often absent. As the area of inflammation extends, the affected part becomes tense and brawny, and vesicles or large blebs form. The tension may become so great that firm pressure with the finger scarcely makes any impression. In a few days from the commencement of the disease, the greater part of a limb may be involved. If unrelieved by treatment, the tint of the redness becomes more dusky, and dark purple patches appear. At the same time the tension becomes less, and gives place to a soft, boggy feeling, indicating sloughing of the subcutaneous cellular tissue. Then livid patches appear, which break down into sloughs. As these sloughs separate, large shreddy masses of gangrenous cellular tissue can be drawn out, leaving the undermined skin connected with the deeper parts only by bands containing the larger vessels. Finally, the remaining skin being insufficiently nourished may become thin and melt away, leaving large tracts of the fascia or muscles beneath exposed to view. In this way, if proper treatment be not adopted in time, the greater part of a limb may be denuded of its cutaneous and subcutaneous covering. The extreme stage may be reached in a week or ten days, but a longer time usually elapses before all the sloughs have separated. In the earlier stages there is much burning and tensile pain, but this subsides as gangrene sets in. The constitutional symptoms are grave from the beginning. There is high fever, the thermometer often reaching 105° F. The tongue is dry and brown, and *sordes* accumulate on the lips and teeth; there is total loss of appetite; and diarrhoea is a frequent symptom. The pulse, at first full and bounding, soon loses force, becoming rapid and weak. Delirium, usually of the muttering type, is always present in severe cases. Death occurs from exhaustion, or from some complication, such as pneumonia, pleurisy, &c. During the separation of the sloughs septicæmia and pyæmia are of frequent occurrence. The disease most commonly attacks one of the limbs, but it is occasionally seen in the scrotum, and a peculiarly virulent form has been described as affecting the face. Phlegmonous erysipelas most commonly occurs in adult patients of broken constitution, suffering usually from the effects of the abuse of alcohol, or from some disease of the liver or kidneys.

**DIAGNOSIS.**—From simple erysipelas the phlegmonous form is distinguished by the great swelling and brawny hardness, by the want of a sharply defined edge, and by the early tendency to sloughing; from spreading gangrene by its slower progress, and the absence of the rapid decomposition and development of gas in the tissues. Acute necrosis somewhat resembles it, but this

disease is limited to young subjects, the swelling is less brawny, and when pus forms there is distinct fluctuation and not the boggy feeling of phlegmonous erysipelas.

**PROGNOSIS.**—The prognosis is always grave, especially if there be any delay in adopting the proper treatment. Early failure of the heart's force, excessively dry tongue, diarrhoea and vomiting are bad signs. The gravity of the case increases directly with the area affected.

**TREATMENT.**—The patient must be supported by good beef-tea, milk and eggs, and stimulants are usually required to be freely given. No depletory measures are ever justifiable. Ammonia with bark is sometimes of service. *Locally* the treatment in very mild and doubtful cases must consist in the application of hot fomentations, and extract of belladonna made into a paint with an equal amount of glycerine. As soon as there are any signs of tension, free incisions must be made to relieve it. The strictest antiseptic precautions should be adopted in making these incisions, and some efficient antiseptic dressing applied afterwards. Hot boric acid fomentations frequently changed, or sheets of salicylic wool (10 per cent.), moistened with boiling water and applied like a poultice, will be found very useful.

**III. Diffuse Cellulitis, or Cellular Erysipelas.**—In this disease the inflammation is confined to the subcutaneous cellular tissue, or to the planes of areolar tissue amongst muscles or beneath fascia. The course of the inflammation is similar in many respects to that of phlegmonous erysipelas, the only important difference being that the skin remains unaffected, or is only implicated in the later stages, as a consequence of the sloughing of the subcutaneous tissues.

**ANATOMICAL CHARACTERS.**—The *post-mortem* appearances are similar to those of phlegmonous erysipelas.

**SYMPTOMS.**—The local signs of diffuse cellulitis, when occurring in the subcutaneous tissue or beneath the superficial fascia, are marked œdematous swelling, gradually becoming brawny, slight redness of the skin, and usually mottling from over-distension of the superficial veins. There is no sharp limit to the swelling. There is intense tensile or burning pain, increased by movement, and acute tenderness on pressure. The neighbouring lymphatic glands are in most cases swollen and tender. As the disease advances, the swelling becomes doughy, and possibly an indistinct sensation of fluctuation may be felt. The skin now becomes redder, and the gangrenous inflammation may even extend to it, unless prevented by treatment. An incision in the œdematous or brawny stage merely shows the areolar spaces distended with serum, sometimes clear, more often turbid. If the incision be

delayed till the later stages, the affected cellular tissue is reduced to a mass of shreddy sloughs soaked in pus. Gas does not form amongst these sloughs till after air has been admitted from without. In mild cases the inflammation may localise itself, and lead to the formation of a large abscess. The constitutional symptoms are always grave. The temperature is high, 104° to 106° F. There are the usual symptoms of fever; the tongue is foul and speedily becomes dry; vomiting and diarrhoea are not uncommon; the pulse, at first quick and full, soon becomes feeble and rapid. There is almost always delirium. The disease usually runs a rapid course, two or three days sometimes being sufficient for it to reach its extreme stage.

Diffused cellulitis, as above described, is most frequently the result of a poisoned wound; it may then start from the wound or make its appearance at a distant part. It forms the most fatal variety of *post-mortem* wound; and, as is well known, in such cases the puncture may appear healthy, whilst the areolar tissue in the pectoral region may be the seat of most acute diffused inflammation. The bite of the less poisonous reptiles causes a similar diffuse inflammation. Diffuse cellulitis of the pelvis is a common cause of death after lithotomy, and is not uncommon in women after labour. Diffuse cellulitis beneath the pericranial aponeurosis is of frequent occurrence after scalp wounds. Occasionally the disease arises spontaneously, and it is then most common in the upper limb; but it has been seen in the areolar tissue of the neck and in many other regions. In pyæmia and septicæmia patches of diffuse cellulitis may appear in intermuscular spaces, or in the subcutaneous tissue.

**DIAGNOSIS.**—The diagnosis of cellulitis is often difficult when the mischief is deep-seated. The œdema, pain, and tenderness, with the severe constitutional symptoms, are the chief guides; but even when these are well-marked, the extent of the inflammation and the necessity for active treatment are often difficult to determine.

**PROGNOSIS.**—This depends much upon the cause, and upon the previous health of the patient. It is a very bad sign when the gravity of the general symptoms is out of proportion to the local mischief. In the pelvis cellulitis may be fatal from peritonitis; in the neck it is very fatal; it is much less dangerous in the limbs. When it occurs as a part of pyæmia or septicæmia the prognosis is of course very grave.

**TREATMENT.**—Early incisions into the inflamed cellular tissue, with antiseptic applications and abundant support, form the only reliable treatment in severe cases. In slight cases the application of extract of belladonna and glycerine (equal parts), with hot fomentations, may lead to resolution or limitation of the inflammation.

**IV. Erysipelatous Lymphangitis.**—Inflammation of the superficial lymphatic vessels is a common accompaniment of all varieties of erysipelas; but in some cases it forms by far the most prominent local morbid condition.

**SYMPTOMS.**—This affection is characterised by red lines running in the course of the lymphatic vessels from some local sore or wound. The lines are at first tolerably sharply defined, and about a quarter of an inch in width, but after a short time they spread out and several may coalesce, forming a patch exactly resembling simple cutaneous erysipelas. There is slight œdema, some pain and stiffness, and acute tenderness on pressure. The lymphatic glands to which the vessels lead are swollen and tender. The constitutional symptoms are the same as in simple erysipelas.

**DIAGNOSIS.**—This affection can only be mistaken for phlebitis; but the diagnosis is easily made by observing the course of the lines, and by the absence of the knotted cord formed by the coagulation of the blood in an inflamed vein.

**TREATMENT.**—The treatment is the same as in simple erysipelas. The extract of belladonna and glycerine is especially useful in this form of erysipelatous inflammation.

**V. Erysipelatous Phlebitis.**—Inflammation of the superficial veins, rapidly spreading in the course of the circulation, accompanied by thrombosis, redness of the skin, and acute tenderness, has been supposed by some authors to be erysipelatous in character. The only evidence in favour of this view is that the invasion and the constitutional symptoms resemble those of erysipelas, and that the affection is not uncommon during epidemics of erysipelas. See VEINS, Diseases of.

Various other diseases have been classed as erysipelatous; the chief of these are whitlow, some forms of puerperal fever, and diffuse peritonitis after operations affecting the peritoneum; all these will be described elsewhere. Diffuse inflammation not infrequently occurs after punctures made to allow of the escape of the fluid in the dropsy of cardiac or of renal disease. That punctures made into feebly nourished tissues bathed in decomposable serous fluid should set up diffuse inflammation is not surprising, but evidence is wanting to prove that such inflammation is necessarily connected with erysipelas. MARCUS BECK.

**ERYTHEMA** (*έρυθρός*, red).—**SYNON.**: Rose-rash; Fr. *Erythème*; Ger. *Hautröthe*.

**DEFINITION.**—A non-infective superficial inflammation of the skin, the essential characteristic of which is redness, that disappears on pressure by the finger, reappearing when the pressure is removed. The hue may

vary from a bright rose to a dark blue red; it may or may not be accompanied by swelling; the part may be hotter than natural; and the appearance may present itself as spots, circumscribed or diffuse, or as wheals. It is sometimes attended by a sensation of slight burning or itching, but generally gives rise to no subjective symptoms. After it has disappeared the skin is either normal, or remains slightly pigmented, or desquamates. Generally there is an increase of temperature, with slight feverish symptoms. Erythema may be either *symptomatic* or *idiopathic*.

**Symptomatic Erythema.**—**ÆTIOLOGY AND VARIETIES.**—Erythema occurs in rare instances after the administration of drugs. Cases are recorded after the ingestion of arsenic, belladonna, chloral hydrate, copaiba, cubeb, digitalis, iodides, opium, quinine, salicylic acid, stramonium, strychnine, phenazone, and turpentine. The rash usually appears immediately after the absorption of the medicine into the circulation; after arsenic it appears at a later period. See **DRUG ERUPTIONS**.

Exposure to heat or cold, and contact with various acrid or poisonous substances, are also common causes of erythema. Friction and (in the absence of cleanliness) the secretions of the skin itself, may give rise to it, as when *erythema intertrigo* is produced between the scrotum and thighs by the irritation of profuse sweat and sebaceous secretions. The blush of shame or of anger is an erythema produced by the immediate action of the vaso-motor nervous system.

Variola, cholera, enteric fever, typhus, septicæmia, rheumatic fever, and various other less distinctly defined febrile conditions, are frequently accompanied during various stages of their course by a more or less generally diffused and mostly ephemeral form of erythema.

The *roseola infantilis* of authors is an erythema that accompanies intestinal disturbance, teething, and various other disordered conditions of the system in children. Its appearance may exactly simulate that of measles or scarlatina; but it differs from these in disappearing in less than twenty-four hours, and in leaving no desquamation behind it.

The erythema that accompanies smallpox—*roseola variolosa*—appears generally on the second day of the disease, either as a diffuse redness of the whole integument, or as bright red spots, which are seen first on the face and then on other parts of the body. It lasts from twelve to thirty-six hours, and disappears when the smallpox eruption begins to show itself. A special limited form of erythema has been observed on the second and third days of smallpox, extending from the hypogastrium down the front of the upper two-thirds of the thighs; the affected surface,

when the legs are closed, having the form of a triangle the base of which is across the lower part of the abdomen. This surface remains almost or entirely free from the various pustules, and many of the cases in which it is present end fatally.

From the third to the eighteenth day after vaccination, small or large erythematous patches—*roseola vaccina*—are sometimes seen, generally on the arms, but also on other parts of the body. They usually disappear within twenty-four hours, and leave neither desquamation nor pigmentation.

The forms of erythema mentioned above cannot be considered as being in themselves specific varieties of disease, and pathologically consist in a temporary injection of the capillary blood-vessels of the skin. They are to be distinguished from the erythemata that run a distinct course, terminating in pigmentation and desquamation, and in which the capillary injection is accompanied by exudation.

**Idiopathic Erythema.**—**I. Erythema multiforme.**—This form of idiopathic erythema is most commonly seen in spring and autumn, and is distinguished by its localisation. It begins on the backs of the hands and feet, and frequently is found in these situations only. In some cases it extends upwards to the shoulders and hips, and in very rare cases is also found on the trunk.

The appearance consists in flattened papules, from the size of a pea to that of a bean, of a dark blue or brown-red colour. They are surrounded on their first appearance by a red zone, which soon disappears, and the border of the papule then stands out in fuller relief. The mildest form of this disease consists in papules which disappear after a few days—*erythema papulatum vel tuberculatum*. Instead of thus disappearing it may spread outwards from the edge, and flatten and become pale in the centre, thus forming a red ring, the condition being known as *erythema annulare*. While the first circle persists, a second ring may form around it, and the circles may be constituted by small papules, forming the condition recognised as *erythema iris vel mamellatum*. Another stage may be reached by the enlarging circles meeting, and so forming segments of a circle, constituting the form known as *erythema gyratum vel marginatum*. In consequence of increased exudation erythema papulatum may assume the aspect of a wheal, forming *erythema urticatum* or *lichen urticatus*. This condition is attended with considerable itching; and, in consequence of scratching, the centre of the papular wheal is often covered by a minute bloody crust. The exudation may be sufficient to give the eruption the aspect of vesicles or bullæ, the so-called *erythema vesiculosum* and *erythema bullosum*. Concentric circles of vesicles produce the appearance called *herpes iris*.

At any of these stages the eruption may disappear. The sequelæ are slight pigmentation and desquamation.

The disease, whose different stages have received the several names above indicated, has been designated, on account of the various forms under which it is seen, *erythema exsudativum multifforme*. It is accompanied by a slight feeling of burning, or by very slight itching. In the majority of cases constitutional symptoms are either absent, or are so insignificant as not to excite attention. There are, however, cases in which the symptoms are as marked as those which precede and accompany an eruptive fever. The prodromal symptoms are usually pain in the back, head, limbs, and joints, with gastric disturbance, and sometimes a congested condition of the throat and fauces. In severe cases the prodromal period has been observed during four to six days; and amongst other symptoms sneezing, bleeding at the nose, laryngitis, bronchitis, intense sweating, mental disturbance, and fever have been observed. All these symptoms may become aggravated during the period of eruption. In well-marked cases the disease may last from a fortnight to a month, but in exceptional circumstances the symptoms may be prolonged by fresh outbreaks of eruption. Simultaneously with the eruption on the skin, red spots may appear on the mouth and pharynx, from which the epithelium quickly disappears, leaving the parts raw and painful. Cases are recorded in which symptoms of severe intestinal disturbance, shivering, high fever, intense inflammation and ulceration of the mucous membrane of the throat, severe mental depression, and a painful condition of the joints, have been present.

In some cases the erythema is punctiform, and so much resembles the appearance of scarlatina that authors have described an *erythema scarlatiniforme*. It is distinguished from scarlatina by the absence or very slight degree of fever. The fauces, although red, are not swollen. There is no characteristic appearance on the tongue; and the pulse is little affected. There are no sequelæ.

Hebra relates that in a woman who died whilst an eruption of *erythema gyratum* was on the skin, similar red rings were found in the small intestine. It is most common in adolescence, and is more frequent in males than in females.

II. *Erythema nodosum*.—This name is given to a disease characterised by the appearance, chiefly on the surface of the lower extremities, of pale red hemispherical or oval swellings. These vary in size from that of a pea to that of a hen's egg, and are painful on pressure. Fever is sometimes present.

The swellings are at first pale red with a

yellowish tinge, later dark red, and finally livid; after they disappear they leave behind them a yellow pigmentation similar to that which follows a contusion. The number of swellings may vary from a very few on the lower extremities to successive crops on different parts of the limbs and trunk. In the latter case the feverish symptoms are well-marked. The course of the disease is completed in from two to four weeks. The swellings never suppurate, never itch, are always painful, and the redness never spreads to the adjoining skin. This variety can occur in combination with the previously described forms of *erythema multifforme*.

*Erythema nodosum* is found to be so frequently associated with definite rheumatic symptoms, such as arthritis, sour sweats, sore-throat, endocarditis, and even myocarditis, that it may be considered very probable that the affection is allied with the rheumatic diathesis.

A case has been reported in which phlebitis leading to embolism has complicated the disease.

ÆTIOLOGY.—The cause of erythema nodosum is not definitely known, although, as we have remarked, it is frequently associated with rheumatism. Some authors favour the view that it may be considered as a typical angioneurosis, whilst others believe that it is always an infective constitutional disease. It may occur at every age, but generally from eighteen to thirty, and most frequently in women. It occurs at all seasons, and debility predisposes to attacks.

ANATOMICAL CHARACTERS.—The microscopical appearances are those of an inflammatory hyperæmia. Leucocytes are found around the blood-vessels, and there are extravasations of red corpuscles. There is also an effusion of serum between the cells of the epidermis.

PROGNOSIS.—The prognosis of the special forms of erythema—*multifforme* and *nodosum*—is usually favourable, but cases are reported in which death has been caused by visceral complications. It is assumed that in these exceptional cases both the skin-eruption and the visceral lesions are produced by some infective poison. Those varieties seen in the course of other diseases do not as a rule modify the prognosis of the particular disease which each accompanies.

TREATMENT.—The treatment of erythema consists in palliating the attendant symptoms. Dusting with flour, or the application of spirit-lotion, should be employed when productive of a sense of comfort to the patient. In *erythema nodosum* warm applications of infusion of poppies or chamomile are soothing; while aperients and, when fever is present, gentle diaphoretics may be given internally. In many such cases tonics, especially quinine, are required.

GEORGE THIN.

**ERYTHRASMA** (έρυθρός, red).—This is a chronic, very slowly developing, contagious, epiphytic disease of the skin, characterised by a very slight erythematous inflammation, which usually occupies the cruro-serotal or axillary regions, but is capable of wider generalisation; produced by the presence in the corneous layer of a remarkably minute fungus—the *microsporon minutissimum* (Bueckhardt and Von Bärensprung).

**DESCRIPTION.**—Erythrasma is little known, and generally confounded with eczema marginatum. Fairly common in men, although it occasions little discomfort, it is less frequent in women, in whom it may be found about the axillary or perigenital regions.

The patches are rounded, not marginate or sensibly raised, from  $\frac{3}{4}$  inch to  $1\frac{1}{2}$  inches or upwards in diameter, uniformly roughened, and coloured yellowish-red or brownish. The pruritus and inflammation are very slight.

There are abundant delicate mycelia and conidia present, forming a rich network round the cells. The fungus requires staining and high powers for its demonstration. It is said to occur normally on parts of the skin, but erythrasma has been inoculated by Köbner.

**TREATMENT.**—All parasiticide agents, such as iodine, resorcin, salicylic acid, and chrysarobin, capable of causing exfoliation, succeed in curing erythrasma. Relapses are apt to occur unless treatment is long-continued.

T. COLCOTT FOX.

**ESCHAROTICS** (ἐσχάρια, a slough).—**SYNON.**: Fr. *Escharotiques*; Ger. *Aetzmittel*.

**DEFINITION.**—Escharotics are substances that completely destroy the tissues to which they are applied, and produce a slough. They are distinguished from other caustics simply by the greater intensity of their action.

**ENUMERATION.**—The chief escharotics are: Red-hot metal, Sulphuric Acid, Nitric Acid, Caustic Potash, Chloride of Antimony, Chloride of Zinc, Acid Nitrate of Mercury, Bromine, Chromic Acid, and Lime. Weaker caustics are—Nitrate of Silver, Sulphate of Copper, Sulphate of Zinc, Iodine, Carbolic Acid, Arsenious Acid, Sulphide of Arsenic, and Dried Alum.

**ACTION.**—Escharotics combine with the tissues and destroy them. Around the part thus killed inflammation is set up, and the part is separated as a slough. Besides their local action, these agents act reflexly on other parts of the body through the nerves of the region to which they are applied.

**USES.**—Escharotics are employed, first, to destroy the virus in, and the tissues around, a poisoned wound, and thus prevent the absorption of the poison—for example, in bites by snakes or rabid animals, or in cases of inoculation with syphilis, or with animal-

poison in dissection or *post-mortem* wounds. Secondly, they are used to destroy unhealthy tissue, such as exuberant granulations, and to remove excrescences and morbid growths, as warts, condyloinata, nævi, polypi, hæmorrhoids, and cancer. Thirdly, they are used to open abscesses, especially those of the liver. For this purpose caustic potash is usually employed. Lastly, by means of escharotics it is usual to establish issues, and thus react beneficially on distant organs.

T. LAUDER BRUNTON.

**ESSENTIAL PARALYSIS.**—A synonym for infantile paralysis. See PARALYSIS, INFANTILE.

**ESTCOURT**, in Natal.—See AFRICA, SOUTH.

**ETHER**, Uses of.—See ANÆSTHETICS.

**ETIOLOGY.**—See DISEASE, Causes of.

**EUSTACHIAN TUBE**, Diseases of. See EAR, Diseases of.

**EUTHANASIA** (εὖ, well; θάνατος, death).

**DEFINITION.**—Mental and physical distress too often attends the approach of death; and by the term 'euthanasia' we express the measures by which we alleviate or seek to remove this distress.

**IMPORTANCE OF EUTHANASIA.**—To procure euthanasia is of great moment to the patient, to the patient's friends, and to the practitioner.

It is the duty, and should be the constant aim, of every thoughtful practitioner to acquire the art of successfully smoothing the death-bed of his patient. He will thereby be enabled, when this is necessary, to relieve him of much of the attendant suffering, and to prevent many troubles individually of smaller moment, but collectively the source of much discomfort or distress. The friends' anxiety will be lightened by the relief afforded to the patient. Their own efforts in this direction are frequently unsuccessful; indeed may sometimes be prejudicial. The comfort of the dying is very often interfered with, and actual suffering induced, by the interference of well-meaning but inexperienced friends or relatives. Indeed they will sometimes question the action of the practitioner respecting the employment of a measure of which he may wish to avail himself, or they will administer food and stimulants to the patient in quantities which may be excessive or deficient or given at improper intervals. Death may thus be very often accelerated, and not without discomfort. To the practitioner himself the subject of euthanasia is one of great importance. He is involved in a position of grave responsibility, demanding the display of much tact and judgment, and one by which his professional

character will certainly, in part, be estimated. Much of his success will depend on his natural possession of these qualities, and to lay down any strict or rigid rules for his guidance is obviously impossible.

**THE MORBID STATES CALLING FOR EUTHANASIA.**—1. *Mental Condition.*—The question of dealing with the mental distress of the dying is one which only secondarily concerns the practitioner, and will therefore be noticed but briefly here. The most important point to be decided is: Should the practitioner be the medium of communicating to his patient the hopelessness of his case? Whenever possible, this painful duty should be relegated to the friends of the patient, who are always to be informed of the approach of death, in order that due attention may be paid to the affairs of the sick person, both spiritual and worldly. The practitioner should not hastily take on himself to tell him of the inevitable result of his illness. The patient may or may not desire to know the truth, and when it is communicated to him it is certain to affect his peace of mind in the one or in the other direction. Some welcome the announcement; whilst others are filled with dread. But it is a fact that the majority of persons seriously ill feel less on such matters than they would have felt in health: they 'mate and master,' as it were, 'the fear of death.' With many patients, not to know the truth respecting the danger which they are in, is itself a form of unhappiness which militates against the establishment of euthanasia. The practitioner should therefore most carefully study the individual case and the character of his patient. On the one hand he will exercise the greatest care not to falsely conceal their situation from the dying; on the other hand, he must keep in mind that a fatal termination is not always *certain*, and that in many cases the last chance of an over-sensitive patient may be destroyed by the prospect of problematical dangers (*see* DISEASE, Prognosis of). Good sense, kind feeling, tact, and experience, will prove of the greatest value in discharging this delicate duty as it comes before us in a general practice. *See* DEATH, Modes of.

2. *Bodily Conditions.*—The physical states which specially call for euthanasia are—pain; dyspnoea, cardiac distress, exhausting cough; laryngeal distress; difficult expectoration; restlessness and sleeplessness; thirst; hic-cough; abdominal distension; dropsy; bed-sores; and exhausting discharges. These, and a few of the general and therapeutic measures at our command which ought ever to be present to the mind of the practitioner for abolishing or mitigating sufferings incident to the dying state, will now be briefly touched upon.

The removal of *pain* is essential for the promotion of euthanasia, and no other remedy

is so successful for this purpose as opium or morphine cautiously administered. It is an adjunct of the greatest value to other measures, and, moreover, successfully combats the exhaustion and sinking sensations which, in certain cases, are a source of indescribable distress. Its anodyne effect is enhanced by its cardiac action. Its administration, however, often leads us into much fresh difficulty, by causing a dry tongue and distressing thirst; by arresting the excretions; in other instances by increasing the reluctance to take sustenance; or the presence of kidney-disease may prove a serious contra-indication to its employment.

The question as to the patient remaining out of bed pillowed up in a suitable chair will sometimes arise when *dyspnoea* and *flatulence* are urgent; and will demand much judgment and care. By rendering moist the air of the bedroom with the vapour of hot water in a bronchitis-kettle placed upon a fire (not over gas or spirit-lamp contrivances, which only tend to vitiate the atmosphere of the sick-chamber), we can often insure the relief of pulmonary distress. Uramic dyspnoea will call for the administration of nitroglycerine, nitrite of amyl, or sulphuric ether.

Painful *cardiac* palpitation, and its attendant distress, often derive relief from the administration of antispasmodics along with iodide of potassium, a belladonna plaster at the same time being applied over the præcordia. Digitalis, strophanthus, strychnine, or some of the other well-known excellent cardiac tonics, may also be resorted to with success.

One of the most distressing symptoms in the dying is the exhausting *cough* of phthisis, which greatly exhausts the patient. It may be combated by judicious nursing; by an acid linctus, with or without the addition of a little morphine; by a combination of respiratory sedatives; by the frequent administration of small quantities of alcoholic stimulants; or by hot or cold liquid nourishment. The *laryngeal distress* which frequently attends the final stage of phthisis will best be relieved by morphine insufflations, cocaine sprays, or recourse may be had to rectal alimentation. The loud, noisy, gurgling râles met with in the last phases of bronchitis and other conditions will call for change of posture and stimulants, although they are more distressing to the friends than a source of danger to the patient (*see* STERTOR). When the attempts at *expectoration* are difficult, measures must be employed which possess the power of diminishing the viscosity of the expectoration, or of stimulating the respiratory centre and the respiratory movements. *See* EXPECTORANTS.

The *restlessness* and tossing of the limbs, irregular sighing, with coincident *sleeplessness*, which so often attend the last days

of fatal illnesses, may sometimes be relieved by attention to the weight of the bedclothes, which can be conveniently reduced by employing a suitable cradle. The arrangement of the pillows and attention to the posture of the dying patient constantly tax the ingenuity and resource of the attendants. The temperature and ventilation of the sick-room are also of great importance in this respect, closeness and over-heating being zealously prevented, as well as crowding of the sick-chamber with anxious friends. The feet and legs ought to be frequently examined, and if chilled they should be warmed by a carefully applied foot-warmer. The unpleasant effects of cold sweats will also call for attention. The catheter will remove another often overlooked cause of inquietude—a distended bladder. By these and similar measures we may be able to counteract the cause or causes of insomnia, and thus secure for our patient a few precious hours of sleep.

*Thirst* is often a most distressing symptom. It may best be relieved with ice, or with teaspoonfuls of iced water or acidulated, unsweetened, cold black tea or coffee. But in this connexion let it be understood that we must be careful to avoid the popular mistake of forcing down liquids in cases where life is evidently fast ebbing away. Such a practice necessarily embarrasses respiration, and we have likewise to remember that at this time the act of deglutition very often is in abeyance. Death is occasionally accelerated by injudicious attempts to raise the patient, open his mouth, and administer food.

In the management of the dying we have always to contend with an enfeebled digestion. The *diet* in such cases should be one easy of assimilation. It not infrequently happens that the food given may be excessive or deficient in quantity, or that it is administered at irregular intervals, thereby causing much distress to the patient. Success in this direction depends solely on the tact of the nurse. Whilst in acute disease nothing conduces more to success than definite instructions as to the amount and kind of the food to be given, and the hours for its administration, it is impossible and inadvisable to observe these rigid rules in the feeding of the dying.

In conjunction with nourishment we can often remove much of the suffering by the judicious selection and administration of *alcoholic stimulants*. If the patient tires of one form of stimulant, another should be tried, provided we are careful to use only such as are easily digested and assimilated, and given in divided quantities. A combination of beef-tea, liquor strychninæ hydrochloratis, and brandy is often very acceptable, especially in bad cardiac cases. Another stimulant of extreme service is ether, which

particularly alleviates painful attacks of spasmodic breathing.

A sinapism to the epigastrium in conjunction with the internal use of a little ether and brandy, will frequently relieve that sometimes distressing symptom—hiccough.

Distressing *abdominal distension* must be relieved by the employment of turpentine stupes and by enemata containing some antispasmodic. *See* ENEMA.

The treatment of dropsy, abdominal distension, bed-sores, and exhausting discharges is dealt with in other parts of this work.

In addition to the conditions which call for euthanasia, it is clear that the severe dietetic restrictions which may have been enforced in a case of Bright's disease, diabetes, &c., ought to be reconsidered and removed when it becomes apparent that the patient's condition is absolutely hopeless. The wishes and suggestions of the patient may generally be acquiesced in, and they will prove a useful guide to the practitioner as to the line of action to pursue. It is often the wisest course to humour cravings. Seeming extravagances as to diet, if persistently asked for, had better be granted, especially when kind and gentle persuasion has failed to get the patient to waive his desire for them.

In addition to the means already mentioned, it need hardly be said that we still have all those measures which may be summed up in the expression 'perfect nursing,' which implies carrying out many details as well as exercising sympathy and tact—a nurse not overdrilled. A mere automaton thinks too much of her 'uniform' and of her 'duty.'

JOHN HAROLD.

**EVACUANTS** (*evacuo*, I empty).—

SYNON.: Fr. *Evacuants*; Ger. *Ausleerende Mittel*.

DEFINITION.—Medicines used to produce some evacuation from the body.

ENUMERATION.—The chief evacuants are: Sternutatories, Expectorants, Sialagogues, Emetics, Cholagogues, Purgatives, Diaphoretics, and Diuretics. *See* the several articles upon these subjects.

**EVIAN-LES-BAINS**, in Savoy, France.—Alkaline waters. *See* MINERAL WATERS.

**EXACERBATION** (*exacerbo*, I aggravate).—Increase in the severity of the symptoms of a disease. *See* DISEASE, Duration of.

**EXANTHEMA**; **EXANTHEMATA** (*ἐξ*, out; and *ἀνθῆω*, I blossom).  
SYNON.: Fr. *Exanthème*; Ger. *Ausschlag*.

DEFINITION.—A rash or eruption on the skin attending a specific fever. The specific fevers attended by rash. This term, once denoting any cutaneous eruption, is now restricted to the eruptive fevers called the *exanthemata*. The febrile rashes or exanthes

of local or individual origin — urticaria, erythema, and roseola—are not included with the true exanthemata, which are acute specific infectious diseases, namely, Typhus, Variola, Varicella, Morbilli, Rubella, Dengue, Scarlet Fever, Typhoid or Enteric Fever, and perhaps Erysipelas. As the leading features of these diseases will be discussed under their several heads, it is only necessary further to notice certain less defined and regular eruptions associated with fever.

All the exanthemata are attended with fever and enlargement of the lymphatic glands. Convulsions in children may occur with the first fever; in some of the diseases before the rash appears.

*Typhus*.—The mulberry rash appears suddenly on the fourth and fifth days of illness as a dull red mottling of irregular, persistent, non-elevated spots; the fever is high at the commencement, and continues to be so after the rash is fully developed.

*Typhoid*.—In typhoid or enteric fever the small, raised rose-spots do not appear till the second week of fever; sparsely scattered on the trunk, they fade on pressure, disappearing in three or four days, while new spots arise.

*Smallpox*.—Marked fever of sudden ingress occurs two days before the raised eruption. Sometimes a rose-rash first appears, but the severe symptoms begin a full day before this, and not more than two days before the characteristic spots. The cervical glands are enlarged.

*Varicella*.—The eruption begins on the first day of illness, with slight fever, increasing as fresh spots appear. The fever may be high and come on suddenly, but enlarged cervical glands and spots somewhere are always to be found at the same time.

*Measles*.—Three days of fever and catarrh, with palpable enlargement of the cervical glands, precede the rash; there is then sudden increase of fever, subsiding while the rash is at its height.

*Rubella; Rubeola sine catarrho*.—The rash appears within a few hours of the first feeling of illness, which is slight and soon over. The rash is at first spotted rather than finely diffused. By the time it is fully out the fever has subsided, but the enlarged cervical glands which marked the ingress always remain to indicate a specific disease. Fine desquamation rarely follows, and there is no albuminuria. Rubella spreads by direct infection, the incubation being from two to three weeks. This long period of incubation causes the source of infection to be often overlooked, and even the possibility of it to be denied. When carried to a family or school either an unnecessary alarm of measles is raised; or contagion is denied, and the next sufferers are said to have a rose-rash from heat, or from irritation of the stomach.

*Dengue*.—Widely spread in Africa, the warmer parts of America, and both the Indies,

dengue may possibly be limited to hot climates; its presence with us is as yet undetermined. The rash is at first discrete, like that of measles, but follows soon after infection; and the disease in its general course is allied to influenza.

*Scarlet Fever*.—The finely diffused redness is found on the skin and in the mouth and throat often within a few hours of the sudden ingress of fever. The fever increases with the development of the rash, both persisting for several days. Often the throat is first complained of; the glands at the angle of the jaw are full and tender; the tongue presents prominent red papillæ projecting through the white fur. The skin is not swollen as in erysipelas, nor the redness so circumscribed; the throat also is redder; the cervical glands are enlarged in both. Surgical scarlatina is often declared three or four days after operations; the rash of septicæmia at a later period.

*Epidemic Roseola*.—This is a name for röteln; also for a rash having such relation to scarlet fever as rubella to measles, but with slender claim to autonomy. Mild cases of scarlet fever often begin with a finely diffused redness shortly after some feeling of faintness or giddiness, and an incubation of from three days to a week; sometimes albuminuria occurs as an early symptom. The finely diffused rash, enlarged cervical glands, and slight sore-throat, even with very little elevation of temperature, raise the suspicion of scarlet fever; should albuminuria follow, or any shredly desquamation of the hands and feet, no uncertainty remains. Scarlet fever so modified often spreads and gives rise to the severer forms of the disease, when it has been called only rose-rash or roseola; the use of these terms without a distinctive qualification always leaves a doubt as to the completeness and safety of the diagnosis.

*Erythema*.—Erythema comes nearest to these cases in appearance—so near as often to be spoken of as recurrent scarlet fever or erysipelas, but there is no enlargement of the cervical glands in erythema, and so little fever that the temperature of the reddened skin is barely elevated above the normal. Since Fuller's *Exanthematologia* this kind of flush passes under different names of roseola, according to the variations in shape of the red patches, or the seasons of the year at which they occur.

*Erythema nodosum*.—This disease is often preceded by slight fever for a day or two, which may reach 102° F., but subsides as the red swellings appear. Locally there is little or no elevation of temperature, even when the tender part feels hot.

*Exanthematous Roseola*.—This affection, occurring in the course of other specific diseases, is distinguished from the roseola which depends on nerve-irritation, such as that from acrid ingesta, by the presence either of high

fever, or of glandular enlargement, or of both, as when it precedes the true variolous eruption. During enteric fever this form of roseola may occur quite independently of the special lenticular rose-spots.

An eruption of this kind is not infrequent in the early stages of diphtheria, sometimes as a diffused rash limited to certain parts of the chest and body, or as discrete spots on the limbs and back of the hands and feet. Influenza, and some forms of catarrh, winter 'colds,' or summer diarrhoea as noticed by Bateman, may present various kinds of roseola on the back, shoulders, and chest; the cervical glands are perceptibly enlarged, though there may be little fever. In these cases it is not the roseola, but the specific disease on which it depends, that might, without precautions, be communicated to others.

*Syphilitic Roseola.*—A special roseola marks the secondary stage of syphilis; in appearance it resembles the rash of measles. So does the *roseola ab ingestis* when produced by cubebs, but this has neither fever nor glandular enlargement.

The absence of fever from the roseola after vaccination refers this form of eruption, like that occurring from dentition, to the class of rashes from nerve-irritation. *Vaccinia* is itself an exanthem in the wider definition of the term, reproduced after a definite period of incubation by inoculating a special contagium. Wanting this character, the different forms of herpes are excluded, unless resulting from a general febrile disturbance; though inoculable, ecthyma and impetigo are local affections not belonging to the exanthemata.

WILLIAM SQUIRE.

**EXCITANTS** (*excito*, I excite).—It seems hardly necessary to give any special consideration to this therapeutical class, as all that may be said on the subject ranges itself with greater propriety under the heading **STIMULANTS**. Stimulation is, in fact, a degree of excitement; and it is only when its effects are more vigorously pushed, that we obtain that inebriation or exhilaration which is so commonly observed to follow the use of alcohol, ether, and the anæsthetic vapours. See **STIMULANTS**.

ROBERT FARQUHARSON.

**EXCITING CAUSES.**—See **DISEASE**, Causes of.

**EXCITO-MOTOR DISORDERS.**—See **REFLEX DISORDERS**.

**EXCORIATION** (*ex*, from; and *corium*, the skin).—The superficial destruction of a portion of the skin or mucous membrane.

**EXERCISE.**—**DEFINITION.**—In its widest and most correct signification, exercise is the setting in motion any active body; and when the term is used in a physiological connection, it may refer to the functional

activity of any of the organs, whether muscular, nervous, nutritive, secretory, or reproductive. In this very comprehensive sense, the subject of exercise includes a large portion both of hygiene and of therapeutics. The popular signification of exercise is, however, much more limited than the preceding, having reference only to the muscles directly, and to the parts called into play through the same—especially the circulatory and respiratory systems.

Whether in its wider or in its narrower sense, exercise has several important relations to Medicine. 1. It is essential to the preservation of health (see **PERSONAL HEALTH**). 2. It has to be regarded as frequently associated with the causation of disease (see **DISEASE**, Causes of). 3. Exercise is a most rational and successful means of treatment in certain disorders and diseases (see **MOVEMENT**, Therapeutical Uses of). 4. Exercise is often abused; and excessive indulgence in some forms of it gives rise to serious consequences. The present article will be devoted to the consideration of exercise in the last-named aspect only; and the subject will be discussed according to the more limited and popular definition of the term.

**Abuse of Exercise.**—From the moment an infant is born until the end of life, exercise, duly apportioned to rest, is the normal state of existence; and whilst continued overstrain of any portion of the human machine is the forerunner of disease, so, on the other hand, is equally, if not more so, that want of exercise which induces wasting and degeneration.

**PRINCIPLES.**—The late Dr. Parkes, in his *Practical Hygiene*, has given a very complete statement of the results of the investigations of himself and others on the changes effected by the stimulus of muscular exercise on the various organs and tissues of the body, from which he has drawn the following conclusions:—

'The main effect of exercise is to increase the oxidation of carbon, perhaps also of hydrogen. It also eliminates water from the body, and this action continues—as seen from Pettenkofer and Voit's experiments—for some time; after exercise the body is therefore poorer in water, especially of the blood; it increases the rapidity of circulation everywhere, as well as the pressure on the vessels, and therefore it causes in all organs a more rapid outflow of plasma and a more active absorption—in other words, a quicker renewal.

'In this way, also, it removes the product of their action which accumulates in organs; and restores the power of action to the various parts of the body. It increases the outflow of warmth from the body by increasing perspiration. It therefore strengthens all parts. It must be combined with increased supply both of nitrogen and carbon (the

latter possibly in the form of fat), otherwise the absorption of oxygen, the molecular changes in the nitrogenous tissues, and the elimination of carbon, will be checked. There must also be an increased supply of salts, certainly of chloride of sodium, probably of potassium phosphate and chloride. There must be proper intervals of rest, or the store of oxygen, and of the material in the muscles which is to be metamorphosed during contraction, cannot take place. The integrity and perfect freedom of action both of the heart and lungs is essential, otherwise neither absorption of oxygen, nor elimination of carbon, can go on, nor can the necessary increased supply of blood be conveyed to the acting muscles without injury.'

The proper amount of exercise requisite for health is difficult to determine, in consequence of the varied constitutions of individuals. It may, however, be accepted that whilst in youth the great spirit of emulation tends to an overstrain of mind or body, so, as life advances, one or other or both are liable to be allowed to pass into a state of unhealthy inactivity.

Since the more general practice of gymnastics in this country, and the stimulus that has been given to aquatic exercises by our University competitions, great attention has been drawn to the effect of bodily exercise on health, and more especially with regard to the heart and lungs, these being the organs upon which its influence is most immediately exerted.

*Prolonged and Excessive Exercise.*—Of all exercises, rowing is the one which is generally accepted as the best variety to select, if we are to endeavour by a consideration of its influence upon those who practise it to form an estimate of the effect of a continuous strain on the circulation and respiration; yet the difficulty of procuring trustworthy evidence on such a subject is extreme. Dr. Morgan, in his *University Oars*, by collecting the various experiences of nearly all the men who rowed in the University races from 1829 to 1869, has obtained about the most accurate testimony available in regard to one aspect of the subject. These men are unanimous in their belief that they experienced no injury from the great strain they underwent in their youth. But it must be borne in mind that they were the picked athletes of their colleges, men with large frames and full chests, typical specimens of health, capable of undergoing very prolonged exertion with but passing fatigue, and to whom no permanent injury could be anticipated, after careful training, from an exceptional display of strength. Such evidence affords no clue to the effect of the strain imposed on the heart by the two hundred or more of each University, who annually use the utmost exertion to belong to the chosen few, and many of whom, unguided in their

violent efforts to achieve success, have in after-life to pay the penalty of allowing mere feeling or the spirit of emulation to overrule their reason.

*Exercise under Unnatural Conditions.*—But it is not only the case that exercise which is excessive or too prolonged proves highly deleterious; even a moderate amount of exercise under unnatural conditions may prove equally harmful. Thus the young soldier of light frame, with irritable palpitating heart, who has broken down in his preliminary training, is a marked and good example of the early injurious effect of overstrain of the heart, under the impediments caused by tight clothing and accoutrements to the free expansion of his chest. When at rest he feels perfectly well, and has little or no sensation of throbbing in his chest. So soon, however, as he puts on his tunic and accoutrements, and begins his drill, throbbing occurs with more or less violence, accompanied with a feeling of oppression, and with difficulty of breathing, and this being followed by a sensation of faintness, sickness, or dizziness, he has to fall out of the ranks. At first the condition of the heart is one purely of functional disturbance, which, though rendering him unfit for the duties of a soldier, does not interfere with his gaining his livelihood as a civilian.

This functional derangement of the heart, which is readily shown by the diastolicism in the sphygmographic tracing of the radial pulse when auscultation can detect little or no change in the heart-sounds, is frequently found in those youths of delicate frame in our schools and colleges, who, 'breaking down' in attempting feats of strength or in the preliminary training, experience no ill-effects in the ordinary avocations of after-life from that overstrain of heart which, if neglected, would be apt to lead to graver forms of heart-disease.

Whether it be by sudden or prolonged violent exertion, by rowing, or by running, or by the many other severe exercises of the body entailed by labour or pleasure, there can be no doubt that the heart and lungs have at times an inordinate amount of strain forced upon them, which, in a state of health, or under favourable circumstances, they may reasonably be expected to bear with no more injury than temporary distress, and that this capability to bear strain is greatly enhanced by careful training.

It is customary for the healthy boy, however, owing to the character of his amusements, always to be in training, so far as his body is concerned, and with very little supervision he ought to suffer no harm from sudden and exceptional strains. But it is very different with men who have settled down into the real business of life, who, during their nominal periods of rest from their daily labours, undertake violent exercises without

any preliminary training, and thus throw such an unexpected strain on the heart and great blood-vessels, that instead of mere functional disturbance, as in early life, they sow the seeds of organic disease. Such being the case, how much more injurious must sudden overstrain be to a heart already weakened by disease? There is often found amongst men a great aversion to having their hearts examined, and when disease is discovered it is sometimes considered of questionable advantage to inform the sufferer of his condition; but this is a mistake, for from want of knowledge of his state he may, by unnecessary strain, rapidly aggravate it, and thus shorten a life which might otherwise have been much prolonged.

The purport of these observations is thus to point out that: First, whereas exercise is necessary to preserve our bodies in a proper state of healthy activity, its tendency, when carried to extremes, is to set up organic lesions. Secondly, that as in some athletic competitions a very great strain is thrown upon the thoracic organs, it is essential that no boys of delicate frame should be allowed to take part in them, or in the preliminary training, excepting under careful medical supervision. And, thirdly, that in manhood no violent competition should be undertaken, which would throw a great strain upon the thoracic organs, without their being previously examined and pronounced sound, nor until their full powers have been brought into play by careful preliminary training.

A. B. R. MYERS.

**EXFOLIATION** (*ex*, from; and *folium*, a leaf).—The separation of a portion of dead bone or cartilage from the living tissue, in the form of layers (*see* BONE, Diseases of). The term is also applied to the separation of a false membrane, which has been mistaken for the whole mucous lining of the bladder or uterus. *See* BLADDER, Diseases of.

**EXHAUSTION** (*ex*, from; and *haurio*, I draw out).

**DEFINITION.**—Exhaustion is a phenomenon which all irritable tissues can be made to manifest, and consists in a failure to respond to stimulation. Exhaustion of muscle and nerve is brought about by excessive, quickly repeated, or continuous stimulation. It is favoured by cutting off, or by an alteration in the quality of, the blood-supply; by previous insufficient exercise of function; by exposure to extremes of temperature; by an insufficient supply of oxygen; by an excessive supply of carbonic acid; and by exposure to certain toxic agents. These facts, which have been established by physiological experiments, are fully borne out by clinical experience.

Exhaustion may be *general* or *local*.

**1. General Exhaustion.**—General ex-

haustion is brought about by over-work, whether physical or mental, and especially by unremitting and monotonous duties which keep the same paths of action in a state of constant activity. It is not often, if ever, that any permanent harm is produced in a healthy man by mere physical labour, however great; but excessive mental labour, especially if it be monotonous, is certainly capable of permanently damaging the nervous tissues. When in addition to hard mental work, which is performed voluntarily, some constant stimulus, which cannot be arrested, unceasingly works upon the brain, exhaustion quickly results; as when, for example, a man who is harassed by trying to earn sufficient for his family meets with some shock to his nervous system (such as a railway accident, the sudden death of a dear relative, or a severe money loss) which haunts him like a spectre day and night, robs him of his rest, and deprives him of his appetite. General exhaustion is favoured by all conditions which give rise to anæmia or faulty nutrition, such as hæmorrhage, prolonged pyrexia, inadequate diet, persistent morbid discharges, or venereal excess; by the retention in the tissues of the products of their activity, which is favoured by working in a foul atmosphere, or by derangement of the excreting functions; by exposure to extremes of temperature; and by a previous condition of excessive slothfulness. General exhaustion may occasionally be suddenly induced by physical causes, such as a severe injury (collapse from shock), or psychological causes, such as fright.

**SYMPTOMS.**—The symptoms of general exhaustion are: 1. Loss of sleeping power, persistent dreaming, talking in the sleep, and somnambulism. The patient may wake in the morning feeling totally unrefreshed. 2. Incapacity for work, and inability to seriously apply the mind to one subject for any length of time. 3. Headache, and a feeling of oppression in the head. 4. Languor and general lassitude. 5. A rapid feeble pulse. 6. An anxious expression of face; and (as stated by Dr. George Johnson) a contracted and sluggish pupil. In addition to these we may get tremor, delirium, hypochondriasis, hysteria, epilepsy, chorea, mania, and general paralysis. Two instances have come within the writer's knowledge of transient hemiplegic symptoms having been induced by excessive application to literary work. The digestion is often deranged, and functional disturbance of the heart is common. Occasionally the urine is altered in quality, and may contain alkaline phosphates or sugar. More rarely it manifests excessive acidity.

**2. Local Exhaustion.**—Local exhaustion is the result of excessive local stimulation, and it is particularly liable to occur as a prominent symptom in patients who are suffering from general exhaustion. The loss

of power in the rectum which results from the excessive use of purgatives; the failure of the uterus in cases of protracted labour; and the failure of the voluntary muscles which occurs in those professional ailments of which 'writer's cramp' is the type, may be taken as examples of local exhaustion.

**TREATMENT.**—In the treatment of exhaustion the main indications are to lighten the labour, and obtain rest. In cases of general exhaustion it is often advisable to administer narcotics, such as opium, chloral, sulphonal, or bromide of potassium; and it will be generally found that, when once refreshing sleep has been established, the more aggravated symptoms will subside. Fresh air and a good diet are most necessary. Stimulants must be used with great caution, for it is clearly not desirable to goad the exhausted organs into further action, although it may be necessary to employ stimulants to give temporary power while the faculty of sleeping is being re-established. All causes of anæmia must be removed. When recovery is established, the patient must be encouraged to relieve the monotony of his life by some pursuit which should be, as it were, the complement of his ordinary occupation. Thus the headworker should endeavour to amuse himself in his leisure hours by gentle out-door exercise, by music or painting, or by practising some handicraft. See **DEBILITY**; and **FATIGUE**.  
G. V. POORE.

**EXOMPHALOS** (ἐξ, out; and ὀμφαλός, the navel).—A term applied to umbilical hernia. See **HERNIA**.

**EXOPHTHALMIC GOÏTRE** (ἐξ, out; ὀφθαλμός, the eye; and *guttur*, the throat).—**SYNON.**: Graves's Disease: Basedow's Disease; Fr. *Maladie de Graves*; *Goitre exophthalmique*; Ger. *Glotzaugen-kropf*; *Basedow'sche Krankheit*.

**DEFINITION.**—Enlargement with vascular turgescence of the thyroid gland, accompanied by protrusion of the eyeballs, breathlessness, palpitation, tachycardia, and anæmia.

**ÆTIOLOGY.**—This disease is comparatively rare in men. It occurs most frequently in women between the ages of twenty and thirty, but is met with amongst older persons. Patients suffering from it often belong to the so-called nervous diathesis. Its occurrence is often preceded by menstrual disturbance and anæmia. Sometimes no exciting cause can be discovered, but in many cases it comes on after violent nervous excitement, sometimes after acute disease.

**SYMPTOMS.**—Before exophthalmic goitre makes its appearance, alterations in temper are frequently observed, the patient becoming irritable and depressed. Functional disturbances of the circulation and heart occur at frequent intervals, the heart palpitating,

the face flushing, and a sensation of fullness being felt in the head, eyes, and throat. The palpitation increases, the eyes become prominent, and a visible swelling appears in connexion with the thyroid gland. The eyes are lustrous and projecting, and there is frequently a slight loss of co-ordination between their movements and those of the eyelids, so that when the eyes are quickly cast down the eyelids follow them so slowly that a white ring of sclerotic may be noticed between the iris and the lower margin of the upper eyelid. Usually there is no disturbance of vision. The exophthalmos is most marked during emotional excitement and at the menstrual period, and at these times the patient suffers from an increased feeling of fullness in the eyeballs. Sometimes the projection of the eyeballs is so great that the lids do not perfectly cover them, and inflammation and ulceration may consequently occur. The thyroid is generally unequally enlarged. Its size varies from time to time, increasing, like the protrusion of the eyeballs, with emotion. It is soft and elastic, and pulsates, so that it has sometimes been mistaken for aneurysm. The palpitation of the heart is generally noticed before either the exophthalmos or enlargement of the thyroid, and is the first symptom to attract the patient's attention. It is increased by emotion or exertion; and the violent cardiac action frequently produces a prominence of the præcordial region. The cardiac pulsations are rapid, and sometimes irregular. The cardiac sounds are loud; and a soft, systolic bellows-murmur is frequently audible at the base, and in the large arteries. The carotids are sometimes, but not always, dilated. The circulation appears to be rapid, the veins filling quickly when emptied, and pulsation being felt even in small arteries. The temperature is frequently high. There is a feeling of general debility. The digestion is sometimes normal, at other times the appetite is diminished or capricious, and diarrhoea may occur. The swelling of the neck may give rise to a feeling of difficulty of breathing; and the voice sometimes becomes altered and hoarse, or may be lost entirely. The course of the disease varies considerably: it may sometimes go on increasing for several months, then it becomes stationary for one or two years, and afterwards begins to decline. The temper improves, the appetite increases, and menstruation frequently is re-established. The palpitation, enlargement of the thyroid, and prominence of the eyes gradually diminish, although they rarely disappear completely. Death may occur from intercurrent disease, from organic cardiac lesions, from uncontrollable diarrhoea, or from gradual wasting. Danger is also said to arise from pressure on the trachea by the enlarged thyroid.

**PATHOLOGY.**—The protrusion of the eye-

balls is due either to dilatation of the vessels in the orbit, or to contraction of the involuntary muscular fibres in the orbital membrane which covers the speno-maxillary fissure, or possibly to both causes combined. The enlargement of the thyroid is due to dilatation of the vessels of the gland. After the disease has lasted some time, increased formation of tissue in the thyroid gland may occur. Palpitation of the heart is probably due to stimulation of the accelerating cardiac nerves; and this, as well as the alteration in the nerves of the orbit and thyroid, has been ascribed to disease of the lower cervical sympathetic ganglia, in which increased connective tissue and diminution of ganglionic cells have been observed.

**DIAGNOSIS.**—When the three leading symptoms are present, it is impossible to confound exophthalmic goitre with any other disease. The enlargement of the thyroid is distinguished in this disease from that of cystic goitre by its greater elasticity, by its paroxysmal enlargement, and by its pulsation. The exophthalmos is distinguished from that due to disease of the orbit or cranium by being equal in both eyes, and by the absence of squint. It is distinguished from prominence due to cardiac disease by the lustrous appearance of the eye; from hydrophthalmia by the natural condition of the pupil; and from the prominence which may occur in myopia by the vision being natural, and by the paroxysmal increase of the prominence in exophthalmic goitre.

**PROGNOSIS.**—This must be guarded, the disease not being very amenable to treatment, and very rarely disappearing altogether, although after continuing for some years it may gradually improve.

**TREATMENT.**—The treatment of exophthalmic goitre chiefly consists in securing fresh air, gentle exercise, the avoidance of the least fatigue or emotional disturbance, and careful diet. Iron is sometimes useful, the milder forms, such as the tartarated iron or citrate of iron and ammonia, being preferable to the more powerful preparations. The writer believes he has seen much improvement from saccharated solution of lime. Quinine, alone or in combination with digitalis—and perhaps with belladonna—often produces good results. Digitalis and strophanthus are occasionally, but not always, useful. Aloes and myrrh may be employed to keep the bowels open. Galvanism to the neck has sometimes been productive of benefit, one pole being placed on the nape of the neck and the other along the sides of the thyroid tumour. When the eyeballs are so prominent as to become liable to inflammation and ulceration, care must be taken, by means of a shade, to protect them from irritation; and if this should prove unavailing, the inflammation must be treated by appropriate remedies.

T. LAUDER BRUNTON.

**EXOSTOSIS** (ἐξ, out of; and ὄσσειον, a bone).—A bony outgrowth from any part of the skeleton. See BONE, Diseases of.

**EXPECTANT ATTENTION.**—An important mental state. See MESMERISM.

**EXPECTORANTS** (ex, out of; and *pectus*, the chest).—**SYNON.**: Fr. *Expectorants*; Ger. *Auswurfsmitteln*.

**DEFINITION.**—Medicines which facilitate the removal of secretions from the air-passages.

**ENUMERATION.**—The leading expectorants are:—(A) Ipecacuanha, Antimony, Apomorphine, and Iodide of Potassium; Chlorides of Potassium, Sodium, and Ammonium; and Inhalation of Steam. (B) Squill, Senega, Benzoin, Benzoic Acid, Benzoate of Ammonium; Myrrh, Storax, Balsam of Tolu, Balsam of Peru, Ammoniacum, Galbanum, Asafœtida, Anise, Fennel; Larch Bark, Tar, Terebene, Copaiba; Vapour of Chlorine, Iodine, Ammonia, Creasote, and Carbolic Acid. (C) Ammonia, Carbonate of Ammonium, Strychnine, Nux Vomica, and Belladonna.

**ACTION.**—The mode of action of expectorants is not well understood, and any explanation of it in the present state of our knowledge can only be regarded as provisional. Expectorants may be divided into two classes—(1) Those which modify the nature of the secretions from the respiratory passages; and (2) those which modify the respiratory movements by which the secretions are expelled. In considering the mode of action of the first class it must be remembered that the secretions from the respiratory passages depend, like many other secretions, on two factors, the *direct* influence of the *nerves* upon the secreting structures, and the *amount of blood* supplied to them. Each of these two factors may be influenced to a different extent by various drugs. As has already been said, the exact action of each cannot be determined at present, but the first class of expectorants may be subdivided into two divisions which are distinguished in the foregoing enumeration as A and B. The division A rather diminish than increase the activity of the circulation, and are therefore called *sedative* expectorants. The division B somewhat increase the circulation, and are called *stimulating* expectorants. Those comprised under C *stimulate the respiratory centre* in the medulla oblongata, and increase the respiratory movements.

**USES.**—*Sedative expectorants* (class A) are useful when there is congestion of the respiratory passages, with very scanty, tough expectoration, as in commencing bronchitis. In such circumstances, when dry rhonchi are heard abundantly, with few or no moist râles, the patient often coughs until quite exhausted, bringing up scarcely anything. The administration of sedative expectorants renders the

secretion from the respiratory passages more fluid, abundant, and easy to expectorate. When these expectorants do not succeed in ordinary doses, their action may be much assisted by the administration of a purgative, or, still better, by giving either ipecacuanha or tartar emetic in such a large dose as to produce sickness and vomiting. When the distress of the patient is great, the abstraction of a small quantity of blood by cupping or by venesection may give great relief. The inhalation of steam alone is also beneficial, and the air of the patient's chamber should be kept warm and moist.

*Stimulating expectorants* (class B) do more harm than good when administered in the conditions just described, but are beneficial when the acute symptoms have passed off. When this is the case, but the expectoration is tough and somewhat scanty, squill is a useful expectorant; but when the expectoration is abundant, benzoin, balsams or ammoniacum would be preferable. In chronic bronchitis, inhalations of ammonia, chlorine, iodine, creasote, carbolic acid, eucalyptus, or pine oil are useful. When the expectoration is foetid, chlorine, iodine, and carbolic acid inhalations are best.

*The expectorants which act on the respiratory movements* (class C) are useful in cases of debility, as they stimulate the respiratory nervous centre in the medulla oblongata, as well as assist the failing circulation. They may be advantageously combined with stimulating expectorants, such as squill or benzoin, according to the nature of the secretion.

T. LAUDER BRUNTON.

**EXPECTORATION** (*ex*, out of; and *pectus*, the chest).

**DEFINITION.**—This word, which strictly means the act of expelling anything from the chest, is usually applied to the matter so expelled, which is also called *sputum* or *phlegm*.

**THE ACT OF EXPECTORATION.**—The smaller bronchial tubes are kept free from obstruction by the action of ciliated epithelium. The area of the smaller tubes being greater than that of their trunks, the air passes more forcibly through the latter, and so tends, even in natural breathing, to carry speedily away any accumulated secretion. The forcible acts of coughing (*see* COUGH) and 'hawking' increase the natural force and fulness of the expiratory effort, and clear the air-passages; the repeated closure of the glottis in coughing increases still further the expulsive effect, by causing the air to escape in sudden jerks. If the glottis cannot close in consequence of laryngeal disease, the act of expectoration becomes painful and difficult. Efforts at expectoration are also laboured and futile if the power to take a deep inspiration is lost in consequence of emphysema or muscular weakness.

Inability to expectorate is often the immediate cause of death, the 'suffocative catarrh' of the dying being another name for accumulation of phlegm which the patient is powerless to remove. By teaching the patient 'how to expectorate,' by the administration of a timely stimulant or a quickly acting emetic, or by change of posture, life may in such a case be prolonged. Should the sufferer be allowed to get flurried, the breathing becomes more and more shallow, and deep inspiration and free expectoration are impossible. If, however, the patient can be induced to breathe calmly and deeply, to assume a more easy posture, and to swallow a mixture of ammonia with ether, the breathing gradually becomes less shallow and rapid, air enters the deeper parts of the lung, and power is gained to evacuate the accumulated secretion (*see* STERTOR). The act of expectoration is, as a rule, most easy in that posture in which respiration is most free. Sometimes when the secretion comes mainly from one lung, the aid of gravitation may be called in to empty the obstructed tubes.

**CHARACTERS AND VARIETIES.**—Before auscultation was practised, diagnosis was often based on the character of the expectoration, unwarrantable importance having been attached to the distinction between pus and mucus, on the assumption that pus was diagnostic of phthisis. At the present time we are apt to lose much useful information by falling into the opposite error.

In health, the secretion from the mucous membrane of the air-tubes is a transparent, colourless, slightly glutinous liquid, like thin mucilage; it contains mucin, a varying quantity of saline matter, and water.

The saline matter is abundant in the transparent viscid expectoration, deficient in the opaque and less tenacious kind, least in that which is actually purulent.

The ordinary mucous secretion is increased in quantity and viscosity as a result of simple catarrhal inflammation of the bronchial membrane. When bronchitis has existed for some days, a change occurs in the character of the secretion; instead of being transparent and viscid, it becomes semi-transparent and then opaque, the colour changing to a yellow or greenish hue.

The sputum becomes frothy from the admixture of air; and rusty or prune-juice-coloured if the inflammatory action extends to the ultimate bronchial ramifications, and is of so intense a kind as to allow of oozing from the capillary vessels.

Fibrinous moulds of the bronchial tubes, or chalky masses consisting of inspissated and calcified cheesy matter, are not infrequently expectorated.

The dark grey or blackish stain often seen in expectoration may be derived from carbon in the atmosphere; or, if it fade on the addition of nitric acid, it may be due to pulmo-

nary pigment formed under slight irritation.

The expectoration may afford important aid in diagnosis, as may be illustrated by the following examples: If a person with severe chest-complaint coughs frequently and spits only frothy salivary fluid, we may suspect pleurisy. If the fluid is glairy, like white of egg, we may suspect bronchitis. If it has a rusty tinge and resembles thick gum-water coloured with blood, we are not likely to err in recording pneumonia. If there is a sudden gush of foetid pus we may diagnose abscess in the lung or an empyema.

Purulent expectoration may occur in bronchitis as well as in phthisis; but if long continued, and unaccompanied by distinct rhonchus, it almost always comes from a vomica. The excessively offensive expectoration of pulmonary gangrene, and that arising from a bronchiectatic sacculæ, are diagnostic of the affections from which they spring.

In phthisis the sputum is at first salivary or frothy, the result of irritation; then viscous, indicative of more confirmed affection of the mucous membrane; and subsequently dotted and streaked with blood. Whitish opaque spots, giving a pearly aspect to the expectoration, next appear; these enlarge, become flocculent and ultimately nummular, being fissured and moulded in a cavity. As the disease advances and involves both lungs, the expectoration is entirely purulent, and shortly before death is often surrounded with a pinkish halo. On placing under the microscope one of the small pearly points described, masses consisting of several air-cells choked with granules, or mere fragments of elastic tissue may occasionally be seen. By the addition of acetic acid the sputa may be rendered transparent, and the elastic tissue is then, if present, more certainly detected; but practically the experienced eye is the best guide in the selection of those small pin-head flocculi of expectoration, in which the microscopic particles of lung-tissue are to be detected.

The microscope may also be helpful by indicating, from the character of the cells, the part of the respiratory tract involved, and the degree of disease existing. (For the detection of tubercle bacilli, a point of great practical importance, see PHTHISIS; and SPUTUM, Examination of.) The existence of cancer of the lung may sometimes be detected by a microscopical examination of the expectoration.

**TREATMENT.**—In treatment, much may be gained by study of the expectoration. The cough may often be relieved, and the dyspnoea and other symptoms removed, by effecting an alteration in the nature of the secretion.

If by the frothy character of the sputa congestive disorder is indicated, this may be met by the application of warm poultices,

turpentine stupes, or hot flannels externally; and by such means as are calculated to reduce fever and irritability, namely, the administration of salines with antimony or aconite. If the expectoration is too viscid and glutinous for easy removal, lemon-juice, liquor potassæ, soda, or various inhalations give relief, by lessening the tenacity of the secretion; or the change from tenacious and transparent to opaque, less adhesive secretion may be hastened by giving iodide of potassium with a few drops of antimonial wine.

When muco-purulent secretion is established and shows no sign of diminution, the use of senega and of the gum-resins is indicated; these may be given in the form of lozenge, while benzoin, tolu, and copaiba are also of value. Acetic or tannic acid, given in small and frequently repeated doses, reduces the quantity of secretion.

When the combined glairy and muco-purulent condition of the expectoration and other symptoms give evidence of bronchitis of an established kind, associated with gout or abdominal torpor, a combination of calomel with antimony and guaiacum (as in Plummer's pill) is of the greatest service; but this treatment needs perseverance, discrimination, and watchfulness. The morbid surface may at the same time be medicated by the inhalation of tar, creasote, or oil of juniper, until, with the improvement of the general health, under cod-liver oil and iron, the evil is entirely removed.

E. SYMES THOMPSON.

**EXPOSURE, Effects of.**—Were the term 'exposure' to be taken in its widest significance, it might fairly claim to include a range of subjects only bounded by the limits of practical medicine. Infection naturally implies exposure to some contagious influence, poisonous gas, or malarious or other unhealthy emanation, bearing in its train a formidable sequence of ill results, and probably depending on the introduction within the system of a specific form of microbe. Many of the disorders which impair our comfort and shorten our lives may, indeed, be traced, in some degree, to exposure of some kind, to an excess of heat or of cold, to a variety of complicated reactions, arising, in whole or in part, from unnatural impressions made on the physiological processes of life by various external agencies. Any study of our subject, however, from this extended point of view, would clearly be out of place here, and effects of extreme elevation and depression of temperature will be fully considered elsewhere; so that exposure, in the sense in which it will be treated here, may be narrowed down to the results which ensue when persons of average constitutional power are submitted, for a longer or shorter time, to the influence of ordinary cold, or wet, or damp. Now, it is an old saying, that if five

or six people, of either sex, suffer shipwreck, or are wet through, or excessively chilled by moist cold, each will probably suffer in some different way. One may altogether escape, and not experience inconvenience of any kind; a second may 'catch'—as the popular expression puts it—a sore-throat, or a 'bad cold;' whilst a third may be seized with pneumonia; and a fourth with rheumatic fever.

Individual constitution or idiosyncrasy partly explains these differences, and the familiar term of a 'weak point somewhere,' if not conveying much impression of scientific accuracy, is right so far in showing that the internal congestion caused by sudden contraction of cutaneous arterioles is most naturally directed to that organ whose vessels have been weakened by previous inflammation. If the patient, therefore, have previously suffered from tonsillitis, or bronchial congestion, or rheumatism, he will be predisposed to a recurrence of the same affection on a renewal of the exciting cause. If, again, from sedentary occupation and over-indulgence in nitrogenous food, his blood be overcharged with the products of retrograde metamorphosis or of imperfect assimilation, it only needs the closure of the eliminatory agency of the skin, to provoke the irritation of internal organs, and to induce an attack of gout or rheumatism, or an acute inflammation of kidneys or liver. The effects of exposure, even in this limited sense, are thus tolerably various both in extent and in degree, and may be studied on a large scale during our greater campaigns, and in lesser degree during the ordinary autumn manœuvres, when the weather happens to be unfavourable. Here, however, everything injurious is minimised by the healthy condition of the men, their good clothing and food, and the enforced regularity of habits, which prevent, in great measure, one of the most fertile causes of damage from exposure; and among the most efficient means for preventing the potent influence of bacilli for evil, we must place all hygienic means which improve the bodily vigour. It is now well known that nothing tends so seriously to impair the power of bearing either extreme of temperature as excessive indulgence in strong drink; so that in treating anyone who has been exposed to the influence of cold and wet, we must not forget to take also into account the probable combination of the depressing effects of alcohol on the nervous system. Illustrations of this must be familiar to all, and we must all have met with numerous cases in which tramps or other persons with weakened bodily vigour have been brought into our hospitals, suffering from the effects of exposure in various degree.

**TREATMENT.**—In seeking to remedy the effects just described, it will be found that some of the cases will recover under the

influence of warmth and good food, and the generally invigorating effects of careful nursing; whilst others, on the other hand, may succumb to the rapidly destructive tendencies of acute disease, or to the slower pathological processes of chronic lung or kidney degeneration. In the treatment, therefore, of such cases, we must not only add to the genial influences of home or hospital care such special drugs as the varied development of symptoms may require, but we must carefully take into account the occurrence of various serious or profoundly disorganising complicating causes. Thus if our patient has been found in an insensible state, we may reasonably suspect cerebral hæmorrhage or alcoholic or narcotic poisoning, and act accordingly. And only when all such suspicion is finally removed can we rest on our oars, and confine our attention to the immediate effects which follow exposure of the kind indicated above. Warmth, rest, and the regulated use of stimulants and food will now be indicated, and must be employed, with all the precautions suggested by the ordinary principles of therapeutics and by common sense.

ROBERT FARQUHARSON.

**EXSANGUINE** (*ex*, without; and *sanguis*, blood).—Deprived more or less of blood—bloodless. Sometimes used synonymously with anæmic. See ANÆMIA.

**EXTRA-UTERINE FŒTATION.**  
See PREGNANCY, Diseases and Disorders of.

**EXTRAVASATION** (*extra*, without; and *vasa*, vessels).—SYNON.: Fr. *Extravasation*; Ger. *Extravasat*.

**DEFINITION.**—Extravasation is the escape of any of the fluids of the body, normal or abnormal, from the vessel, cavity, or canal that naturally contains it, and its diffusion into the surrounding tissues. The result of the effusion is also called an *extravasation*.

Extravasation, being the effect of rupture or perforation of the walls of a hollow organ, may be due to injury, to weakness of the parietal structures, to morbid conditions of the blood, or to increase of internal pressure. See PERFORATIONS AND RUPTURES.

The fluids most frequently found extravasated are blood, urine, bile, the contents of the alimentary canal, and certain constituents of morbid growths and fluid collections. The present observations will have reference only to extravasation of blood; other kinds of extravasation being described under the heads of the several organs involved.

**Extravasation of Blood.**—The blood is peculiarly liable to extravasation, the vessels being universally distributed, much exposed to injury—a very common cause of this lesion—and subject also to a constant pressure from within, which may be suddenly and greatly increased. Any portion

of the circulatory system may give way: the heart, as in fatty degeneration; the arteries, as in aneurysm; the capillaries, as in pulmonary hæmorrhage; the veins, as in the subcutaneous rupture of a varix.

When the effusion takes place into one of the serous sacs, it forms a collection of blood, variously named *hæmothorax*, *hæmatocele*, &c. If the subcutaneous, submucous, or other connective tissue, or the substance of an organ, be invaded, the effused blood finds its way between the elementary textures, separating and compressing them; and there are formed what are described simply as *extravasations* of blood, or more definitely, according to their extent, *parenchymatous* or *interstitial hæmorrhages* or *apoplexies*, *suffusions*, *ecchymoses*, *petechiæ*, or *vibices*. The extravasated blood generally coagulates; the fibrin and albuminous substances are absorbed; and the products of the decomposition of hæmoglobin, which are much more slowly removed, give rise to the familiar discolorations. The seat of effusion first appears of the colour of blood or its derivatives; but if much below the surface, the extravasation may not be visible for some days, after which time it appears of a bluish, greenish, or yellowish hue. Much less frequently the effused blood becomes encapsuled, forming a *hæmatoma* or *blood-cyst*, such as is occasionally seen in connexion with the chest or abdomen. In other cases it decomposes, and sets up gangrene around, or inflammation of the nature of severe cellulitis, which may end in ulceration. When the blood is forced between the coats of the perforated vessel, there is formed what is called a *dissecting aneurysm*. If the extravasated blood escape from the surface of the skin or mucous membranes, one form of *hæmorrhage* or a *bloody flux* is the result.

The *symptoms* of extravasation of blood are so various that they cannot be stated in general terms. If extensive and affecting vital parts, the effusion may be attended with shock, syncope, or death; and it may lead to constitutional disturbance in many forms. Local pain is not common unless the extravasation be severe. The pressure of the effused blood upon the vessels, nerves, and muscles of the part—in a limb, for example—may produce paralysis, disturbances of sensibility, œdema, loss of temperature, and even gangrene. As a rule, however, extravasations of blood are limited in size, give rise of themselves to no serious symptoms, and readily disappear by absorption.

J. MITCHELL BRUCE.

**EXTROVERSION** (*extra*, outwards; and *verso*, I turn).—The eversion or turning inside out of a part, as the eyelids or bladder. In the bladder, extroversion is associated with that condition, usually congenital, in which the anterior wall of this organ and of

the abdomen is deficient, and its posterior wall projects through the opening thus formed.

**EXUDATION** (*exudo*, I sweat).—The process by which certain of the elements of the blood pass through the walls of the blood-vessels into the surrounding tissues, as in inflammation. The term is also used to indicate the products of this process, when they are of a fibrinous or coagulable character. See INFLAMMATION.

**EXUDATION - CORPUSCLES.**—The cells found in inflammatory products, whatever their origin may be. See INFLAMMATION.

**EYE, and its Appendages, Diseases of.**—SYNON.: Fr. *Maladies des Yeux*; Ger. *Augenkrankheiten*.

It will be most convenient to arrange the consideration of the diseases of the eye according to the anatomical order of the structures affected, namely: I. Diseases of the Conjunctiva; II. Diseases of the Cornea; III. Diseases of the Sclerotic; IV. Diseases of the Iris; V. Diseases of the Crystalline Lens; VI. Glaucoma; VII. Diseases of the Optic Nerve and Retina; VIII. Diseases of the Choroid; IX. Diseases of the Vitreous Body; and, X. Diseases of the Eyelids.<sup>1</sup>

#### I. Diseases of the Conjunctiva.

1. *Inflammation.*—SYNON.: Conjunctivitis. The conjunctiva is exceedingly liable to inflammation, and its inflammations are commonly arranged in groups, which are sufficiently distinct in their typical examples, but are not separated by any definite boundary lines.

In their earlier stages inflammations of the conjunctiva possess many characters in common. They are all attended by the four signs of inflammation—heat, redness, swelling, and pain; although both the heat and the swelling are usually kept within limits by the discharge, which is often free, sometimes profuse, at first mucous, afterwards mucopurulent or truly purulent in character. If, however, the exudation be of a firmer consistence than usual, it not only produces a superficial discharge, but distends the meshes of the sub-mucous tissue, elevates the conjunctiva from the sclerotic, and causes it to overlap the corneal margin as a swollen ridge; a condition which is known as *chemosis*. The redness depends, of course, on the degree of the congestion, which may or may not be sufficient to obliterate the intervascular meshes, and to produce a uniform colour; and the pain is not severe, except in cases of very dense sub-conjunctival swelling. From other forms of inflammation, that of the conjunctiva is distinguished by certain negative characters. Unless as a result of secondary changes, or as a mechanical effect of the

<sup>1</sup> See also VISION, Disorders of; OPHTHALMOSCOPE; and OPHTHALMOSCOPE IN MEDICINE.

presence of a film of turbid secretion, the transparency of the cornea is not affected, and the acuteness of vision is not impaired. The congestion is limited to the conjunctiva, and the distended vessels can be emptied for a moment by pressure through the lower lid, so as to reveal a glimpse of the white sclerotic underneath.

In mild cases conjunctivitis is an unimportant affection, but in its more severe forms it is attended by two distinct sources of danger. During its acute stage it may produce partial or complete necrosis of the cornea, leading to great impairment of sight and not seldom to blindness; and when chronic it often occasions great hypertrophy of the papillæ of the portion of the membrane which lines the lids. These papillæ may even become converted into shaggy or warty excrescences, which mechanically irritate the corneal surface, and cause the development of vessels beneath its epithelium. In time the papillæ dwindle, and the effusion round about them contracts, thus rendering the tarsal cartilage (especially that of the upper lids) incurved, and bringing the cilia to rest upon the surface of the eyeball. Great distress and permanent impairment of vision may be thus occasioned; and the progress of the contraction may be so slow as to deprive it of any manifest connexion with the inflammation in which it had its origin.

The chief varieties of conjunctivitis are: (a) *infantile*; (b) *simple* or *catarrhal*; (c) *contagious*; and (d) *diphtheritic*.

(a) *Infantile Conjunctivitis*.—SYNON.: *Ophthalmia neonatorum*.

**ÆTIOLOGY AND SYMPTOMS.**—Infantile conjunctivitis is probably due, in most cases, to direct inoculation with vaginal secretion. It usually commences on or about the third day after birth, and passes rapidly into the purulent form. It is attended by considerable puffy swelling of the lids, and by profuse thick discharge, which soon dries upon the tarsal margins, and often causes them to cohere. If neglected or improperly treated, the disease often leads to sloughing of the cornea; but it may always be cured if it is seen while the cornea is still bright.

**TREATMENT.**—The treatment required is to wash away the discharge carefully and frequently; to apply an astringent lotion (the best is a solution of two grains of nitrate of silver to an ounce of distilled water) to the conjunctival surface every four hours, or less frequently when improvement is established; and to anoint the edges of the lids with simple ointment to prevent their agglutination. In cases of inherited syphilis mercurial inunction should be prescribed. The infant's food must be carefully regulated if it is brought up by hand; and if it is very feeble, it may take a little cod-liver oil, combined in an emulsion with two-minim doses of liquor einchonæ. Where the mother's milk fails, it

is often desirable to obtain a wet-nurse; but the local treatment is that which is chiefly important, and upon which, in most cases, entire reliance may be placed.

(b) *Simple* or *Catarrhal Conjunctivitis*.

**ÆTIOLOGY AND SYMPTOMS.**—This form of conjunctivitis is usually due to exposure to cold or to some chemical or mechanical irritant. Its most distinctive character is that the discharge is chiefly mucous, and has not much tendency to become purulent.

**TREATMENT.**—In every case the surface of the eyeball, and the lining membrane of the lids, should be carefully examined for any foreign body which may be the cause of the trouble; and if such should be found and removed, there will seldom be need for further treatment. If there is no foreign body, the two-grain solution of nitrate of silver, or of sulphate of zinc, may be placed within the conjunctival sac, by means of a quill or dropping-bottle, every two or three hours, and speedy recovery will usually be the result. In cases of very mild type, one or two applications of the astringent may be sufficient; and these may be supplemented by the use, in the same manner, of a solution of cocaine with boric acid. A good formula for this purpose contains two grains of cocaine hydrochlorate, and four of boric acid, to the ounce of distilled water. During the treatment the eyes should have functional rest, and should be sheltered from external cold, from dust, and from glare.

(c) *Contagious Conjunctivitis*.—SYNON.: *Purulent Ophthalmia*.

**ÆTIOLOGY AND SYMPTOMS.**—In this form the discharge rapidly assumes a purulent character. This may happen when the simple form is aggravated by the state of the patient or by accidental circumstances; or when the disease is produced by inoculation with the discharge from a similar case, or with contagious pus from the urethral or vaginal mucous membrane. The state of the patient which is most likely to promote the development of the contagious form is that in which the conjunctiva is beset with the granular semi-transparent bodies, formed by aggregations of lymph-corpuscles, which are known as 'sago-grains' or follicular granulations. These bodies are commonly present in the eyelids of persons (especially of young persons) who are crowded together under insanitary conditions of living, as in barracks, camps, or badly regulated schools; and their presence renders the conjunctivitis of simple irritation prone to assume a purulent character. It is impossible to say how soon the discharge of conjunctivitis becomes contagious; or capable of reproducing the disease in others; but its activity in this respect seems to bear some proportion to the intensity of the inflammation which produces it; and the activity of gonorrhœal pus is probably greater than that of any conjunctival product. In the

more intense forms of purulent ophthalmia there is great swelling of the lids and of the ocular conjunctiva, early chemosis, and a tendency to speedy sloughing of the cornea; while the milder forms pass into the catarrhal by imperceptible gradations.

**TREATMENT.**—In the space available for the purpose in these pages, the treatment of contagious conjunctivitis cannot be described in detail. It mainly rests upon the action of local astringents, graduated in strength according to the severity and the stage of the disease. In the worst cases the eyelids must be everted, and the whole of the palpebral conjunctiva carefully touched with a stick composed of one part of nitrate of silver fused with four parts of nitrate of potash. The caustic should be neutralised by a drop or two of a solution of common salt, applied by means of a camel's-hair pencil, before the lid is suffered to return into contact with the cornea; and the cauterisation must be done carefully and with a light hand, so that the resulting eschar may include only the epithelium; for if the basement membrane is destroyed, there will be danger of subsequent adhesions between the eyelids and eyeball. The cauterisation should be repeated about every eight hours, or as soon as the eschar falls, and in the intervals, if the patient is awake, the conjunctiva should be gently sprayed or syringed every hour with a weak antiseptic lotion at a comfortable temperature. Diluted Condy's fluid, solution (1 in 5,000) of mercuric perchloride, or (15 per cent.) of Barff's boroglyceride, are all suitable for this purpose. The tension of chemosis may be diminished by radial incisions, outwards from the corneal margin, carried nearly down to the sclerotic. The strength must be supported by good diet and tonics; and the nervous system calmed by anodynes. In milder cases the principle of treatment must be the same, but the local applications less severe; and in chronic cases the local applications must be continued, after apparent recovery, so long as any residual thickening, capable of undergoing eventual contraction, can be found lurking in the palpebral folds.

(d) *Diphtheritic Conjunctivitis.*—This malady has chiefly been made known to us by the observations of German writers, and very few authentic instances have been recorded in this country. But it has from time to time prevailed extensively in Berlin, and might at any time make its appearance amongst ourselves. The writer has seen one remarkable instance in which the disease occurred with great severity in a child, whose eyes were bathed with milk as a domestic remedy for what seems to have been a mild ordinary conjunctivitis; and the coincidence seems to have a special interest in relation to recent researches into the aetiology of diphtheria.

**SYMPTOMS.**—Diphtheritic conjunctivitis is

attended with great heat and pain, and with very hard, brawny swelling of the eyelids; but its most characteristic symptom is the infiltration of the sub-conjunctival tissue by the so-called diphtheritic effusion, which does not form a pellicle upon the surface, but distends and fills the cavities of the areolar tissue. The result is to produce a pale, firm swelling of the conjunctiva, and a great tendency to rapid sloughing of the cornea. The subjects of the malady are mostly feeble and badly fed children, and the cases are described as being almost hopeless unless early admitted into hospital.

**TREATMENT.**—The indications for treatment are chiefly to support the strength by suitable regimen; to apply ice or bags of freezing mixture to the lids during the hot stage; and to change the cold applications for hot ones as soon as the period of resolution, absorption, or repair can be said to have commenced. In the meantime iron and quinine should generally be administered internally.

*Pemphigus of the Conjunctiva.*—A very formidable, but fortunately rare, variety of conjunctival disease appears to be a kind of pemphigus. See PEMPHIGUS.

**ÆTIOLOGY AND SYMPTOMS.**—In its early stage pemphigus of the conjunctiva displays itself by the formation of bullæ, which are of small size and inconspicuous, and are seldom followed by the raw surfaces commonly left elsewhere. This stage seldom excites much attention, and it is followed by a slow process of conjunctival atrophy, which, in time, leads on to disturbance of the corneal epithelium, and ultimately to complete opacity. The conjunctival atrophy gradually obliterates the folds of the membrane, and renders the lids immovable upon the globe, frequently causing also inversion of cilia, which add to the pre-existing irritation.

**TREATMENT.**—The only hope of dealing successfully with conjunctival pemphigus appears to depend upon the early recognition and constitutional treatment of the general malady; for which purpose the chief reliance must be placed upon the preparations of arsenic, in combination with tonics and anodynes. When the period of atrophy has commenced, it is seldom, if ever, arrested. The apparent adhesions between the lids and the globe often suggest operative measures, but these are not recorded ever to have been successful. In one case, the writer dissected off the whole of the affected conjunctiva of the globe, and replaced it by transplanting that of a rabbit. The graft united well, and, for a time, the disease appeared to be arrested. In a few weeks, however, or as soon as the graft had become completely dependent upon the nutritive processes of the patient, the new structure became affected by atrophy, and the malady pursued its usual destructive course.

2. *Episcleritis*.—An affection which is apparently but not really conjunctival, being situated in the tissue between the conjunctiva and the sclerotic, is that which has received the name of *episcleritis*, and which was formerly described as 'scleritis with inflammation of the insertion of a rectus muscle.'

**SYMPTOMS.**—It appears as an elevated patch of congestion, gradually passing into the natural level and appearance of the parts, and seated on the ocular surface near the corneal margin, most frequently on the temporal side. On close examination, the congestion, with the exception of a few dilated vessels, is seen to be sub-conjunctival, and to be attendant upon a circumscribed but not sharply defined swelling or thickening, which is adherent to the sclerotic, and which presents, in the interstices between the blood-vessels, an appearance as if it consisted of some new deposit, generally of a yellowish tint, external to that membrane. The swelling is indolent, chronic, and in itself generally painless, although it is sometimes accompanied by severe neuralgia. The subjects are most frequently women, especially such as are anæmic or otherwise out of condition. *Episcleritis* may last for months with little change, and it seems to be harmless as regards the other structures of the eye.

**TREATMENT.**—The writer has found *episcleritis* resist all medication except the internal administration of mercury, to which it will often yield in the course of a short time. The best preparation is the perchloride, in doses not exceeding  $\frac{1}{16}$  of a grain, which may usually be combined with five or ten minims of the tincture of perchloride of iron, and often with quinine. At the same time it is often desirable to sprinkle a little dry calomel over the swelling once in twenty-four hours; but this application is less important than the internal treatment, and should not be continued unless it is soon and distinctly beneficial. When the congestion of the patch is considerable, the action of other remedies will often be promoted by the occasional application of a leech to the temple of the affected side.

3. *Hæmorrhage*.—Effusion of blood beneath the conjunctiva may occur spontaneously, but is generally traceable either to a direct injury or to violent exertion. Thus it may follow slight blows upon the eyeball, as from a twig or switch; or may be produced by a paroxysm of coughing, especially in pertussis; or may occur from the rupture of a vessel during parturition, or upon lifting a heavy weight. It is always unsightly. When traceable to any of the foregoing causes, the hæmorrhage is usually a matter of no moment; but when it happens during the night in young people, it should lead to a suspicion of nocturnal epilepsy, which has often been first discovered by its means. Moreover, in advanced life, more especially when occur-

ring without adequate cause, it may point to arterial brittleness, of a kind which may indicate danger of a like hæmorrhage within the cranium. On these grounds it is a symptom which always calls for full inquiry into its causes, and which may sometimes afford useful warning of impending danger.

**TREATMENT.**—Absorption may be promoted by covering the closed lids by a compress moistened with a lotion of spirit and water, or of tincture of arnica and water if an appearance of more decided medication is desired.

II. **Diseases of the Cornea.**—Diseases of the cornea, as already indicated, are often secondary to those of the conjunctiva, and may arise in their course as complications.

### 1. *Ulceration*.

**ÆTIOLOGY AND SYMPTOMS.**—To the present group belong all the corneal ulcerations of purulent ophthalmia, whether infantile or of a later period of life; and also the forms of corneal ulcer which are produced by the friction of eyelids rendered rough by inflammation, or by the friction of eyelashes which have been turned inwards by distortion of the tarsal cartilage. When ulceration of the cornea occurs in the course of conjunctivitis, it at once invests the latter malady with a highly formidable character. The corneal tissue, once destroyed by ulceration, is not reproduced in its original transparency, but only as a more or less dense and opaque white cicatrix, which is at the same time disfiguring to the appearance and an impediment to vision. If the ulcer should perforate, the iris almost necessarily becomes adherent to the cicatrix; and if the loss of substance is of large superficial extent, the resulting cicatrix is often thin and feeble, so that it is rendered prominent by the pressure within the eye, producing the condition which has been called 'staphyloma,' and gradually elevating and distorting the surrounding portions of clear cornea. The first effect of the healing of a corneal ulcer is generally to flatten the natural curvature of the membrane; but the secondary effect, if the cicatrix becomes prominent, may be to modify this curvature in various ways. Hence it follows, even when a cicatrix of the cornea is surrounded by a still transparent annulus, behind which an artificial pupil may easily be made by the excision of a portion of the iris, that the surgeon cannot predict with any certainty the quality of the vision which will be obtained, unless he is able, before operating, to determine the state of the corneal curvature. This is only possible when the margin of the pupil is so far free that it can be dilated with atropine sufficiently to render the fundus of the eye visible with the ophthalmoscope. When this can be done, any portion of cornea through which a clear view of the retinal vessels can be obtained by the surgeon

will also afford clear vision of external objects to the patient; but, if no such place can be discovered, a very cautious opinion should be given with regard to the degree of benefit which may be hoped for from an operation.

**TREATMENT.**—The extreme importance of the cornea to the visual function renders it necessary that its integrity should be guarded with the greatest possible care. In any case of conjunctivitis, of even moderate severity, the cornea should be watched from day to day, and any appearance of turbidity about its central portion, or of elevation or irregularity of the epithelium at its margin, should lead to a reconsideration of the treatment which is being pursued. The former of these conditions is the ordinary precursor of sloughing ulcer or necrosis; the latter, of the extension to the cornea of an inflammatory process.

The general principles which govern the treatment of sloughing ulcer are, that the eye should be kept under the influence of eserine, which has a marked effect in checking the extension of ulceration by arresting the migration of white corpuscles; that any astringents which may be applied to the conjunctiva should be prevented from coming into contact with the cornea; that strength should be supported, pain relieved, and local nutrition stimulated by hot applications. When the ulcer continues to spread, its progress may often be arrested by diminishing the tension of the globe by the evacuation of the aqueous humour; and this may be accomplished either by repeated paracentesis at the corneal margin, or by Saemisch's method of cutting through the base of the ulcer, and reopening the incision daily until a process of repair is well established, or by the performance of iridectomy. Of these three courses, the last-named is the most generally applicable. It not only produces the immediate effect which is desired, but it has also the incidental advantage of establishing an artificial pupil at the side of the cicatrix.

The application of eserine is best effected by using a solution of the neutral sulphate, of the strength of four grains to the ounce of distilled water; and a drop of this solution may be placed within the conjunctival sac twice or thrice daily.

The sloughing ulcers of the cornea which arise from causes other than conjunctivitis, as injury, or failure of nutrition, must be treated upon the principles which have already been laid down. There are, however, inflammatory ulcers which require the use of local applications of a stimulating kind, among which dry calomel and other mercurials hold a prominent place. Such ulcers have usually a leash of vessels running from the corneal margin, and are often obstinately recurrent; whence they are known as 'recurrent vascular ulcers.' They are usually connected with some manifest systemic de-

range, and are often attended by photophobia. They leave scars upon the cornea, of a size and opacity proportionate to their extent and depth; and on this account it is desirable, whatever constitutional treatment may be required, to heal the ulcers themselves as speedily as possible, by the aid of local applications. In the early stages of the ulceration, eserine should be used; but afterwards either dry calomel or an ointment containing from ten to thirty grains to the ounce of the precipitated yellow oxide of mercury. This ointment was introduced into practice by the late Dr. Pagenstecher, of Wiesbaden, and is often called by his name. In very obstinate cases the late Mr. Critchett recommended setons in the temporal regions; but the operation of iridectomy will often afford a still more efficacious remedy. Where there is much photophobia, the use of eserine is especially indicated, because, in addition to its action above mentioned, it produces contraction of the pupil, and thus gives comfort by lessening the quantity of light which is admitted into the eye. If the photophobia is very severe or intractable, it is often beneficial to divide the orbicularis muscle freely at the outer canthus, so as to diminish the pressure which is caused by its spasmodic contraction, and which sometimes seems to be a chief cause of the irritability. After such an incision, a cold compress should be applied, and the patient kept in the dark for a few hours, when there will often be a marked alleviation of this distressing symptom, and a greatly increased general amenability to treatment.

**2. Inflammation.**—**SYNON.**: Keratitis.—Inflammation of the cornea presents three distinct types, the *suppurative*, the *vascular*, and the *interstitial*.

(a) *Suppurative Keratitis.*—Suppurative inflammation or abscess of the cornea seems to be essentially a phlegmon or boil of the corneal tissue, a portion of which dies, and is cast off in the form of a slough.

**SYMPTOMS.**—The abscess commences as a very tender grey spot in the cornea, surrounded by a zone of turbidity, and accompanied by a good deal of ciliary neuralgia, as well as by a variable degree of lachrymation, conjunctival congestion, and intolerance of light. Under the influence of atropine, hot fomentations, and such constitutional treatment as the state of the patient may demand, the threatened suppuration is sometimes averted, and the turbidity clears away. More commonly pus is formed, and makes its way either externally, leaving an ordinary ulcer; or internally, producing the condition called *hypopyon*, in which there is pus in the anterior chamber. Sometimes it separates the corneal laminae, by gravitation, to a considerable extent before perforating them, and is then called *onyx*, from a resemblance to the lunula at the base of a finger-nail.

**TREATMENT.**—When suppuration is no longer doubtful, the best practice is to evacuate the abscess by a puncture from within, by thrusting a cutting needle into the anterior chamber near the corneal margin, and then causing its point to penetrate the cavity of the abscess. The mingled pus and aqueous humour will escape as the needle is withdrawn, and its wound of entrance may be reopened once or twice daily by a probe, so as to insure the complete removal of all inflammatory products, until the healing process has made some way. If the abscess has burst internally before the case is seen, atropine should be applied without delay. If the pupil dilates fully, and the quantity of pus in the anterior chamber is but small, the case may be left to the *vis medicatrix natura*, care being taken to enforce rest and to exclude noxious influences. If the quantity of pus is large, it should be let out by paracentesis; and if the atropine reveals adhesions of the iris, iridectomy should at once be performed. An abscess which has burst externally leaves simply an ulcer, generally with a disposition to heal readily, and requiring only such treatment as has already been described.

(b) *Vascular Keratitis.*—Vascular inflammation of the cornea is often a very severe and protracted malady, which usually leaves behind permanent opacity and impairment of sight.

**SYMPTOMS.**—It commences, in typical cases, by the formation of two crescent-shaped patches of vascularity, one at the upper and the other at the lower portion of the cornea. The patches are formed by the development on the corneal surface of innumerable fine blood-vessels, so closely packed together that the interstices which separate them are scarcely discernible by the naked eye, and the affected parts present a uniform aspect of vivid redness. The crescents are somewhat elevated above the general corneal surface, and each crescent is bordered, along its concave or advancing edge, by a line of precursory epithelial turbidity. At the same time that the crescents increase in size, the borders of precursory turbidity are pushed before them; until at first these, and afterwards the crescents themselves, may meet and coalesce on the horizontal diameter of the cornea, so that its whole surface may become uniformly red. When this stage is reached, vision is almost abolished, but there may still be much intolerance of light. The corneal tissue is softened, and its margin is surrounded by a zone of sclerotic vascularity, which is visible through the congestion of the conjunctiva. As the inflammation subsides, the vascular crescents slowly recede from the centre of the cornea towards its circumference, and finally disappear, leaving behind them a dense opacity of an extremely obstinate

character. A severe case of vascular keratitis generally affects both eyes, and threatens a long period of actual blindness, followed by a long period of very imperfect vision. The worst examples of the malady are those which have been treated at the outset by astringent or irritating applications; and, even in the slighter forms, the malady is nearly always obstinate and protracted. As long, however, as the march of the vascular crescents, or their precursory turbidity, has not encroached upon the portion of cornea in front of the pupil, so long vision is not seriously jeopardised.

**TREATMENT.**—The great object of treatment is to arrest the new vascular development at a comparatively early stage; and for this purpose it is necessary to have recourse to eserine and sedatives locally, and to such constitutional treatment as the general condition may require. A solution of two grains of the neutral sulphate of eserine, with from two to four grains of hydrochlorate of cocaine, in an ounce of distilled water, should be dropped into the eye twice daily, and the closed lids should be frequently fomented with hot poppy decoction, or with cold solution of extract of opium in water, according as one or the other temperature is the more agreeable to the feelings of the patient. Apart from any special indication, the medicines most generally useful are those which appear to influence local nutrition through the central nervous system, such as the iodide and bromide of potassium, and the sulphate of quinine. These, if they are likely to exert a beneficial effect, generally do so speedily; and if the malady should be rapidly extending, it is always prudent to reconsider the prescription without much loss of time. In severe cases, especially when the lids are somewhat tumid and there is much photophobia, a leech may often be applied with advantage, usually over the temporal muscle, close to the outer margin of the orbit, or a little blood may be taken more rapidly, by means of Heurteloup's artificial leech; and when, notwithstanding treatment, the malady pursues its course unchecked, a large iridectomy should be made without undue delay. The operation not only tends to arrest the vascular formation, but it also leaves a lateral pupil through which good sight may often be obtained long before the transparency of the central parts of the cornea is restored. In some cases, the writer has seen excellent results from peritomy; that is, from excising the annulus of conjunctival and subconjunctival tissue which immediately surrounds the cornea. The strip excised should be about 3 mm. in width; and the excision may sometimes be limited to the bases of the vascular crescents.

*Development of vessels upon the cornea.*—It is necessary carefully to distinguish vascular keratitis, properly so called, from that

development of vessels upon the cornea which may occur in connexion with the cicatrisation of ulcers, or in consequence of the friction of lids left granular by conjunctivitis. In both these forms the new vessels are arborescent and irregular in their distribution, instead of being closely packed together; and they are not attended by the pink zone of circum-corneal congestion, which is never absent in true corneal inflammation.

The vessels which attend the formation of cicatrices generally dwindle in course of time; while those produced by granular lids, and which, when they are very abundant and closely set, constitute the condition called *pannus*, will often disappear without direct treatment if the state of the lids themselves can be favourably modified by the application of astringents or by other means. When residual granulations are large and numerous, they may be treated by crushing them with forceps, by scraping, or by electrolysis; for which purpose they should be separately punctured by a platinum needle, connected with the negative pole of a galvanic battery. A weak current only is required, and it may be suffered to pass through each granulation for from ten to twenty seconds. In many cases the vascular network of *pannus* will be comparatively absent from the lower third of the cornea, so that sight may be much improved by an artificial pupil made in a downward direction. When *pannus* covers the whole cornea with a close vascular network, so that sight is almost destroyed, and when it resists milder treatment, it may sometimes be cured by inoculation with the discharge of infantile purulent ophthalmia. The pus is inserted between the lids, and the artificial malady suffered to run its course unchecked, except by cleanliness and frequent bathing. When the discharge has ceased, the cornea will often clear in a surprising manner, and its abnormally vascular state protects it, to a very great degree, against the risk of sloughing. Still, this risk is by no means absent, and the treatment by inoculation should be regarded only as a last resource. A somewhat similar effect may be obtained by the inflammation which is excited by bathing the eyes with an infusion of the seeds of jequirity; but this method is scarcely less dangerous than that by inoculation.

(c) *Interstitial Keratitis*.—This is a chronic malady which is seen chiefly, or perhaps exclusively, in the subjects of inherited syphilis, who possess the peculiar teeth and *facies* which Mr. Hutchinson has shown to be characteristic of their inheritance. The disease was long described as a variety of 'strumous ophthalmia;' and, although the late Sir William Wilde pointed out how frequently it was associated with deafness, and also laid stress upon the value of perchloride of mercury in its treatment, its

syphilitic character seems to have been first suspected by Mr. Hutchinson, who, having once obtained the clue, followed it with characteristic diligence until he arrived at a conclusive demonstration of the accuracy of his suspicion.

**SYMPTOMS.**—Interstitial keratitis commences as a slight cloudiness of the central portion of the cornea, with some roughening or irregularity of the epithelium. It extends from the centre towards the margin, and is liable to be attended, in different cases and at different stages of its course, by variable degrees of ciliary and corneal congestion, and intolerance of light. If neglected, or if treated by irritants, it is liable to assume the characters of vascular keratitis, and also to extend to the iris, in such instances often doing irreparable mischief. It is most common during childhood, but its appearance may be delayed until adolescence, or even until adult age; and, in such cases, usually appears to be determined by some general or local exciting cause, such as bodily illness, or direct injury. It attacks both eyes, one commonly somewhat later than the other, and its course is often protracted over several months. When severe, it leaves some residual cloudiness of the cornea, and, even when mild, it is doubtful whether the cornea ever entirely regains the transparency of health. Still, when a case is seen and judiciously treated early, the prognosis may generally be a favourable one.

**TREATMENT.**—The treatment consists primarily in the avoidance of all irritants; the use of eserine and local sedatives, especially cocaine; and the administration of perchloride of mercury, or of iodide of potassium, with or without iron or cod-liver oil. When they are not contra-indicated by any special circumstances, the perchloride of mercury and the oil are the remedies on which the greatest reliance may be placed. The earliest indications of photophobia, showing, as they do, that light is acting as an irritant, should be met by confinement to an almost darkened room, and by frequent bathing of the closed lids with cold water; but in fine weather daily exercise should be taken in the open air, the eyes being covered for the time with a black silk bandage and compresses of carded wool, so as to exclude light entirely. As soon as photophobia subsides, these precautions may be left aside—the eserine and mercury being still continued, at least until the acute stage of the malady has entirely passed away. After this the absorption of residual opacity may be promoted by the application, once daily, of a morsel of an ointment containing a small quantity (about ten grains to an ounce) of the precipitated yellow oxide of mercury.

In cases which have been neglected, or aggravated by irritants in their early stages, and in which the phenomena of ordinary

vascular keratitis become grafted upon the interstitial, it is generally desirable to perform iridectomy with as little delay as possible.

3. *Arcus Senilis*.—The condition thus named (although the adjective is not always appropriate) is fully described elsewhere (see *ARCUS SENILIS*). It may be distinguished from the peripheral zones of opacity, which are sometimes left after the subsidence of certain forms of keratitis, by the circumstance that arcus never extends quite to the margin of the cornea, but is always surrounded by an annulus of transparent tissue.

Besides the foregoing, there are a few other forms of corneal disease, of comparatively rare occurrence, which it would be beyond the limits of these pages to describe, but which must be treated on the same general principles; and there is also the malformation known as 'conical cornea,' which falls wholly within the domain of surgery.

III. *Diseases of the Sclerotic*.—Diseases of the sclerotic, which were once regarded as a numerous and important group, have been reduced by recent investigations to comparatively insignificant proportions.

*Inflammation*.—*SYNON.*: Scleritis. — Excepting in a narrow annulus immediately around the cornea, the sclerotic is almost extra-vascular; and any real inflammation of its structure is almost confined to this particular region, where it seldom occurs excepting as a complication or as a part of some of the more severe forms of iritis or keratitis, especially when either of these affections extends to the ciliary body. In such cases we often see the sclerotic undergo inflammatory softening, as a result of which the ciliary region may be much altered in shape, yielding to the distension of the eyeball and to the traction of the recti muscles, and becoming distinctly elongated. Occasionally the sclerotic may be so much softened and thinned as to bulge into irregular prominences around the cornea, generally under the upper lid; and this condition is described as *sclerotic staphyloma*.

*TREATMENT*.—The inflammations thus arising call for no other treatment than that which is demanded by the more important inflammations of the cornea, the iris, or the ciliary body, with which they are associated; except that any evidence of yielding of the sclerotic would be a reason in itself for the performance of iridectomy, in order to preserve the shape of the eyeball by diminishing its tension. It was once believed that the sclerotic, in its character of a fibrous membrane, was especially prone to gouty or rheumatic inflammation; and it is perhaps true, though certainly not proven, that the tendency of iritis or of keratitis to spread to the anterior sclerotic zone is more marked in persons of gouty or rheumatic diathesis than in others. The possibility is at any rate sufficient to require, in all these cases, an in-

vestigation of the tendency to lithic acid formation, and the use of appropriate remedies when this tendency is discovered. But a large proportion of the examples of supposed gouty or rheumatic ophthalmia are nothing more than cases of the sub-acute or remittent form of glaucoma; and the pain associated with them is not really rheumatic but simply tensive. Vision has been irremediably lost, in hundreds of instances, because a belief in the rheumatic character of these affections has interfered with the timely performance of iridectomy; and the proposal to adopt the epithet 'rheumatic,' in any form of eye-disease, is one which should be scrutinised very closely before it is accepted as a guide to practice.

IV. *Diseases of the Iris*.—In so far as they come into the province of the physician, diseases of the iris are not numerous, and are almost limited to the varieties of inflammation of that membrane.

1. *Inflammation*.—*SYNON.*: Iritis.—Iritis may be classified, according to its actual or supposed causes, as rheumatic or syphilitic; or according to the nature of the morbid process, as *plastic*, *serous*, or *suppurative*. The former classification must often rest upon very slender grounds, and the latter has the great advantage of expressing facts rather than inferences.

(a) *Plastic Iritis*.—*SYMPTOMS*.—In plastic iritis the first symptom is usually some loss of the natural lustre of the surface of the iris, and of the clear definition of its fibres, together with some damping or alteration of its colour. These changes are probably always due to turbidity of the aqueous humour, and they may be imitated more or less closely by turbidity of the cornea, and especially by disturbance of its epithelium. In iritis, however, they are associated with a diminished range and quickness of pupillary variation under variations of light; and, in a short time, with the effusion of plastic lymph, by which the margin of the pupil becomes tied down, here and there, to the surface of the anterior capsule of the crystalline lens. At the same time there is usually some congestion of the conjunctiva, and of the zone of fine vessels immediately around the cornea in the sclerotic. There is frequently more or less pain, especially towards night, but this is a very uncertain symptom. In severe cases, and especially in such as are clearly syphilitic in their character, the quantity of effused lymph may be very considerable, so as quite to cover the pupil, while in mild cases it is only sufficient to fasten down the margin here and there. Iritis is sometimes a very insidious and seemingly slight affection, the real gravity and importance of which may be wholly overlooked by the patient.

*TREATMENT*.—The first principle of treatment is to prevent the formation of adhe-

sions, or to break them if they have been formed; and for this purpose our main reliance must be placed upon the instillation of atropine. The anatomical structure of the eye is such that a moderately contracted pupil is in contact with the lens-surface, while a fully dilated pupil is separated from it by a film of aqueous humour. Hence, as long as the pupil is contracted, any lymph which is effused tends to the immediate formation of adhesions; while, as soon as the pupil is dilated, the lymph diffuses itself harmlessly in the surrounding fluid, and no adhesions are produced. In ordinary circumstances, and in cases of only ordinary severity, the iritis then runs its course without inflicting any permanent injury, and vision is completely restored as soon as resolution has taken place. When, on the contrary, the pupil cannot be dilated, the lymph deposited in the area of the pupil forms an impediment to vision; and the adhesions themselves tend to render the iritis a recurrent affection, which is apt to return again and again until the eye is disorganised and destroyed.

The first principle of treatment is, therefore, to produce and maintain dilatation of the pupil; and for this purpose it is necessary to use some active mydriatic. The neutral sulphate of atropine is usually the most convenient agent for the purpose, and it should be used in a solution containing four grains to the ounce of distilled water. The pharmacopœial solution of atropine, which contains spirit, should be avoided on account of its irritating action. Of the pure watery solution, a drop should be carefully placed within the lids by a dropping-tube or quill, repeated in five minutes, and again in another five minutes, and this threefold application should be repeated three times a day. A mydriatic agent still more powerful and more rapid in its action than atropine has lately been introduced into practice in *duboisine*, the active principle of an Australian shrub, *Duboisia myoporoides*. Duboisine should be used in a watery solution, of the strength of four grains to the ounce; and it is said to have less tendency than atropine to produce local irritation. The hydrochlorate of cocaine may be combined in solution with either of the foregoing, and will not only assist their mydriatic action, but will also greatly promote the comfort of the patient. If the pupil can be fully dilated in twenty-four hours, no other treatment will be necessary than to maintain the dilatation by using the mydriatic less frequently, and to protect the eye from being injured by exertion, or by exposure to great variations of temperature or of light. If, on the contrary, after the use of atropine or duboisine for twenty-four hours, the pupil either remains contracted or dilates irregularly, showing that it is bound down here and there, it is necessary to have recourse to mercury without further delay, and

to use one of the preparations, such as blue pill or calomel, with which the effect of the medicine upon the system can be rapidly secured. There is never any occasion to carry the effect of the mercury farther than to the formation of a slight line upon the gums; and, in most cases, as soon as this line is perceptible, a notable amelioration of the eye-symptoms will be observed. It is desirable, however, that this degree of mercurial influence should be reached quickly, in order to cut short the disease as soon as possible; and when it has been reached, it will usually require to be maintained, for some days at least, by the administration of smaller and less frequent doses. In favourable cases, under the combined influence of mydriatics and mercury, the effused lymph will be absorbed, the adhesions broken through, and the eye restored to its original condition. In those of a less favourable character, the inflammation will, indeed, subside; but the adhesions will remain, and the pupil will be left permanently more or less crippled and distorted. Whether or not sight will be impaired will chiefly depend upon whether the effused lymph has formed a film or membrane across the pupillary opening. In the worst cases, notwithstanding treatment, the inflammation may extend to the ciliary body and choroid, and may produce functional destruction of the eye. This scarcely happens except when the iritis has been of great original severity, and when it has been neglected, or aggravated by irritants, in its early stages. Even the suspicion of iritis should absolutely preclude the use of the astringent applications on which we have mainly to rely in the treatment of the inflammations of the conjunctiva.

If an iritis is not seen until it has been three or four days in existence, so that the adhesions have had time to acquire a certain degree of firmness, it is not desirable to wait twenty-four hours before having recourse to mercury. The mineral should be given without further delay, so that it may be abandoned if atropine should dilate the pupil, and may be pushed if dilatation cannot be produced.

While the atropine, or atropine and mercury, are being employed, the remainder of the treatment must be governed by general considerations. Rest of the other eye must be strictly enforced; local depletion may be practised whenever the congestion is considerable in degree; and such a regimen and mode of life must be prescribed as the patient can bear. Unless there is photophobia, it is seldom or never necessary to exclude light altogether from the eye; and, when photophobia is present, it is better to apply a protective bandage than to keep the patient in a dark room. The latter practically excludes him from cheerful companionship, and leaves him to dwell upon his troubles in darkness and

solitude. Pain, if present notwithstanding the use of cocaine, should always be subdued, either by combining a sufficient dose of opium with the mercury, or by the subcutaneous injection of a solution of morphine. However the anodyne is administered, provision should be made for repeating it sufficiently often to produce and maintain the desired effect.

When recovery takes place leaving adhesions, these will, in the majority of cases, lead to a second attack of iritis, and this is almost always the predecessor of regular recurrence. In a few cases, however, the second attack does not follow; but, whenever it occurs, the tendency to future mischief should be stopped by surgical means, either by the detachment of the adhesions or by the performance of iridectomy.

(b) *Aplastic Iritis*.—The aplastic form of iritis differs from the plastic in the greater quantity and the more liquid condition of the effusion, which does not form adhesions, but distends the eyeball and compresses its contained structures. In a well-marked case the pupil is contracted and insensible to atropine (which in all probability is not absorbed); the aqueous humour is turbid; the iris is pushed back and its anterior surface appears concave; vision is greatly impaired; and the eyeball is perceptibly hardened to the touch.

TREATMENT.—Until the distension is relieved, no remedies will be effectual; and, when it is relieved, they generally cease to be needed. The treatment should be either by frequently repeated paracentesis of the anterior chamber or by a large iridectomy or sclerotomy, and the latter is generally to be preferred. Aplastic iritis occurs chiefly in persons of unhealthy or broken-down constitution; which may perhaps account for the unorganisable character of its products. As soon as the distension of the eyeball is relieved, the pupil is readily dilated, and the iritis soon subsides.

(c) *Suppurative Iritis*.—In a small number of cases iritis assumes from the first a suppurative character, and leads to the formation of pus in the anterior chamber.

TREATMENT.—Such a condition calls for atropine; for stimulating and tonic medicines rather than for mercury; and for the evacuation of the pus by paracentesis if it is considerable in quantity, or if its presence appears to be a source of increased irritation.

(d) *Serous Iritis*.—This term, which would perhaps be more appropriately used for the aplastic form of iritis, has been applied by continental writers to a form of disease which was described in old books as 'aquo-capsulitis,' and in which the posterior surface of the cornea participates with the iris in the inflammation.

ÆTIOLOGY AND SYMPTOMS.—Serous iritis usually first attracts attention by occasioning a slight dimness of vision. There will often

be no congestion, or scarcely any, but the movements of the pupil will be sluggish, and careful examination will discover one or more fine points of adhesion to the anterior capsule. But the most characteristic appearance of the disease is situate upon the lining membrane of the cornea, which will be found covered by a number of minute circular dots, ranged in a pyramidal outline. The base of the pyramid corresponds with the lower corneal margin; the apex encroaches more or less upon the pupillary region. As the disease progresses, the dotted pyramid increases in density and conspicuousness, and the pupillary adhesions increase in number and extent. The malady usually runs a very chronic course, and often leaves some permanent diminution of vision. In a certain proportion of cases it seems to be independent of any known cause; in others it follows some constitutional affection (the writer has seen a very severe case, in a young lady, consecutive to mumps); in others it is associated with constitutional syphilis. After injury to an eye, serous iritis sometimes appears in the other as the first stage of sympathetic ophthalmia.

TREATMENT.—This will include, in the first place, careful consideration of the state of the general health, and endeavours to correct whatever may be amiss. The eyes should be kept at rest, the pupils dilated but protected from bright light, and mercuric chloride, or iodide of potassium, or both, may be given in suitable doses, the efficacy of these remedies being manifest in the non-syphilitic as well as in the syphilitic subjects of the disease. Where the adhesions are numerous, iridectomy may be required to relieve dragging. The cases are usually chronic, and many months may elapse before the eyes are restored to usefulness.

2. *Inflammation of the ciliary body and choroid*.—SYNON.: Cyclitis; Irido-choroiditis. In some instances, which fortunately are not numerous, iritis is not confined to the membrane in which it originates, but spreads backwards to the ciliary body and the choroid. The most marked examples are those in which the original inflammation has been excited by morbid changes, resulting sometimes from disease, but more frequently from injury, in the opposite eye; and these cases are called *sympathetic ophthalmia*. They have been generally supposed to depend upon the propagation of peripheral irritation through a nervous centre, and to point very clearly to the presence of some central nerve-irritation, or functional failure, as the essential point of difference between the iritis which dies out as a localised affection, and that which spreads by continuity to the deeper parts of the eye. Recent observers have endeavoured to connect the phenomena with the presence and the migrations of microbes. However occasioned, the

issue of declared irido-cyclitis or irido-choroiditis is generally disastrous; for the inflammation is always of a plastic character, and the effused lymph is scarcely ever absorbed in time, or with sufficient completeness, to prevent its contraction from inflicting profound injury upon the visual apparatus. The perceptive layer of the retina is not only in contact with the choroid, but the rods and cones derive the materials of their nutrition from the chorio-capillaris; and hence, as regards these delicate structures, an inflammation of the choroid, upon which they are directly dependent, is of far greater importance than an inflammation of the retina itself, which may be limited to the connective tissue of the fibre layer, and may leave the precipient elements almost unaffected.

**SYMPTOMS.**—The first sign of the extension backwards of iritis is usually furnished by tenderness of the ciliary zone, so that this region feels acutely the slightest touch of a probe, or of the end of a rolled paper spill, which is a more delicate instrument for testing the sensibility of the ocular surface. At the same time there is always a greater degree of impairment of vision than the iritis alone will explain, together with increased general congestion of the eye, and, in many cases, with an appearance of visible vessels on the iris.

**TREATMENT.**—The treatment to be pursued does not differ materially from that which is required by the more severe forms of simple iritis; and consists of depletion from the temple, generally by means of Heurteloup's leech; the application of atropine; the administration of mercury, both internally and by inunction upon the brow; the control of pain; and the maintenance of strength. In sympathetic cases, the eye in which the mischief originated should be removed, even although the usefulness of this proceeding is somewhat doubtful when once the secondary affection is established. It is often necessary carefully to continue the use of mercury for a long period, generally in the form of small doses of the perchloride combined with iron; and it is chiefly when this has been done that some small vestige of vision is saved out of the wreck. The lens, in such cases, often becomes coated by lymph, and ultimately requires removal; and it may be necessary to perform iridectomy for closure of the pupil more than once, in consequence of the tendency of the artificial opening to be drawn together again by contraction. On account of the severity of sympathetic ophthalmia, and of its generally unfavourable termination, it is a rule of practice to anticipate its occurrence, and to remove a diseased or injured eye which is likely to produce it, before the mischief has been done.

**V. Diseases of the Crystalline Lens** are separately treated of under the article **CATARACT.**

**VI. Glaucoma.**—In its modern significance, this word is applied to denote all the conditions which are produced by a morbid increase of tension within the eye—that is to say, by an excess of its contained fluids; and the different forms of the affection are mainly due to differences in the rate at which the tension increases.

**SYMPTOMS.**—If the increase be rapid, the loss of sight will be rapidly produced, and will be associated with other changes occasioned by sudden interference with the circulation and innervation, and by sudden stretching of the ocular tunics. When, on the other hand, the increase of tension is very gradual, so that the eye has time to become accustomed to the new conditions as they are produced, the symptoms often present a deceptive resemblance to simple atrophy; and these cases were at one time described as 'atrophy with excavation of the optic nerve.' In some of the more acute forms of increased tension, the pupil presents a clouded aspect of a greenish colour; and it was to cases of this class that the word glaucoma (from *γλαυκός*, sea-green) was originally applied, at a time when the pathology of the condition was not understood. When this pathology was rendered clear, and when it became known that the glaucoma of the ancients was in all essential respects identical with cases in which the most manifest symptoms were of a different kind, the word was retained as a convenient general term, to express states of disease to which it had no longer any apparent reference; and hence we still speak of glaucoma, although, in the great majority of the cases in which we do so, the green aspect of the pupil is conspicuous by its absence.

The normal tension of the eye appears to be maintained, in the face of continued internal secretion of fluid, mainly by the transudation of this fluid, at the so-called 'filtration area,' in the angle formed between the cornea and the iris, into the plexus of veins contained in the canal of Schlemm. In order that this transudation may be free, it is necessary that the angle should be open, and the access of fluid to the filtration area unimpeded. In certain relations of the lens to the neighbouring structures, and, possibly, in consequence of increased bulk of the lens in advancing life, the periphery of the iris may be so pushed forward towards the cornea as to produce partial closure of the angle, and impeded access of fluid to the filtration area. Such conditions tend necessarily to increase; and they are liable to be increased still further by dilatation of the pupil, which implies thickening of the peripheral portion of the iris, and the occupancy, by this portion, of an abnormal amount of space. At a later period, as the tension continues to increase, the peripheral portion of the iris may even be applied to the margin of the cornea after the

fashion of a valve, and transudation may be completely arrested.

The estimation of increased tension by the fingers is a matter which requires the *tactus eruditus*, and is best accomplished by directing the patient to close the eyes gently, and to cast them downwards. The tips of the two forefingers should then be placed upon the upper lid immediately below the margin of the orbit, and, when one finger has fixed the eye by holding it gently back against the orbital contents as far as it will recede, the other estimates the degree in which it may be dimpled by slight pressure. This degree varies to some extent in different persons within physiological limits, but a morbid increase of tension can scarcely be missed if it is looked for. Moreover, the diagnosis of glaucoma does not rest upon increased tension alone, but upon the combination of increased tension with decreasing sight. The rate of slow increase which may simulate atrophy, or of rapid increase which may simulate inflammation, are matters of detail which should not, in either case, be suffered to obscure the true nature of the morbid process.

The symptoms of glaucoma depend upon the more or less gradual interference with the ocular circulation, and compression of the ocular nerves, by reason of increasing pressure within the eye. In consequence of the interference with the circulation, the blood enters and leaves the eyeball with difficulty. The first indication of this difficulty is afforded by the small veins which pierce the anterior portion of the sclerotic near the cornea, and course backwards beneath the conjunctiva towards the equator. These veins, which are invisible, or at least inconspicuous, in health, become distended and tortuous when the impediment to the venous outflow reaches a certain degree. At the same time, the ophthalmoscope will usually discover pulsation of the retinal vessels upon the optic disc, a pulsation first observed in the veins, and afterwards also in the arteries. It would seem that the contents of the veins are pushed back by the entering arterial current, and that the latter is only able to make good its way at the acme of the pulse-wave.

The symptoms which depend upon compression of the nerves will vary, of course, with the functions which these fulfil. Commencing with ordinary sensation, a moderate compression of the filaments derived from the fifth produces diminished sensibility of the corneal surface; while a pronounced increase of tension, especially if rapidly produced, occasions severe pain by the stretching of the ocular tunics. Proceeding to the motor filaments, compression of those derived from the third produces paresis of the ciliary muscle and of the sphincter pupillæ; the former evidenced by rapidly increasing impairment of accommodation, the latter by sluggishness and dilatation of the pupil, the

dilatation usually moderate in degree, but often causing the aperture to assume an elliptical outline, with its major axis horizontal. This pupillary dilatation is apt to be increased by the forward pressure of the crystalline lens; and, however produced, it tends, in the way already described, to occasion a still further increase of tension. The compression of the retina causes impairment of visual function, first manifested in the portions most remote from the central blood-supply; and hence taking the form of contraction of the field of vision, usually first displayed and most marked on the nasal side. Contemporaneously with this, central vision will be more or less reduced; and, at the same time, the increasing intra-ocular pressure will occasion yielding from within wherever such yielding is possible, and hence chiefly over the area of the optic disc, the structures composing which will be to some extent pushed back out of the eye, leading to the formation of a 'cup' or excavation, which is one of the most conspicuous of the ophthalmoscopic signs of heightened tension. Finally, the general nutrition of the eyeball will suffer, in a manner which is often first displayed by the surface epithelium of the cornea, which is apt to become disturbed, irregular, or, as it were, 'steamy,' presenting an aspect somewhat as if it had been breathed upon, and constituting a cause of further serious impairment of the already diminished vision.

Such being the chief symptoms, one or more of which will be recognisable by examination in glaucoma, those complained of by the patient are often exceedingly characteristic. Glaucomatous tension is apt to increase in waves, separated by intervals in which it may decrease, or in which the structures pressed upon acquire some tolerance of the new conditions. We therefore often hear complaints of sudden 'clouds' or 'obscurations' of vision, which pass away after a few minutes or a few hours, and return at uncertain intervals. The intervening recovery in such cases is seldom complete; each 'cloud' usually leaving the sight permanently worse than before. The depth of the cloud, the period of its duration, and the presence or absence of pain, are conditions which will be governed by the extent and the rapidity with which tension has been increased. When the cloud has passed away, we shall usually find diminution of central vision, contraction of the field, especially on the nasal side, and sluggishness of the rather large pupil. At the same time, it will often be found that the sensitiveness of the corneal surface is diminished; and the patient will often admit, on inquiry, that artificial lights of feeble intensity—candle flames, for example—are frequently surrounded by coloured halos. But the combinations and the order of occurrence of symptoms will differ widely in

different cases; and all that can be called essential, in order to constitute glaucoma, is a combination of impaired central vision and diminution of the field, with increased tension discoverable by the educated finger. When this combination is present, there should be no doubt as to the diagnosis, as there will certainly be no time to waste in inefficient treatment.

**TREATMENT.**—The treatment of glaucoma is entirely surgical, the affection being capable of arrest by iridectomy or sclerotomy in the great majority of cases. In the more acute forms, an operation, if performed sufficiently early, usually restores vision to its integrity; but the time during which this can be done is limited, and in the more chronic forms no operation will do more than preserve what amount of sight is still retained. Hence it is important that treatment should not be delayed by any error of diagnosis; and the points to which attention should chiefly be directed, in any case of impairment of vision in which the question may arise, are increasing hardness of the eyeball as determined by palpation, and gradual contraction of the field of vision. Whenever the eye is becoming harder, and the circumferential extent of vision is narrowing in, the case is one in which an operation should be accomplished with as little delay as possible. At the same time, until the operation can be performed, a four-grain solution of eserine should be applied every four hours; this drug having a marked effect in controlling and diminishing tension, and being apparently indebted for this power to its mechanical action upon the iris, which, by the contraction of the pupil, is thinned out and drawn away from the filtration area. It is hardly necessary to add that in any case of actual or threatened glaucoma, the use of a mydriatic would be highly dangerous. The more acute cases of glaucoma are attended by much pain from the distension of the ocular tunics, and often by congestion and inflammation, the results of this distension; and such cases were at one time described as ‘acute internal arthritic ophthalmia,’ or by some similar name. Vision has been irretrievably lost, in hundreds of cases, by endeavours to control this form of glaucoma by medical treatment, to the neglect of operation; and the erroneous practice has been kept alive by the circumstance that some of the cases will undergo partial and temporary amendment. In such circumstances, however, the vision never rises to the degree of acuteness which existed prior to the attack, and the amendment is never more than temporary. Another increase of tension soon occurs; and, without surgical aid, blindness sooner or later closes the scene.

**VII. Diseases of the Optic Nerve and Retina.**—As shown by the ophthalmoscope, these diseases cover a very wide

field of pathology. In order to understand them accurately, it is necessary to bear in mind the anatomy and relative arrangement of the affected tissues. The optic nerve in the orbit is invested by a double sheath, and the interval between the layers of this sheath, which is continuous with the sub-arachnoid space, terminates in a *cul-de-sac* towards the eyeball, the two layers becoming intimately united where they blend with the sclerotic. At this point, the opening in the sclerotic for the admission of the optic nerve is crossed by a film of perforated connective tissue, the *lamina cribrosa*, with which the sheaths of the nerve-fibres blend, or in which they are lost, so that the nerve anterior to the lamina consists of a bundle of unsheathed fibres, enveloping the central artery of the retina, which enters the eye with them, and the central vein of the retina, which passes out in the same position. On entering the eye the nerve-fibres bend round to form the anterior layer of the retina, which contains also the retinal blood-vessels almost to their ultimate divisions, together with some delicate connective tissue. The capillary circulation of the nerve itself is derived from the anterior cerebral artery, and is distinct, save for a very slight amount of anastomosis, from the capillary circulation of the sheath, which is fed by the arteries of the pia mater. It follows that hyperæmia of the sheath, or of the circle surrounding the nerve, may exist without hyperæmia of the proper nerve-tissue, and it has been supposed that fluid pressure in the intervaginal space may interpose an obstacle to the circulation in the vessels which pass through the sclerotic foramen, and may thus occasion dropsy of the termination of the nerve within the eye. Of the two foregoing conditions, either may undergo resolution harmlessly, or may produce such changes in or around the nerve as to occasion atrophy and loss of sight. Neither of them interferes with sight directly, because the circulation may be seriously disturbed by a degree of pressure which is insufficient to stop the conduction of impressions through the nerve-fibres. There is yet a third condition, properly called optic neuritis, or *neuritis descendens*, in which the capillary network of the nerve itself participates in changes propagated downwards, and in which impairment of vision holds a very early place among the symptoms.

1. *Perineuritis.*—The true perineuritis, in which the unchanged nerve-disc is surrounded by a zone of high vascularity, is only seen as a result of meningitis, which may be either tubercular or due to other causes. It was at one time hoped by Bouchut, by whom this especial phenomenon was first described, that perineuritis might serve as a diagnostic sign in cases of doubtful meningeal inflammation; but this expectation has not been realised. The perineuritis itself has only

been observed in cases the character of which was scarcely doubtful, and which, in most instances, have terminated fatally. Very probably, however, it would be found, if looked for, in those cases of exanthematous and other fevers which are attended by cerebral symptoms, followed after recovery by impairment of vision. In these we eventually find, as a rule, a partial nerve-atrophy, which does not lead on to complete blindness, but which does not appear to be susceptible of improvement; and this partial atrophy may no doubt be due to the pressure of perineural exudation during its contracting stage.

**TREATMENT.**—The treatment of perineuritis must generally be that of the affection in which it has its origin; but, in any fever in which this symptom had been detected, it would be a question whether mercury should not be cautiously administered for a considerable time after convalescence, in order to promote the absorption of any effused lymph by which the optic-nerve entrance might be constricted.

2. *Choked Disc.*—**SYNON.**: Dropsy of the Optic-Nerve Entrance.—This is a condition chiefly seen in connexion with intracranial tumours, whether syphilitic, tubercular, or of some other kind; and it almost invariably affects both eyes. It was formerly supposed to be due to the pressure of fluid driven down the intervaginal space around the nerve, and so constricting the latter at the terminal *cul-de-sac* as to impede the outflow of venous blood. This hypothesis is not now universally accepted, but the condition visible with the ophthalmoscope is one of distension and tortuosity of the retinal veins; arrest of the capillary circulation; impediment to the arterial inflow; dropsical effusion into the disc-tissues; and, sometimes, secondary inflammatory changes, such as effusion and cell-proliferation, comparable to the erythema which occurs in the integument of a dropsical limb. All these changes may exist without impairment of vision, because they neither affect the perceptive layer of the retina, nor arrest the conduction through the nerve-fibres. At a later period, however, when any plastic elements in the exudation begin to undergo contraction, the interference with the circulation becomes more complete, the nerve-fibres themselves become compressed, and then failure of sight commonly occurs. At the same period, the nerve passes into a state of atrophy from the interference with its circulation; and these cases of nerve-atrophy were once sources of great perplexity to surgeons, and were referred to alcohol, tobacco, and to other causes which probably had very little to do with them. In consequence of the sight not being affected during the preliminary stage, the occurrence of this stage was for a long time absolutely overlooked, and was only discovered when physicians began to use the ophthalmoscope, as

an ordinary instrument of diagnosis, in all cases of cerebral affection. It was then soon established, notably by the labours of Drs. Hughlings Jackson, Buzzard, and Clifford-Allbutt, that the atrophy had been preceded by swelling of the disc and by obstruction of its circulation; and it was shown before long that the cases of atrophy which had this history were recognisable, after the swelling had passed away, by the tortuosity of the retinal veins which was left behind, and by the way in which these veins were lifted into bold curves at the margin of the disc; this alteration of their original course having been due to the swelling, and remaining after the swelling had subsided. The contraction of the effusion, and the consequent atrophy of the nerve-fibres and closure of the capillary vessels, would be likely to occur earlier in some parts of the disc than in others; and hence, at the time when commencing failure of sight first induced the patient to seek advice on account of it, the disc was commonly seen to be invaded by sectors of whiteness, but to retain its vascularity, or perhaps more than its normal vascularity, in other parts; while, at the same time, the sight was first lost in those regions of the retina the fibres from which were first compressed. Hence it follows that a partial invasion of the disc by atrophic changes, and a partial invasion of the field of vision by blind portions, are among the earliest symptoms in cases of the class under consideration; and these symptoms were at one time referred rather to the nervous centres than to the retina or the disc itself, to changes in which they are now attributed. A not uncommon clinical history in such cases is that there has been constitutional syphilis, imperfectly treated, and ultimately producing headache or other cerebral symptoms, which have probably called for the administration of iodide of potassium and have been relieved by it. Some weeks afterwards there is for the first time a complaint of failing sight; and then the ophthalmoscope reveals that the discs are passing into atrophy, that the retinal veins are lifted into prominent curves at the disc-margins, and that their further course over the retina is generally serpentine. In many cases, the recovery of the patient, as far as general health is concerned, leaves the precise character of the intracranial mischief doubtful; but, in fatal cases, a tumour is the morbid condition most frequently discovered. **See OPHTHALMOSCOPE IN MEDICINE.**

When the merely passive dropsical effusion into the disc becomes complicated with inflammatory changes, as results of the disturbance of the tissues, the sight begins to fail before atrophic changes become manifest; and such cases are very difficult to distinguish from those in which there is *primary or descending neuritis*. The blood-supply of the optic nerve being derived from

the anterior cerebral artery, we may reasonably expect to find capillary engorgement of the nerve-substance of the disc in connexion with arterial hyperæmia of the brain; and this capillary engorgement may pass into inflammation, either of local origin or by transmission downwards from above. In any case, if the first changes in the disc are of the character of neuritis rather than of obstruction, we see capillary or arterial hyperæmia of the nerve-substance rather than venous congestion; and effusion of plastic material upon the disc itself, with comparatively little prominence or disc-swelling, and with comparatively little extension over the disc-margins upon the surrounding retina. At the same time, even in the early stage of the affection, we find great impairment of sight, the conducting power of the nerve-fibres being seriously injured. Such cases are frequently syphilitic, and, unless the absorption of the effusion should be quickly brought about by treatment, its contraction, like that of the effusion of obstruction, soon occasions atrophic changes. In these cases, however, the swelling having been absent or inconsiderable, the vessels do not show that elevation into bold curves at the disc-margin which has already been described; and the contraction being interstitial in the nerve-substance, and from the first affecting veins and arteries in an equal degree, the arterial inflow is diminished *pari passu* with the diminution of the vein-channels, and the latter vessels are seldom distended in such a manner as to render them distinctly varicose or tortuous. The ultimate result is a white disc, on which the arteries and veins are dwindled to threads, or at least greatly reduced from their normal calibre.

We may therefore have three conditions which in their typical forms are tolerably distinct, but which are prone to run into one another by almost imperceptible gradations, and which may all lead on to atrophy and complete blindness: namely, perineuritis, neuritis, and choked disc. The liability to the last-mentioned condition should be carefully remembered by physicians, and should lead to careful ophthalmoscopic examination in all cases of obstinate headache or other cerebral symptoms of obscure origin, more especially in a patient with a syphilitic history.

**TREATMENT.**—It is manifest that the best hope of preventing ultimate blindness in persons in whom choked disc has occurred will be secured by the administration of medicines calculated to assist the absorption of the effusion, and by continuing these medicines, with comparatively small reference to the general symptoms, until the discs have cleared. The writer has seen such clearing occur, without loss of sight, in circumstances which rendered it almost certain that neglect of the disc-effusions would have been followed by blindness at no distant

date. The same general rule will apply, of course, to the more directly inflammatory effusions of neuritis or perineuritis; and, when we find any one of the three conditions passing into atrophy, or when we find commencing atrophy in discs which show traces of past effusion, the principle of treatment is to endeavour to promote the absorption of any contracting material which may be the physical cause of the atrophy; and then, when this has been done, to seek to stimulate the nutrition of the nerve-fibres, and to assist them to recover from the shock which they have sustained. The mode of fulfilment of the first indication must depend mainly upon whether there is a history of syphilis, and, if so, upon the manner in which it has been treated. In the numerous cases in which a short course of mercury has been administered, enough to alleviate secondary symptoms, but wholly insufficient to eradicate the disease, it will generally be desirable to give iodide of potassium in full doses for a time, and to follow this by the prolonged administration of the perchloride of mercury, in the hope of really curing the patient. There are, in the writer's opinion, few things better worth remembering in therapeutics than that the iodide, immeasurably the most valuable drug which we possess as a remedy for a late syphilitic symptom, is, none the less, almost useless as a remedy for constitutional syphilis. It will remove the present symptom, speedily and often completely; but it can scarcely be said to have any tendency to prevent the recurrence of syphilitic symptoms at a future time, in the same or in some different form. For this purpose, the only trustworthy agent is mercury; and therefore, while the administration of the iodide for a sufficient time, and in sufficient quantities to test its power of doing good, will be enough in the cases in which syphilis is neither known nor suspected, the iodide must be followed by mercury whenever a syphilitic history of the affection is either clear or highly probable. The second indication, to stimulate the nutrition of the nerve-fibres, is usually best accomplished by strychnine, given at such intervals and in such doses as to produce evidence of its constitutional effect before its administration is abandoned. It may perhaps be most effectually given by hypodermic injection; but this is a point which must be settled in accordance with the circumstances of the case in each individual instance. During the last two or three years, in cases of great disc swelling with impairment or loss of sight, the writer has on four occasions incised the sheath of the optic nerve behind the eye, so as to give exit to any accumulation of fluid in the intervaginal space, or to release an inflamed nerve from the pressure of its unyielding covering. The results have been generally satisfactory; and in one of the patients, who had been totally

blind for several days, vision was completely restored.

3. *Toxic Amblyopia. Toxic Central Neuritis.*—Much attention has been bestowed of late years upon a form of gradual failure of vision which first affects chiefly the central portions of the retina, and which appears to depend upon a chronic neuritis attacking originally the portion of the optic nerve-trunk which contains the fibres destined for the region of the macula lutea. This affection has often been connected with the excessive use of the stronger forms of tobacco, as well as with the abuse of ardent spirits, or with both these conditions in combination. It is characterised by impairment of sight, and also of colour-sense, the impairment being at first limited to the central portion of the field, and the colour-sense often becoming changed before form-vision is very noticeably affected. It may extend, if neglected, over the whole of the nerve-fibres, and may ultimately lead to practical, but seldom to absolute, blindness, perception of light and of large objects being often retained. In order to test for central loss of colour-sense, it is necessary to be provided with small red and green objects which may be moved in succession over various parts of the field, and which, in typical cases, will appear much less bright in the centre than when they are held a little to one side of the direct line of vision.

TREATMENT.—This consists, firstly, in the abandonment of tobacco and alcohol; secondarily, in endeavours to improve the general health; thirdly, in the use of medicines, such as iodide of potassium and mercury, by which chronic inflammatory conditions are likely to be controlled; and, lastly, in stimulation of the weakened nerves by the careful employment of strychnine. A very careful prognosis should be given in the first instance; but, if improvement once commences, the case will usually terminate in recovery.

4. *Sclerosis of the Optic Nerve.*—Besides the consecutive forms of atrophy above enumerated, there is yet another of common occurrence, which is either a primary sclerosis of the optic nerve, or a sclerosis secondary to a similar affection of other parts of the nervous centres. This form of atrophy is not preceded by effusion, nor is it attended by any marked decrease in the calibre of the central vessels of the retina, even when the capillary circulation of the disc has almost wholly disappeared. It is often seen in connexion with disease of the spinal cord, as in locomotor ataxy; and also occurs in apparently healthy people, seemingly as a purely local affection.

Sclerosis is easily distinguished from the atrophies consecutive to effusion, whether active or passive, by the circumstance that the effusion, as it undergoes contraction, tends to render the nerve-tissues opaque as

well as to bleach them, and thus leaves a disc-surface of an almost ivory whiteness and of uniform colour. In sclerosis, on the other hand, the nerve-tissue disappears to a great extent, and reveals the mottled surface, often of a bluish-white tint, of the lamina cribrosa. When this is plainly seen, and when, at the same time, the vessels are neither much diminished in calibre nor altered in their normal curvatures, sclerosis may be assumed to exist; and this form of atrophy may also be distinguished from that which is produced by the most chronic forms of glaucoma, by the circumstance that in the latter the vessels bend into the excavated disc at its margin, while in the former they pass over the margin in straight lines or nearly so. Chronic glaucoma would also usually be distinguished by the character of the failure of sight, which would be marked by the ordinary contraction of the field of vision, even when central vision was only a little impaired; and also by the gradual hardening of the eyeball, which would be present in glaucoma and absent in nerve-sclerosis. Still it cannot be denied that this particular diagnosis is not without its difficulties, and that in certain cases it has given rise to differences of opinion between men of large experience on all sides of the question at issue.

The diagnosis is important as well as difficult, since the mischief of glaucoma may admit of arrest by iridectomy or sclerotomy; so that to mistake chronic glaucoma for atrophy, and to neglect operation, may be to condemn the patient to unnecessary blindness. The opposite error can at least do no harm; and therefore, whenever a doubt really exists upon the point, the most proper course is to give the benefit of that doubt to the patient, and to advise the performance of an operation which cannot injure, and which may relieve him. The atrophy of sclerosis scarcely admits of treatment, but it may perhaps sometimes be delayed, or even prevented from becoming complete, by the administration of full doses of strychnine and iron.

5. *Atrophy from other causes.*—Besides the foregoing forms of atrophy, there is a variety which appears to be associated with chronic lead-poisoning, and in which the discs may acquire a peculiar grey or bluish tint; and the optic nerves may also undergo secondary wasting in consequence of other conditions presently to be mentioned, such as obstruction of the central artery by an embolus, or the long continuance of pigmentary retinitis.

6. *Retinal Hæmorrhage.*—The chief disorders of the retinal circulation displayed by the ophthalmoscope are hæmorrhages, which may be attended by very different circumstances, and may present widely different characters.

a. *Single.*—When blood proceeds from one of the larger veins of the retina, which yield

a considerable quantity, and which are situated immediately underneath the limiting membrane, the hæmorrhage usually spreads out over the fundus as a red patch of uniform colour and aspect, and vision is suddenly, and sometimes almost totally, obscured. The writer has seen such bleeding occur from the yielding of a vessel during parturition; but this accident is extremely rare, and the large hæmorrhages in question are certainly more common in women at the period of cessation of the menstrual function than in any other circumstances. At this time, and when the health is not seriously affected, a favourable prognosis may be given with some confidence; for the blood will before long be absorbed, and restoration of vision, at least in a considerable degree, may be expected. The writer has once seen complete restoration to the normal standard, but this is an exceptional occurrence.

**TREATMENT.**—The only treatment necessary is to pay attention to the requirements of the general health; and to prescribe such diet, medicines, regimen, and habits as may tend to calm and equalise the circulation, and to prevent local congestions. The occurrence of sudden loss of sight in one eye will justify the suspicion of hæmorrhage; but the suspicion can only be converted into certainty by the ophthalmoscope.

*b. Multiple.*—A form of venous hæmorrhage which at first seems less formidable, because it is attended by a smaller degree of immediate interference with sight, but which calls for a less favourable prognosis, is that in which the hæmorrhages are multiple, often singly of small size, and scattered over the whole fundus of the eye. The appearances which they present differ, apparently in accordance with their precise position in the retina. If they proceed from vessels which are superficial, the blood is spread out, as in the last variety, in round or oval patches beneath the limiting membrane; but if the vessels lie a little deeper, and are fairly engaged in the fibre-layer, the blood will separate the fibres and find its way between them, forming flame-shaped or brush-like patches, which are often very numerous. Such multiple hæmorrhages are very slowly absorbed, and have a tendency to recur; so that they must always be regarded as placing the sight in serious jeopardy. They are often monocular, and they do not point to any definite disturbance of the general health. The only endeavour so to connect them with which the writer is acquainted was made by Mr. Hutchinson, who described some cases of flame-shaped hæmorrhage in persons all of whom he said were 'gouty'; but it will certainly be the experience of most practitioners that flame-shaped hæmorrhages occur in many patients who are not 'gouty' in the ordinary sense, and that they do not occur in vast numbers of those about the

reality of whose gout there can be no question. The presence of multiple hæmorrhages is sometimes attended by a considerable degree of irritation, or even inflammation, in the tissues among which the blood has been effused; and this condition, in which the retina between the blood-spots may become opalescent or turbid, has been described as a form of retinitis—*retinitis apoplectica*. The element of inflammation, in such instances, is probably merely a reaction consequent upon the injury inflicted upon the tissues, and it cannot be inferred that the bleeding is itself the result of any inflammatory process.

**TREATMENT.**—In this, as in the former variety, there is no special indication for treatment, which must be confined to the correction of any manifest disorder of the general health, followed, in most instances, by the administration of iodides or bromides, as medicines calculated to assist in the absorption and removal of the effused products. Any indication of a general hæmorrhagic tendency, or of a state allied to purpura or scurvy, would require, of course, full consideration and appropriate treatment. The extent of the ultimate injury to sight will usually depend upon the extent to which the perceptive elements of the retina have been compressed or disorganised, either by the bleeding itself, or by other changes consecutive to it.

*c. Arterial.*—Hæmorrhages which are distinctly arterial are not uncommon in the fundus of the eye, and can generally be distinguished without difficulty from the venous variety, not only by the colour of the effused blood, but also by the situation of the blood-patch, and by its manifest relations to a small arterial branch, which may often be seen to have dwindled or closed beyond the point at which it has given way. Arterial hæmorrhages are mostly multiple, but of small individual extent; and, when not in the immediate neighbourhood of the optic disc, are most commonly seen near the outer limits of the ophthalmoscopic field of view. They are generally attended by sufficient impairment of vision to occasion complaint, and thus to lead to their detection; but they seldom occasion blindness. They call for an examination of the urine for albumen, and, failing any evidence of renal mischief, they are chiefly important as indications of a weakened and brittle state of the arterioles, likely to lead to intracranial hæmorrhage.

**TREATMENT.**—Arterial hæmorrhages into the retina point to the necessity of diminishing, as much as may be possible, the strain upon the arterial coats, by such means as the avoidance of muscular exertion or mental emotion, and by seeking to diminish the quantity of the circulating blood by a diminution in the quantity of fluid ingested. Even when all precautions have been taken, arterial

retinal hæmorrhages are common forerunners of apoplexy.

7. *Embolism of the Central Artery of the Retina.*—Embolism of the central artery of the retina, or of one of its branches, is a condition of not infrequent occurrence.

SYMPTOMS.—When sudden blindness of one eye occurs in a person who is the subject of valvular disease of the heart, the diagnosis can scarcely be doubtful; but the ophthalmoscopic appearances will suffice to remove doubt if it should exist. The immediate effect of the sudden arrest of the arterial circulation of the retina is to render that membrane opaque and of a milky whiteness, except over the macula lutea, where the absence of connective tissue prevents any such change from being produced. Here, and here only, the original transparency is retained, and the colour of the choroid is still visible, with the result that the macula appears as a cherry-red spot in the midst of a white surface. When not concealed by the opacity, the larger veins of the retina are diminished in calibre and contents, and their blood is sometimes broken up into detached portions separated by interspaces. The arteries are empty, and are either invisible or traceable as white lines of fibrous tissue in the general milkiness of the fundus. The disc is usually bleached, but it will sometimes happen that its condition may be temporarily obscured by arterial hæmorrhage, occurring from some twig given off just below the seat of the embolus, and entering the eye independently.

The driving home of the embolus will throw upon such a twig the whole force of the circulatory *vis a tergo*, and may thus rupture it—an occurrence of which the writer has seen several examples. The blood so effused is usually absorbed in a very few days, revealing the white disc and the collapsed arteries, and removing any uncertainty which might have existed with regard to the diagnosis. The opalescence of the retina also disappears before long, and then only the secondary nerve-atrophy and the disappearance of the arteries remain to disclose the nature of the original affection. Embolism seems to be a perfectly hopeless condition, because there is no anastomosis between the retinal and other vessels of a sufficient extent to maintain a collateral circulation. The writer has met with one instance in which only a sector of the field was affected, and with one in which embolism of a very small branch produced loss of sight over all the peripheral parts of the field, leaving central vision almost intact; but such cases are among the curiosities of ophthalmology, and complete and permanent loss of sight of the affected eye is the result which must always be anticipated.

8. *Retinitis.*—Retinitis is commonly described as occurring in three chief forms—the *albuminuric*, the *syphilitic*, and the *pig-*

*mentary*; but the writer is inclined to believe that only the last of these three is a genuine retinitis, and that in the others the inflammation, if it should exist, is merely a secondary consequence of the irritation produced by the presence of adventitious deposits.

a. *Albuminuric Retinitis.*—In the so-called albuminuric retinitis, the sequence of events appears to lend support to the contention of the late Sir William Gull and Dr. Sutton, to the effect that the renal disease is not an original affection, but only a result of morbid or degenerative changes which are common to the whole of the small arteries of the body.

SYMPTOMS.—In many cases of albuminuria, the sight is not affected from first to last, and the retina remain healthy. In some, the retinal changes precede the appearance of albumen in the urine; and, in the majority, the renal and retinal changes are coincident. The retinal changes are of two kinds: namely, arterial hæmorrhages, occurring in the fibre-layer, so that the blood-patches assume a fibrillated aspect with brush-like terminations; and the formation of white patches, either of cholesterine deposit or of fatty degeneration, or of both combined, scattered irregularly over the fundus, but often grouped into a stellate figure around the macula lutea, and into an irregular ring around the disc. To these appearances are added, in some cases, those of swelling of the disc-margins with effusion into the retinal fibre-layer; and, when the last-named appearances are presented, there is always a far greater deterioration of sight than when they are absent. It is a matter of daily occurrence that the existence of renal disease is not suspected until impairment of sight leads to an ophthalmoscopic examination, and this to the discovery of the retinal changes; and, in every hospital, cases which apply for relief to the ophthalmic department are constantly, on this ground, transferred to the physician.

TREATMENT.—The treatment of the renal maladies which produce albuminuria is in no way modified on account of the presence of a retinal complication; and the unfavourable prognosis which must generally be given as regards life throws into comparative insignificance the gradual failure of vision, which seldom proceeds to complete blindness.

b. *Syphilitic Retinitis.*—This is usually an incident of the most advanced stages of the disease, and is most frequently seen in persons who have been inadequately treated during the primary stage, but who have for some months or even for a year or two been free from symptoms.

SYMPTOMS.—Dimness of sight is then complained of, and the retina is found to present scattered patches of very irregular outline, and of a filmy whitish aspect. Such patches may be more or less obscured by slight

general turbidity of the retina itself, or of the vitreous body in its immediate vicinity, the latter condition being of itself almost conclusive of the nature of the malady.

**TREATMENT.**—The treatment must be greatly governed by the past history of the case, but may in most instances turn upon the use of iodide of potassium for the relief of the retinal troubles, followed by a sufficient course of mercury for the eradication of the syphilitic taint.

*c. Pigmentary Retinitis.*—This appears to be a true inflammation of the retina, differing from the foregoing affections in that it attacks the percipient elements, instead of the fibre-layer or the connective tissue of the membrane.

**ETIOLOGY.**—The subjects of pigmentary retinitis are of all ages, from nine or ten years to seventy; and, in some instances, the duration of the disease has been as much as twenty years, from the first appearance of the symptoms to their ultimate termination in blindness. As a rule, however, the patients are young adults, or persons not past middle age.

It is a remarkable feature of pigmentary retinitis that it almost invariably attacks more than one member of a family; and it has been said to be especially frequent in the offspring of marriages of consanguinity, but this statement is not borne out by English experience. During the last twenty years the writer has only met with one family in whom the malady had this history.

**ANATOMICAL CHARACTERS.**—From the extreme chronicity of its course, from its obstinacy, and from its peculiar anatomical distribution, pigmentary retinitis should probably be regarded, together with some forms of choroiditis, as having its analogies among some of the chronic diseases of the skin. It usually appears to commence in a narrow annulus near the equator of the eyeball, and gradually spreads inwards towards the optic disc; the tissues affected are the percipient and pigmentary layers of the retina and the subjacent chorio-capillaris, which slowly become disorganised and matted together in one common degeneration. Coincidentally with the progress of the disease, pigment is deposited in the parts affected, and in the retina superficial to them, in the form of irregular lines and striations, and especially along the course of the main arterial branches. As the annulus of disease gradually closes in upon the macula, the optic disc undergoes atrophy of a kind which gives it a peculiar tint of whiteness, very readily recognisable when it has once been noticed, and the central vessels, both veins and arteries, dwindle in size.

**SYMPTOMS.**—The subjective symptoms are as characteristic as the ophthalmoscopic appearances. Over the region actually invaded, the percipient elements of the retina are de-

stroyed, and the power to receive visual impressions is lost. The fibre-layer not being implicated, the conduction of impressions from parts of the retina more peripheral than the disease may remain unaffected; and hence we may have a blind zone surrounding the centre of the field of vision, and surrounded itself by a zone still more external, in which dim vision is preserved. But the salient symptoms are two—the gradual contraction of the field of vision due to the progressive encroachments of the disease; and night-blindness, due to the nerve-atrophy, which interferes with the conduction or perception of any but strong impressions. When these symptoms co-exist; when the field of vision is small and becoming gradually smaller; and when the patient, who can still see fairly in the daytime, can scarcely find his way about as dusk begins to fall, we may predict the ophthalmoscopic appearances with a very near approach to certainty. The optic disc will be unnaturally pale, and the fundus overstrewn, towards the periphery, with irregular black lines and stripes, of which it is quite possible that none may be visible within that portion of the field of the ophthalmoscope which includes the disc.

**DIAGNOSIS.**—Pigmentary retinitis may possibly be mistaken for the most chronic form of glaucoma, on account of the contraction of the field of vision; but it may be distinguished by the absence of high tension, by the night-blindness, and by the pigmentation of the retina. It may also be mistaken for the atrophy of sclerosis, but only if the ophthalmoscopic examination is limited to the nerve-disc, to the exclusion of the surrounding parts of the fundus.

**TREATMENT.**—In the treatment of a disease so essentially chronic, it is difficult to arrive at any trustworthy evidence concerning the efficacy of a remedy, but the prolonged administration of iron, rather as a food than as a medicine, is at least of a certain degree of utility in arresting the progress of the malady. The preparation employed is probably not material, and some may be found to suit particular persons better than others; but the writer is accustomed to begin with the tincture of the perchloride, in doses of five minims, well diluted and given three times a day as part of a meal.

*9. Detachment of the Retina.*—**SYNON.:** Subretinal Dropsy.—This is a condition the causes of which have never been satisfactorily explained, but it has been attributed to the contraction of inflammatory effusions in the vitreous body, which have been supposed thus to exert traction on the retina.

The first symptom which attracts the attention of the patient is the loss of part, usually either the upper or the lower part, of the field of vision; and it is manifest that loss of the upper part of the field means detachment of the lower part of the retina, and

*vice versâ*. Detachment is sometimes produced by a blow or injury, but more frequently it occurs without any assignable cause, either local or constitutional. One or both eyes may be affected.

The diagnosis of the disease is rendered easy by the ophthalmoscope, which exhibits the detached portion as a sort of floating prominence, projecting into the interior of the eyeball, generally bluish-white in colour, and crossed by the retinal blood-vessels.

The prognosis is very unfavourable in the majority of instances, and treatment is seldom effectual.

**TREATMENT.**—Cases have been recorded in which disappearance of the sub-retinal fluid, and restoration of vision, have followed prolonged confinement in the supine posture; and the occurrence of improvement after spontaneous rupture of the detached portion suggested to von Graefe the advisability of producing such a rupture by artificial means. Various operations have been undertaken for this purpose, and also for the evacuation of the sub-retinal fluid through a puncture in the outer tunics of the eye, and have in a few instances been partially successful; but the evidence in their favour is at present very feeble, and hardly establishes more than that attempts of such a nature may be made, if it is certain that the sight will be irretrievably lost in the absence of interference. The writer once obtained an excellent result by puncturing a detached retina in an eye from which cataract had been removed eleven years before; but the puncture was greatly facilitated, and its risks were in a corresponding degree diminished, by the absence of the lens. Two or three years later, however, the detachment returned and became complete. The tendency of detachment, especially in the upper portion of the retina, is to increase until the whole membrane is elevated from the choroid, and vision is entirely destroyed. It must be borne in mind that detachment may be simulated, or may even be caused, by the growth of intra-ocular tumours, sarcomatous or gliomatous, which may demand the early removal of the eyeball. Such cases would usually be distinguished from simple detachment by the increased hardness of the eyeball, which the morbid growth almost necessarily occasions, and which would be the more significant inasmuch as detachment alone is usually accompanied by diminished tension. It must nevertheless be remembered that normal tension does not absolutely preclude the presence of tumour; as there are well-authenticated cases in which the absorption of the ocular fluids has, for a time, precisely kept pace with the increase of the growth.

10. *Glioma*.—This name has been given by Virchow to a malignant growth which has its origin in the neuroglia, or connective tissue of the nervous system, and which was formerly

described as encephaloid cancer. When originating in the retina, it early produces loss of sight, and presently shows through the pupil as a substance of a primrose-yellow colour, by which the still transparent lens is pressed forward towards the cornea. It is chiefly a disease of childhood, and has been seen by the writer as early as the fifth week of infant life. It is liable, by superficial observers, to be mistaken for congenital or infantile cataract, an error which must be carefully guarded against, because the early and entire removal of the eye, together with as much of the optic nerve as can be reached, furnishes the only hope of preserving the life of the patient. When the operation is performed sufficiently early, it has in a few instances been completely successful, cases having been recorded in which no recurrence of cancer has happened after the lapse of years. In the great majority, however, recurrence and death have terminated the history.

11. *Sarcoma*.—This differs from glioma in having its origin in the choroid, and in being of a darker colour, and sometimes pigmented or melanotic. It is at least equally malignant, produces similar symptoms, and requires the same treatment.

**VIII. Diseases of the Choroid.**—Diseases of the choroid, recognisable by the ophthalmoscope, are almost limited to certain chronic forms of inflammation and of atrophy; for, in any acute choroiditis, there is always too much turbidity of the vitreous body to allow the state of the membrane to be seen.

*Chronic Choroiditis.*—The chronic forms of choroiditis are remarkable for leading to an undue formation, or to a great displacement, of the choroidal pigment; and to the ultimate complete wasting and disappearance of the portions of the choroid which are affected, so that over these portions there will ultimately be no choroid visible, and the ordinary red colour of the fundus will be replaced by the ivory whiteness of the inner surface of the sclerotic.

Chronic choroiditis may be divided into two chief varieties—the *disseminated* and the *diffused*. The *disseminated* occurs chiefly in children, and chiefly, perhaps exclusively, in those who are the subjects of inherited syphilis. It is seldom seen until its period of activity is passed. A child is brought on account of defective vision, which has probably existed from birth or from a time but little subsequent to it; and the ophthalmoscope displays a number of small white spots, with black borders, scattered irregularly over the fundus of the eye. The white spots are patches of choroidal atrophy, and the black borders are rings of increased pigment-formation, by which the spots of inflammation, which must have been comparable to little pimples, have been surrounded.

**TREATMENT.**—Such cases admit of no treat-

ment, except in the rare instances in which some active mischief may be detected, in the shape of small patches or spots in which effusion has not yet passed into atrophy, and in which such an antisyphilitic treatment should be employed as the state of the patient may otherwise permit or indicate.

*Diffused choroiditis* is more frequently an affection of adult age; and, although very frequently syphilitic, is not invariably so. It differs from the foregoing chiefly in the absence of any defined shape or precise limitation of the parts affected. In the early stages the choroid is seen to be troubled by congestion or effusion, and these conditions pass gradually into abnormal pigmentation and atrophy. The course of the disease may be very chronic and irregular, and different stages of it may be seen at the same time in different parts of the same eye.

The prognosis may in general be moderately favourable; for, although the choroiditis destroys the portion of retina immediately in front of it, its extension is very capricious, and it may often be arrested in time to leave large portions of the eye, and especially the central portions, unharmed. When it occurs in the vicinity of the macula lutea, so as to imperil central vision, it is much more formidable than when confined to the more peripheral parts of the choroidal membrane.

**TREATMENT.**—Whenever there is a history of syphilis, this must be taken as the clue to treatment; and, if no syphilis can be discovered, the chief reliance must be placed upon rest of the eyes, occasional depletion from the temples by Heurteloup's leech, counter-irritation by blisters or setons, and such internal medication as the general state of the patient may suggest.

### IX. Diseases of the Vitreous Body.

Diseases of the vitreous body are as yet very imperfectly understood, and we know little more concerning them than that this substance is liable to become turbid in certain forms of acute general inflammation of the eye; and that it is sometimes rendered turbid, without inflammation, by the presence of floating films which may be readily seen by the ophthalmoscope, and which may be so numerous as to form a serious impediment to vision.

1. *Turbidity.*—Turbidity of the vitreous is very common in syphilitic cases; but the films referred to are seen when no syphilis can be suspected. Their number, and their free movements, show that the vitreous must in great measure have lost its natural semi-solid consistence, and have become fluid; but little or nothing is known of their actual pathology.

**TREATMENT.**—The most effectual treatment for flocculi in the vitreous is usually diaphoresis by the subcutaneous injection of from two to four minims of a 10 per cent. solution of hydrochlorate of pilocarpine, which may

be repeated on alternate days. Local counter-irritation with iodine may also be practised; and iodide of potassium may be given internally in such doses as circumstances will allow.

2. *Musæ Volitantes.*—A phenomenon referred to the vitreous body is the appearance of the moving particles, or strings of beaded filaments, which are commonly called *musæ volitantes*. True musæ are known by the negative character that the particles which produce them cannot be seen by the ophthalmoscope; and by the positive character that they never so intervene between the eye and an object, however small, as to exclude the latter from view. They are seen most readily against a white field, as a white wall or a white cloud, or in the illuminated field of a microscope when there is no object in view; and they float about with uncertain movements, but always a little out of the direct line of sight. They are occasioned by the filamentous framework of the vitreous body, and by the cell-nuclei or other irregularities upon the filaments. These bodies, without being opaque, yet differ in the precise degree of their transparency from the fluid which surrounds them; and hence they cast upon the retina shadows, which are then mentally projected outwards into space as floating objects. The projected shadows appear, of course, enormously larger than the microscopic specks which produce them, and the latter are wholly unimportant and of no morbid signification. Musæ may be discovered by any person by the simple expedient of looking through a very fine perforation in a metal disc at a bright surface; and they are more conspicuous to some persons than to others, on account of the varying differences which may exist in different eyes or in the same eyes at different times or under different conditions, between the index of refraction of the filaments and nuclei and that of the surrounding fluid. Moreover, by the operation of an obvious physical law, the more distant the particle from the retina, the larger will be its shadow upon that membrane, and the larger and more conspicuous will it appear. For this reason, and on account of the elongation of the myopic eyeball, musæ are usually more complained of by the short-sighted than by others. They are often sources of great uneasiness to patients; but, when once their true character is known, they may be entirely disregarded as harmless appearances, the natural results of physiological structure. It is often important that the physician should be able to make their nature understood, in order that he may dissipate, once for all, the unfounded apprehensions which may be occasioned by their presence.

X. *Diseases of the Eyelids.*—The external surfaces of the eyelids, as parts of the common integument, are liable to all its

diseases, and may thus participate in erysipelatous inflammation, in eruptions, and in the results of injury, besides becoming the seats of *nævi*, moles, warts, and other growths. Among the diseases special to the formation of the eyelids, the most important are the variations of shape to which they are subject, generally from the contraction of inflammatory exudations, but sometimes from perverted muscular action; the cystic tumours which are produced by obstruction of the orifices of Meibomian glands; the inflammation of the follicles of the eyelashes, or blepharitis; spasmodic closure, from abnormal muscular contraction; and either patency or passive closure, from paralysis. Many of these affections are distinctly surgical, and others are only parts or symptoms of more general disorders.

1. *Blepharitis*.—Blepharitis, or inflammation of the follicles of the eyelashes, has received a great variety of names from different writers, and is frequently known as *tinea tarsi*, or, in its more advanced stage, as *lippitudo*. The disease consists essentially of an inflammation of the lining membrane of a hair-follicle from which an eyelash springs.

**SYMPTOMS.**—The first manifest symptoms are a small swelling close to the edge of the eyelid, generally of the upper lid; and the formation of a crust around the bases of the cilia which proceed from the swollen part. The swelling does not extend farther up the lid than to the breadth of about a line, but it soon spreads along the border until the whole length is involved, and it usually spreads also to the lower lid, manifestly in consequence of the contagious character of the discharge. If the crust is removed, and if the part from which it springs is magnified and carefully examined, it will be seen that the mouths of the follicles are somewhat open, no longer fitting closely to the issuing hairs; and, in a few moments, a clear fluid will be seen to exude, and speedily to dry into a crust or film, which covers the opening as if with a varnish. Many of the hairs in the affected follicles are loosened, and fall readily, or may be removed painlessly by slight traction. If the case is neglected, the follicles are before long destroyed as hair-bearing organs, so that the lost cilia are no longer reproduced; and, at the same time, the exudation which constitutes the subcutaneous swelling of the lid-margin begins to undergo contraction, and in this way gradually everts the cartilage of the lid. The edges of the lids become red, swollen, and unsightly; the lachrymal puncta are displaced outwards in such a manner that they can no longer take up the tears; the eyes have lost the protection of the lashes, and are exposed to numerous sources of irritation from atmospheric particles and other causes, so as to be especially prone to conjunctival

and corneal inflammations; and these results are almost incurable. It is therefore very important that blepharitis should be effectually treated in its early stages, when, if only due care be taken in the selection and use of remedies, it is an exceedingly trivial affection.

**TREATMENT.**—The most essential part of the treatment is to remember that the secretion which forms the crust is of such a nature that it is not very easy of removal, and that, while it remains *in situ*, no remedies, however judiciously chosen, can obtain access to the parts really affected by the disease. The crust is composed partly of the already mentioned secretion from the inflamed surfaces, partly of the greasy secretion of the Meibomian glands; and it is the admixture of the latter which renders the crusts difficult of removal by water alone. A solution of bicarbonate of sodium, of the strength of five grains to the ounce of warm water, will remove them readily; and this solution should be applied in such a manner as to soak into the crusts and loosen them thoroughly before any attempt is made to detach them. As soon as they are detached, the surface beneath should be gently dried with a morsel of absorbent rag, and then an astringent should be applied immediately, so that it may find its way down into the depths of the hair-follicles, and may thus reach the seat of the malady. The best astringent is generally the ointment of the precipitated yellow oxide of mercury, or Pagenstecher's ointment, already recommended for the cure of ulcers of the cornea; and this may be applied to the affected part by the tip of a finger. If amendment does not speedily follow, it may be suspected that the crusts have been imperfectly removed, or the applications imperfectly made, and it will be well for the practitioner personally to superintend the process. When this has been done, if the affection continues obstinate, some other astringent should be tried, and the nitrate of silver is among the best for this purpose. Amendment of the lid-margin may generally be quickly produced; but the disease will for a long time lurk in the depths of the follicles, and the treatment must be continued until all subcutaneous swelling has disappeared from the lid-margins. Unless this be done, speedy relapse is inevitable, the inflammation soon creeping out of the follicles again and recovering the ground of which it had been deprived. Such a result is constantly seen in hospital practice, in spite of all efforts to guard against it, and, in prescribing for blepharitis, it is always desirable to warn parents of the perseverance which will be required, and of the great importance of obtaining a radical cure. There can be no doubt that blepharitis is exceedingly contagious through the medium of its secretion, conveyed upon sponges, towels, or fingers,

and this should be fully recognised whenever it attacks children who are attending a school. The name 'tinea tarsi' may perhaps be taken as the expression of a belief that the disease is allied to tinea tonsurans, and that it is produced by the growth of a parasitic fungus. The writer does not at present see any sufficient ground for the adoption of this opinion.

2. *Entropium and Ectropium*.—Incurvation and excurvation of the eyelids may be looked upon as purely surgical maladies. The former exposes the eyes to injury from the irritation of inturned eyelashes (*trichiasis*); the latter from foreign bodies of various kinds.

TREATMENT.—The remedy for both, when any is practicable, must usually be sought in a surgical operation. An exception depends upon the fact that ectropium is sometimes produced by paralysis of the facial nerve, which renders the orbicularis muscle flaccid and powerless, and permits the lower lid to fall downwards under the influence of gravity. The cure of the general nerve-affection may restore the power of the muscle, and may in time lead to complete recovery of the natural position of the lid. In such cases, even if electricity does not form part of the general treatment of the paralysis, it may often be applied with benefit to the orbicularis.

3. *Blepharospasmus*.—This term is generally employed to denote an intermittent closure of the eyelids by an involuntary action of the orbicularis, in response to some concealed source of irritation; and is thus broadly distinguished from the spasm which accompanies photophobia.

SYMPTOMS.—The spasm is most liable to occur in circumstances of mental excitement. Thus, in one of the writer's patients, who was a skilful cook, the eyes were apt to close, and to remain closed for some minutes, at the critical period of an important dish. Another patient was a schoolmistress, and the spasm would interrupt the progress of a lesson to a class, being doubtless to some extent excited by the dread of its occurrence. In a third case, the patient being a gentleman habituated to riding and driving, the spasm would be excited by physical irritants, such as wind or dust, and, almost certainly, by circumstances which required the eyes to be wide open as a condition of safe guidance. The motor nerves appear, as a rule, to be merely the passive conductors of a reflected impulse, and the trouble seems usually to be dependent upon a morbid condition of the fifth, or upon a source of irritation in some peripheral part from which a twig of the fifth passes to the centre.

TREATMENT.—In the treatment of such cases, it is sometimes possible to find the twig which conveys the impression; that is to say, to discover a point where pressure, sufficiently firm to arrest conduction, will at once relax the spasm. Such points should

be looked for at the supra-orbital notch, over the malar bone, and in any other situation suggested by special circumstances; and, if a point at which pressure will arrest the spasm is discovered, we learn at once by what branch of the fifth, and therefore approximately from what region, the irritation is conveyed, and where its source is to be sought for. If nothing can be discovered by careful examination, decayed teeth, accumulations of cerumen in the ears, and conjunctival granulations are possible conditions which should be looked for, and which should receive attention if they are found. When all other treatment has failed, the spasm has sometimes been stopped by subcutaneous section of a sensory nerve; and this may always be practised hopefully if the spasm can be arrested by pressure on some definite spot, which must then serve for the guidance of the knife. If no such spot can be found, section of the supra-orbital nerve, and next of the subcutaneous malar, may be attempted; since neither of these is sufficiently important for its temporary disablement to be set against even the possibility of relief from a very distressing affection. In some cases, however, it would appear that the mischief must be central, and that no section of an afferent nerve can be useful. The division of the motor nerves of the orbicular muscles, if it could be successfully accomplished, would produce a paralysis even more injurious than the spasm; and the cases in which the latter is due to central irritation or other trouble, unless they can be relieved by medicine, and by the rectification of whatever may be manifestly wrong in the condition of the patient, offer very small hope of improvement. See FACIAL SPASM.

4. *Ptosis*.—Ptosis is a condition of permanent passive closure of an upper eyelid as a consequence of paralysis of its levator muscle, or it may happen in consequence of this muscle having been torn from its attachment to the tarsal cartilage, so that it can no longer modify the position of the lid.

SYMPTOMS.—Paralytic ptosis may be either partial or complete, according to the degree of the nerve-affection; and as the levator palpebræ is supplied by the third nerve, which supplies also the superior, the internal, and the inferior rectus, as well as the inferior oblique, the sphincter pupillæ, and the ciliary muscle, ptosis is usually accompanied by paralysis of one or more of these muscles. When they are all affected, the eyeball is turned outwards by the action of the external rectus, and is immovable in other directions excepting feebly by the superior oblique. The pupil is dilated, and the power of adjusting the eye for near vision is impaired or lost, although, when the lid is raised, near objects can still be distinctly seen by the aid of a convex lens. When all the muscles supplied by the third nerve are affected, the inference

is that the cause of paralysis is acting upon the common trunk of the nerve; and such a cause is not infrequently the presence of periosteal swelling at the sphenoidal fissure. If only some of the muscles are affected, the inference is that the cause of the paralysis is either limited to the central nuclei of origin of certain filaments, or else that it is situated anteriorly to the division of the main trunk into the branches which proceed to different parts; and it is conceivable that the limitations of the paralysis may point, with tolerable certainty, to the precise locality of the disorder.

The causes of ptosis, as of other paralytic affections of single cranial nerves, apart from injuries and the pressure of morbid growths, may almost be reduced to syphilis and to impaired nutrition of the centres, the latter usually connected with hard mental work and worry. In every case, evidence of syphilis should be carefully sought for; and, if found, should determine the nature of the treatment, as it will also of the prognosis, which, in such instances, may be generally favourable. In cases of the second class, where there is no evidence of syphilis, and where the symptoms point to general impairment of nervous energy, the administration of iodide of potassium, in combination with tonics, will sometimes be useful; but the

main reliance must be placed upon rest, good living, and external surroundings favourable to the restoration of health.

5. *Diplopia*.—Double vision, although it has no proper relation to the subjects treated of in the present section, is yet so far allied to ptosis that, when occurring suddenly, it is almost always an effect of paralysis or of paresis either of the sixth nerve of one eye, supplying its external rectus, or of the branch of the third which supplies its internal rectus. In the former case the affected eye will deviate inwards, and will have limited range of movement towards the outer canthus; while in the latter case these conditions will be reversed.

As regards the causes and treatment of these limited forms of paralysis, there is nothing to add to what has already been stated about ptosis. It is sometimes desirable, while the diplopia continues, to exclude the deviating eye from vision by a shade, an opaque spectacle-glass, or other suitable contrivance, on account of the vertigo and uncertainty of gait which may be occasioned by the double images.

See also EXOPHTHALMIC GOÛTRE; LACRYMAL APPARATUS, Diseases of; LAGOPHTHALMOS; ORBIT, Diseases of; STRABISMUS; STYE; and VISION, Disorders of.

R. BRUDENELL CARTER.

## F

**FACHINGEN**, in Nassau, Prussia. Acidulous gaseous waters.—See MINERAL WATERS.

**FACIAL PARALYSIS**.—SYNON.: Paralysis of the Portio dura; Bell's Paralysis.

**DEFINITION**.—Paralysis of the muscles of the face, due to disease or injury of the nucleus or fibres of the seventh nerve (the portio dura of the seventh pair, according to the older nomenclature).

Above the nucleus, the motor path for the face decussates, and joins that from the arm and leg; damage, therefore, in the pons (above the crossing), in the crus, internal capsule, or hemispheres, may produce facial paralysis, but does so usually as a part of hemiplegia. This paralysis, which is on the same side as that in the limbs, is partial only, affecting chiefly the muscles of especial unilateral use (as the zygomatici); and very little those of solely bilateral use, in the upper part of the face—*e.g.* the frontales. In this article paralysis from damage to the fibres or nucleus of the nerve itself will alone be considered.

**ÆTIOLOGY**.—(1) The most common causes of facial palsy are unilateral, and damage the nerve as it passes through the narrow canal in the temporal bone. There, the slightest inflammatory effusion will cause pressure on the nerve. Such neuritis may be due to exposure to cold (and is often then called 'rheumatic'); to contiguous bone-disease, as caries; and sometimes to syphilis. Hæmorrhage into the sheath may also compress the nerve. Cold is the most common cause of palsy, but the exposure is often forgotten; it has been supposed to act by paralysing the peripheral nerve-twigs, but this is never the case in one-sided paralysis; in all cases lasting more than a few days, evidence of changed nutrition may be detected in the nerve-trunk as it emerges from the stylomastoid foramen. External cellulitis may extend into the canal. Tumours of the bone and fracture of the base may also damage the nerve as it passes through the bone. (2) Injury to the nerve outside the skull by blows, pressure by forceps in instrumental delivery, incised wounds—accidental or surgical, or parotid and other tumours, are occasional causes. (3) Within the skull the nerve may be dam-

aged by meningitis, acute or chronic, and especially by syphilitic inflammation, or by pressure of neighbouring growths. The radi- cular fibres within the pons, or the nucleus beneath the fourth ventricle, may be damaged by hæmorrhage, softening, or by tumours involving that part.

*Double facial paralysis* is very rare, and is due to damage to both nerves at the base of the brain from meningitis or symme- trical syphilitic disease; to symmetrical otitis; or to an affection of the nuclei by disease of the pons, or to loss of function of the nerve-fibres in various forms of peri- pheral neuritis. It is probably by the latter agency that double facial paralysis some- times results from toxic blood states. Syphilis and diphtheria are the most common ante- cedents of double facial palsy not due to demonstrable injury to the nerves. Disease of the nuclei affects both sides most fre- quently when it is degenerative in nature; it is then commonly partial, involving espe- cially the lip muscles in labio-glossal para- lysis, or the orbicularis palpebrarum in rare cases of nuclear ophthalmoplegia. Lastly, it may be mentioned that the facial muscles (generally those in and about the lips—rarely all) are occasionally involved in idiopathic muscular atrophy, and an affection of the nerves may be thus simulated.

**SYMPTOMS.**—The onset of facial paralysis is usually rapid, when due to its common causes, including neuritis within the canal. It occupies from a few hours to three or four days in its development. It is found, for instance, one morning, in drinking, that the fluids run out of the side of the mouth; the face is noticed to be a little unsymmetrical; at night the eye cannot be completely closed, and next morning the paralysis is found to be complete. Rarely the onset is in a few minutes; probably then a hæmorrhage into the nerve-sheath is the cause. When infla- mation is excited by cold, the symptoms often commence within twenty-four hours of the exposure. They are usually preceded or accompanied by some pain about the ear or side of the head.

In complete unilateral facial paralysis all the muscles on one side of the face are paralysed. At rest, the smooth forehead and lowered angle of the mouth are the chief indications, but on movement the difference between the two sides becomes very marked; the one half of the forehead moves alone in frowning or elevation of the eyebrow. The eyelids cannot be brought together, and in the attempt to close the eye, the globe is rolled upwards, so that only the sclerotic appears between the gaping lids; the patient then often fancies that the eye is shut. During sleep the eye remains open. In smiling, the lips may be displaced altogether to the healthy side, from the unopposed action of the zygomatic muscles; the nostril

of the affected side cannot be dilated; the upper lip cannot be raised; the cheek flaps loosely, from the relaxation of the buccinator; and, from the same cause, food accumulates between the jaws and the cheek. Whistling is impossible, from the paralysis of half of the orbicularis, and the lips cannot be ap- proximated sufficiently even to permit of a candle being blown out. When the lesion is between the junction with the large petrosal and that with the chorda tympani nerves, taste is partly or entirely lost in the front of the tongue. The loss of power of recognising acid and saline substances is most marked; but bitters and sweets are also not tasted in this part. Such loss is common in neuritis from cold, and shows that the inflammation has extended some distance up the canal. In rare instances, loss of taste has followed division of the nerve outside the skull, prob- ably always because secondary inflamma- tion has passed up the nerve. The palate is never paralysed from disease of the facial nerve. It is certain that the opposite statement rests on defective observation or interpretation. Obliquity of the uvula is common under normal conditions. Defect of movement, to be significant, must follow the course of any associated palsy of external muscles. The nerve-supply of the palatine muscles has been proved to be from the spinal accessory.

In some cases giddiness attends the onset of facial paralysis. In slight forms there may not be complete loss of power; but the defect is at first pretty equally distributed over all parts of the face.

In cases which recover, some return of power takes place in from a week to two months, and improvement is usually earliest in the upper part of the face; the power of frowning, winking, and closing the eye being soonest regained; that of moving the lips and mouth returning last. Even after several months of immobility, recovery may take place, but in these cases it is rarely complete, and a troublesome condition is apt to super- vene: some of the muscles, especially the zygomatici, become shortened in late rigidity, and hence at rest the naso-labial wrinkle is deeper on the paralysed than on the healthy side, although the possible movement may be much slighter. This condition sometimes comes on rather rapidly; there is generally, in addition, an associated over-action of the upper and lower facial muscles, whereby, for instance, the orbicularis palpebrarum and the zygomatic and other muscles about the mouth act together in undue degree; in smiling, the eye shuts; and on closing the eye, the mouth is drawn upwards. Sometimes there are also spontaneous clonic contractions. This late rigidity and over- action may prove very troublesome and con- stitute a nuisance, especially to the young, not less than the original palsy, and more

serious in that it is enduring. The writer has known it induce a good-looking girl to put an end to her life. If the interruption of the nerve is absolute and permanent, the muscles remain toneless, and no contracture supervenes.

The electrical condition of the muscles is very important. It is that always seen in paralysis from nerve-lesion. The muscles, after a day or two of slightly increased irritability to both faradism and the slowly interrupted voltaic current, lose gradually their irritability to the former, retaining that to the latter, and even exhibiting to it increased irritability, so that they act to a smaller number of cells than do those on the healthy side. In the nerve, on the other hand, the irritability is lost to both forms of electricity, this loss proceeding *pari passu* with the degeneration which follows separation of the nerve from its nutrient centre. In slighter and more transient forms of facial paralysis, the change in irritability of muscle and nerve may be less; but even in most, which last only a few days, a slight change in irritability may be discovered. In the slightest cases (and in the earliest stage of more severe forms) there may be an increased irritability in the nerve, often most marked to the induced (faradic) 'shocks'—*i.e.* isolated currents. Considerable change in irritability is proof of considerable disease of the nerve, and is thus of the highest prognostic importance, but it is not distinct until after a week; if absent at the end of a fortnight, the disease is slight, and although the paralysis may still be complete, it will probably not last long.

**DIAGNOSIS.**—The diagnosis of facial paralysis (commonly so-called—*i.e.* that due to disease of the nerve or its nucleus) is easy. It depends (1) on the implication of all the facial muscles, including those of bilateral use that escape in hemiplegia, because related to both cerebral hemispheres; (2) on the indication afforded by the electrical reactions that the nerve or its nucleus is diseased. Hence it is important to observe all the muscles of the face, and to ascertain the electrical reaction, in order to determine whether it is the variety now described, or is cerebral and really part of hemiplegia. The recognition of the place of the lesion is less easy. When within the pons, it is often associated with paralysis of the sixth nerve, or with hemiplegia of the opposite side from damage to the motor path to the limbs. At the base of the brain the auditory nerve is usually affected at the same time, but care must be taken not to mistake deafness due to ear-disease for an affection of the auditory nerve within the skull. When there are no other paralyses, the disease is probably within the bony canal. Deviation of the uvula is of no significance; affection of taste in the front of the tongue indicates disease within the canal. Special inquiry should be made

for exposure to cold, ear-disease, syphilis, or a blow.

**PROGNOSIS.**—The majority of cases of facial paralysis are due to disease of the nerve, and the prognosis depends on the evidence of the degree of damage to the fibres, especially on the indications afforded by an electrical examination, and also on the cause of the lesion. When this is progressive in nature, the interference with the nerve is necessarily persistent in duration. Thus, in facial paralysis due to a growth damaging the nerve, the prognosis must be grave. In syphilitic disease, on the other hand, it is good, provided the duration of the damage to the nerve is short. This is also the case in paralysis from cold, in which, however, the evidence afforded by electricity is of special importance.

In double facial paralysis, the evidence of central mischief renders the prognosis unfavourable, but recovery may be hoped for if the lesion is acute and the palsy incomplete. In syphilitic cases, the prognosis is, on the whole, good. In all cases of double facial palsy from peripheral neuritis, there is a similar affection of other parts, and the prognosis must be determined by the general character and course of the affection, and especially the degree to which the morbid blood-state is amenable to treatment.

**TREATMENT.**—The treatment of facial paralysis will depend on the probable cause. When due to its common mechanism—neuritis from cold, hot fomentations to the side of the head and face should be employed in the early stage of the affection, followed by a blister behind the ear. A blister should never be applied in front of the ear, over the opening of the canal, because it will cause some subcutaneous cellulitis, and such cellulitis may actually cause facial neuritis by spreading to the nerve-sheath in the canal. The side of the head should be protected from cold. Internal treatment of neuritis must vary according to the cause. If this is syphilis, iodide of potassium in 10 gr. doses may be given; if exposure to cold, diuretics and small doses of mercury in the early stage, followed by tonics. Electricity to the muscles is needed if the damage to the nerve is so severe as to entail secondary degeneration and consequent changes in nutrition. If there is no change in irritability at the end of a fortnight, recovery will probably occur so soon as to make electrical treatment superfluous. If needed, the voltaic current should be used, slowly interrupted by a commutator, or by the positive or negative pole being moved over the individual muscles (according as they react more readily to the one or the other), the opposite pole being placed over the nerve. Only such strength should be employed as produces distinct muscular contraction. Although electricity probably does no more, it keeps up the

nutrition of the muscular fibres while the nerve is recovering. Ultimate recovery is thus more speedy and more complete than without local treatment. The application can be made quite well by the patient, one electrode being kept over the place where the nerve divides, the other moved (1) across the forehead; (2) along the eyebrow; (3) along the lowered upper eyelid; (4) along the raised lower eyelid; (5) beside the nose to the upper lip; (6) along the upper lip; (7) along the lower lip; (8) from the zygoma to the angle of the mouth. In (3) and (4) the current should be weaker than in the other places. Rubbing may be employed, the individual muscles being subjected to a process of gentle shampooing. The late contraction which occurs in severe cases is but little influenced by treatment. Gentle elongation of the muscles may be practised, and electrical applications should be stopped, since the stimulation of the sensory nerves may increase the late contraction by a reflex influence; its occurrence shows that a point has been reached when the influence of electricity for good can be but small.

In external mischief, and in intracranial disease, the treatment of the facial paralysis is usually subordinate to that of its cause.

The treatment of double facial paralysis presents no special points for consideration, except when it is part of multiple neuritis, the indications for which are described in a special article. W. R. GOWERS.

**FACIAL SPASM.**—**SYNON.**: Mimic cramp; Fr. *Tic convulsif*. When affecting the eyelids, Blepharospasm; Nictitation.

**DEFINITION.**—Spasm, sometimes tonic but more often clonic, or both, involving some or all of the muscles supplied by the facial nerve, on one side or on both.

**ÆTIOLOGY AND SYMPTOMS.**—Spasm in the face may be part of a wider convulsive movement, as in epilepsy, hysteria, chorea, or torticollis, diseases dependent on central changes. That which begins in, and is limited to one side of the face, may be due to irritation of the trunk of the facial nerve by growths, by an aneurysm, or caries of the temporal bone, or to an actual lesion of the motor centre for the face in the cortex of the brain. Spasm due to disease of the nerve may follow facial paralysis (*see* FACIAL PARALYSIS). Much more frequent, however, are cases, unilateral or bilateral, in which the affection comes on gradually, chiefly in females in the second half of life, after depressing emotion. It is probable that such cases are due to deranged functional action of the cortical motor cells, through which, we may remember, emotion is so extensively expressed. In such cases the affection usually begins on one side, often in the orbicularis palpebrarum; and if it spreads to the other side, this muscle is first involved. In another group of cases, affec-

tions of the eye lead to spasmodic closure of the lids—*blepharospasm*. In other cases beginning in the eyelids, no local cause can be detected, as in the local clonic spasm affecting the orbicularis palpebrarum, known as ‘involuntary winking’ or *nictitation*, which seems to be a pathological development of the normal process. This is also a common seat of the spasm in the face that is part of ‘habit-spasm’ or ‘habit-chorea,’ but it has no connexion with true chorea.

Facial spasm is painless. The spasm is usually increased by emotion, by voluntary movement, and by stimulation of the fifth nerve. Slight aural symptoms have been observed, referable to contraction of the stapedius. Usually the movement remains limited to the face; sometimes it spreads to the muscles of mastication, of the neck, &c. The spasm, in typical cases, is both tonic and clonic, clonic paroxysms occurring from time to time, and being fixed by tonic contractions, or *vice versa*. All forms cease during sleep.

The only secondary form of facial spasm (besides that which supervenes on a paralysis of the nerve) is the clonic spasm in the frontales that is often met with in the form of ‘torticollis’ to which the writer has given the name of ‘retrocollic spasm.’ In this there are bilateral contractions in the muscles of the back of the neck, jerking the head backwards. These muscles are physiologically associated with the frontales, as may be seen when a person looks up, and this association is reproduced in disease.

**PROGNOSIS.**—The prognosis of facial spasm is seldom good. If the cause can be discovered, it can rarely be removed, and the affection is generally most obstinate.

**TREATMENT.**—General tonics and local sedatives are the chief agents in the treatment of most forms of facial spasm. When there is evidence of direct irritation of the fifth or the facial nerve, counter-irritation by blisters may be tried. A careful search should be made for reflex causes, and if possible they should be removed, decayed teeth being extracted, and neuralgia relieved, although the result is generally disappointing, if the spasm has long existed. When there are tender places in the course of the fifth nerve, pressure on which stops the spasm, the hypodermic injection of cocaine at these places should be employed. Morphine, used as a sedative, has a general influence which makes the remedy almost worse than the disease. Iron, quinine, or valerianate of zinc are the best general tonics. The writer has once known recovery to follow the administration of bromide, Indian hemp, and nuxvomica; the case was characteristic, occurring late in life. The operation of nerve-stretching has often arrested the spasm for a time, and, in a few cases, permanently. It deserves a trial in severe cases, acting, perhaps, by the effect on the centres of the arrest of

afferent impulses produced by the muscular contractions. These must tend to maintain the morbid state of the deranged cells. Its effect might probably be augmented by the simultaneous use of sedatives, such as the injection of cocaine to lessen the influence of the fifth nerve on the facial centre. Where the affection springs from an habitual movement, facial gymnastics may be of service. A weak voltaic current, applied from the ear to the muscles, unbroken, has been recommended; but it rarely, if ever, effects a cure. The same is true of the application of voltaism to the sympathetic and to the back of the head. *See EYE, AND ITS APPENDAGES, Diseases of.* W. R. GOWERS.

**FACIES HIPPOCRATICA** (Lat.)—A peculiar expression of the face, so named from having been graphically described by Hippocrates. It is most strikingly observed in persons exhausted by copious discharges, as in cholera, by prolonged wasting diseases, or by starvation; and especially before impending death. It is thus described by Hippocrates: 'A sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended, and parched; the colour of the whole face being green, black, livid, or lead-coloured.'

**FÆCES, Examination of.**—*See STOOLS.*

**FÆCES, Involuntary Discharge of.**  
*See DEFÆCATION, INVOLUNTARY.*

**FÆCES, Retention of.**—**ÆTIOLOGY.**—In the absence of mechanical obstruction, such as that due to adhesions, bands of false membrane, uterine pressure, stricture, tumours or hæmorrhoids, the main causes of fecal accumulation are:—(a) A sub-paralysis of the intestinal muscular fibres from defective innervation, or from over-distension of the walls of some portion of the large bowel; (b) loss of reflex irritability of the rectum; and (c) dryness of the mucous surface of the colon. Hence it is frequently met with in the debilitated, the bedridden, the paralysed, the aged, and the sedentary; and women are more prone to it than men. Loss of reflex sensibility in the rectum is frequently the sole cause. The fæces delivered into the rectum by the contractions of the sigmoid flexure and the descending colon no longer excite the act of defæcation; and collecting there as a large desiccated mass, they determine a gradual and painless retention in the large intestine, and particularly in the most distensible parts—the sigmoid flexure and the cæcum. The accumulation once set up tends also to perpetuate itself, by arresting more and more the peristaltic movements of the distended bowel around the collected fæces.

**SYMPTOMS.**—Fæces often accumulates slowly and without the knowledge of the patient. Hence in cases in which sensibility has been

blunted by age, disease, or great debility, the discovery of large collections in the rectum or colon may surprise even the practitioner, who is generally led to a local examination by disturbances set up by the retention. There is usually constipation or an insufficient discharge of solids; a regular and possibly excessive relief of the bowels does not, however, exclude accumulation, for even fluid fæces may pass through or over old collections. The evacuations are lumpy, or consist of detached hard, dry, dark scybala, or of a single mass; and when the accumulated matters are dislodged by aperients, they often emit an offensive and sour odour. Not uncommonly, however, the alvine discharges are entirely fluid or semifluid, and consist chiefly of mucus tinged with fæces. Fæcal collections in the rectum and sigmoid flexure are apt to excite tenesmus and frequent voiding of mucus and blood—a condition resembling that of chronic dysentery, but without the putrid-flesh-like odour of dysenteric evacuations; and the finger encounters a mass of hardened fæces in the rectum. A collection of fruit-seeds—as of the fig—in the rectal pouch will also set up these symptoms.

An accumulation in the cæcum, or in any part of the colon, may be detected through the abdominal wall as a tumour more or less movable, uneven or nodulated, and doughy to the touch; and on the finger being firmly pressed into it, presenting a depression which may last for some minutes. In rare cases the large bowel throughout may be so greatly distended as to apparently fill the abdomen with a solid mass, which, like other fecal collections, yields to the firm pressure of the finger. A tympanitic state of the abdomen may, however, so obscure the examination as to prevent the recognition of even moderately large accumulations. The fecal tumour in the cæcum is globular; but in any part of the colon it is usually elongated; and in the descending colon and sigmoid flexure it is harder than elsewhere, and very distinctly nodular from scybala. It may drag the transverse colon downwards, even as low as the symphysis pubis; and when confined to the right half of the transverse colon it may be mistaken for an enlarged liver.

But in other cases the retained fæces set up more or less severe abdominal disturbance—such as colicky pains, great distension, &c.—which may terminate either gradually or suddenly in complete intestinal obstruction, now and then proving fatal (*see* **INTESTINAL OBSTRUCTION**). Usually, however, several obstructive attacks of increasing severity succeed one another at intervals, being relieved either spontaneously or by the use of enemata and aperients. Ulceration of the intestinal mucous membrane is also apt to follow the continued retention of fæces, and may lead to fatal perforative peritonitis, or to stricture from the healing of the stercoral

ulcer. See CÆCUM, Diseases of; COLON, Diseases of; and CONSTIPATION.

**TREATMENT.**—A hard ball of fæces, or of agglutinated scybala, or fæcal concretions in the rectum resisting dislodgment by aperients or enemata, should be broken up and removed by introducing within the sphincter two or three fingers and a scoop, or the handle of a spoon; this procedure in the female being facilitated by the fingers in the vagina. An attempt may be made as a preliminary measure to macerate the hard fæcal mass by a cacao-butter suppository or injecting a few ounces of warm olive-oil. Sometimes a glycerine suppository or glycerine injection (5ij.) will excite and aid the natural efforts in expelling the obstruction. Symptoms of obstruction should be met by injecting by the stomach-pump tube, or Davidson's syringe, large quantities—from 80 to 120 oz.—of some warm fluid such as soap-water or gruel, through the long rectal tube passed its full length; and it is often advisable to repeat at intervals the injection of even large quantities until relief is obtained. Care must be exercised in the use of the long tube not to injure the bowel; as well as in the injection of large quantities of fluid. If the indurated mass (as when in the lower parts of the large bowel) can be traversed by the tube, warm olive-oil should be slowly injected, and afterward the warm fluids in ample measure. As a rule, purgatives merely aggravate the vomiting induced by the obstruction, while large enemata usually afford direct relief. In the absence of vomiting, however, a brisk aperient—such as calomel, castor-oil, or colocynth with croton-oil—may afford timely aid in clearing away the fæcal accumulation. When aperients and enemata fail to dislodge the fæcal collections, and the abdomen becomes painfully distended, a combination of opium, belladonna, and aloes, given at regular intervals—the dose of aloes, at first small, being increased as the pain diminishes—may enable the bowel to overcome the difficulty; but when there is much pain, and, above all, obstinate and especially stercoraceous vomiting, opium and belladonna, or subcutaneous injection of morphine, should be given until the subsidence of these symptoms. In such cases belladonna alone pushed to large doses has been known to effect relief after the failure of other means.

Massage may afford aid in dispersing fæcal collections and improving the peristaltic action of the bowel; to be applicable, there should be little or no tenderness, and it should be addressed first of all to the distal side of any detectable fæcal tumour. Electricity may prove useful. See CONSTIPATION.

Distressing tympanites, which, thwarting the peristaltic movements, often intensifies obstruction, has been overcome by puncturing the bowel with a fine trocar or aspirator-needle, after failure of other measures.

The general medicinal treatment of the conditions that favour the recurrence of the difficulty should consist of a prolonged course of gentle, yet efficient, evacuants, combined with tonics. The gradual collection of fæces, the toneless state of the walls of the large bowel, the scanty secretion of intestinal mucus, and the loss, in many cases, of reflex sensibility in the rectum, require the persevering use of these remedies. A pill containing aloes, iron, belladonna, and nuxvomica generally affords the most satisfactory results. See CONSTIPATION.

GEORGE OLIVER.

**FAINTING.**—A popular synonym for syncope. See SYNCOPE.

**FAINTNESS.**—Faintness signifies a feeling of great weakness or exhaustion, as if the subject of it were about to become exhausted, or to sink or faint.

**FALLING SICKNESS.**—A popular synonym for epilepsy. See EPILEPSY.

**FALLOPIAN TUBES, Diseases of.** The oviducts are liable to the following morbid conditions:—(1) Malformations; (2) Displacements; (3) Contractions; (4) Dilatations; (5) Inflammation; (6) New-Growths; and (7) Tubal Pregnancy.

1. **Malformations.**—The Fallopian tubes may be congenitally wanting, either on one or on both sides; or they may be impervious; or, instead of a single opening into the abdominal cavity, there may be two or more.

2. **Displacements.**—The tubes may be stretched or widely displaced from their normal position by growths or effusions in their neighbourhood, such as ovarian, or parovarian.

3. **Contractions.**—These canals may be impervious, from inflammation of the lining membrane, or from peritonitis, pressure, or torsion.

4. **Dilatations.**—The Fallopian tubes may be distended, even to a great extent, by (a) mucous or serous fluid (*hydro-salpinx*); (b) pus (*pyo-salpinx*); or (c) blood (*hæmo-salpinx*). In hydro-salpinx the tubes, if much distended, become sacculated, giving rise to a string of cystic tumours. The fluid collects chiefly at the abdominal end of the tube, but occasionally it may escape, and in large quantity, through the uterus. Should the tube burst and discharge its contents into the peritoneal cavity, the results will be serious, especially if the fluid be pus. When hæmo-salpinx is the result of menstrual retention from atresia uteri, bursting of the tubes internally is liable to follow operations for the relief of the obstruction.

5. **Inflammation.**—Inflammation of the Fallopian tubes (*salpingitis*) is apt to be caused by gonorrhœal infection, or it may occur during the puerperal state. Stenosis or *pyo-salpinx* may be the result.

6. **New-Growths.**—The Fallopian tubes may be the seat of the following new-growths: fibroid tumours, mucous polypi, cysts, cancer, and tubercle. Fibroids resembling those of the uterus may attain a considerable size. Small polypi growing into the canal may partially obstruct the duct. The cysts are usually the so-called *hydatis of Morgagni*, an embryological relic; but other small simple cysts may be met with at the orifice of the tube, around the fimbriated extremity. Primary cancer of the Fallopian tubes rarely, if ever, occurs. Tubercle, however, may be primary, and may occur before puberty. It usually begins at the abdominal end, and may lead to blocking of the tube.

7. **Tubal Pregnancy.**—An important affection of the Fallopian tubes is that arising from disintegration of the lining membrane of the tube, producing destruction of the ciliated epithelium, and so affording a nidus for the attachment of the misplaced product of conception, which thus grows within the tube in some part of its course. This abnormality is apt to be attended with very grave results, bursting of the tube frequently occurring about the third month of gestation; and serious, sometimes fatal, internal hæmorrhage may hence ensue. Tubal gestation usually occurs on one side only, while other affections of the tubes are often symmetrical, a point of diagnostic importance. See PREGNANCY, Disorders of.

**Regurgitation** of blood, of septic matters, and of fluids injected into the uterus, sometimes takes place along the Fallopian tubes, and this accident is always attended by grave consequences. Hence the danger of injudicious intra-uterine irrigation with corrosive sublimate and other antiseptic solutions during the puerperal period, and more especially after early abortions.

**Mechanical Obstruction** of the Fallopian tubes is not an infrequent cause of sterility.

**TREATMENT.**—The diagnosis of affections of the Fallopian tubes being difficult, their treatment is likewise obscure, and must be in a measure guided by general principles in each case. In pyo-salpinx severe peritonitis and death may result from rupture of the sac, as may speedy dissolution from internal hæmorrhage in tubal gestation. In the former case, and in hydro-salpinx, puncture with the aspirator might be permissible were a clear and unequivocal diagnosis made; or, failing this, the removal of the affected tube by salpingotomy as the only alternative. But, although in the hands of some eminent specialists this procedure has been attended with satisfactory results in many cases within the past few years, as a rule, under ordinary circumstances, the diagnosis is surrounded by so many difficulties that such an operation can only rarely be justified.

ALFRED WILTSHIRE. T. MORE MADDEN.

**FALSE MEMBRANE.**—An inflammatory exudation of a fibrinous character, which is deposited in layers, chiefly on mucous surfaces, and occasionally on abrasions of the skin. It is well exemplified in diphtheria and plastic bronchitis. See DIPHTHERIA.

**FAMILY DISEASES.**—Diseases which are found to run in families, or diseases to which members of the same family seem peculiarly liable. See DISEASE, Causes of.

**FARADISATION, Uses of.**—See ELECTRICITY IN MEDICINE.

**FARCY.**—See GLANDERS.

**FASCIÆ** (*fascia*, a band).—The fasciæ are subjects of medical and surgical interest, with respect to their anatomical relations, the diseases to which they are liable, and certain points of diagnosis in connexion with them.

**I. Anatomical Relations of Fasciæ.**—The situation and connexions of the fasciæ, according as they are fasciæ of *investment* or fasciæ of *attachment*, are of the greatest practical importance in the following classes of diseases: (1) Suppuration; (2) Extravasations, and Cellular Emphysema; (3) Herniæ; (4) Dislocations and other injuries; (5) Diseases of Encapsuled Organs; and (6) New-Growths.

**1. SUPPURATION.**—The physical influences exerted by fasciæ upon pus are chiefly two. First, fasciæ may limit the size of an abscess, determine its tension and the many results of the same, and thus affect both the local phenomena and the general symptoms. Secondly, when the pus is not confined, the fasciæ serve to determine the course that it will take, and the situation in which it will discharge. Every abscess may be said to be influenced in this way by the relations of fasciæ, but certain fasciæ have to be specially noted as causing pus to burrow, and hence they should be enumerated here.

(a) *Fasciæ of the Head and Neck.*—The fasciæ of the scalp; the temporal fascia; the cervical fascia; and the post-pharyngeal fascia, which conducts pus from the cervical vertebræ to the parotid region and tonsil, and to the region of the carotid vessels.

(b) *Fasciæ of the Upper Extremity.*—The axillary fascia; and the deep fascia of the upper extremity generally, including the palmar fascia and the sheaths of the tendons.

(c) *Fasciæ of the Thorax.*—The fasciæ of the intercostal spaces and of the mammary region; the fasciæ reaching from the neck to the upper part of the pericardium and the aorta, and to the posterior mediastinum, respectively; the fasciæ of the anterior mediastinum; and those connected with the diaphragm—all of which determine the course of intrathoracic abscesses

(d) *Fasciæ of the Abdomen and Pelvis.*—The transversalis fascia; the fasciæ connected with the transversalis muscle, especially posteriorly, which influence the course of lumbar abscess; the sheaths of the psoas and the iliacus muscles; the pelvic, recto-vesical, obturator, and anal fasciæ; the fascial investment of the prostate; the superficial and deep layers of the superficial fascia of the perinæum; the superficial and deep layers of the triangular ligament; and the fascial investments of the rectum, bladder, uterus, and vagina, which determine the course of purulent collections in the pelvis.

(e) *Fasciæ of the Lower Extremity.*—The fascia lata and its processes; the tensor fasciæ femoris; the popliteal fascia; the deep fascia of the leg; and the plantar fascia and its compartments.

2. EXTRAVASATIONS AND CELLULAR EMPHYSEMA.—When blood or urine escapes from its natural reservoirs, or when air or gas has found its way amongst the tissues, the direction that the extravasated substance takes is markedly influenced by the fasciæ with which it comes in contact. The principal fasciæ of importance in this respect are as follows:—

(a) *Fasciæ of the Head and Neck.*—The fascia of the scalp, and the cervical fascia.

(b) *Fasciæ of the Thorax.*—The fasciæ of the intercostal spaces; and the mediastinal fasciæ, through connexions with the cervical.

(c) *Fasciæ of the Upper Extremity.*—The deep fascia in general.

(d) *Fasciæ of the Abdomen and Pelvis.*—Scarpa's fascia, or deep layer of the superficial fascia; both layers of the superficial and of the deep perineal fascia; and the fasciæ of the prostate and bladder—all being of the greatest importance in cases of extravasation of urine or feces.

(e) *Fasciæ of the Lower Extremity.*—The fascia lata in general; the popliteal fascia; the deep fascia of the leg; and the plantar fascia.

3. HERNIÆ.—The occurrence of herniæ, and the direction that they take, are in a great measure determined by the condition of the fasciæ in contact with the viscera. Most important in this relation may be mentioned the fascia transversalis, the iliac fascia, the sheath of the femoral vessels, the obturator fascia, the cribriform fascia, and the fascia lata.

4. DISLOCATIONS, &c.—Certain fasciæ serve as supports for the heads of bones, and for the viscera; and these will have an important influence either in promoting or in preventing dislocation, displacement, or other injury of these parts, as the case may be. The shoulder is supported by the costo-coracoid fascia, and this relation considerably affects the signs of dislocation at that joint. In fractures of the patella, the fasciæ of the knee-joint promote separation of the frag-

ments. The fascia of the neck helps to support the pericardium, and must limit the displacements of the heart. The bicipital fascia of the forearm protects the brachial artery beneath it in venesection, at the bend of the elbow. On the other hand, the attachment of the cervical fascia to the jugular veins facilitates the entrance of air into the circulation through a wound at this point. And, lastly, the pelvic viscera are supported by the transversalis, pelvic, iliac, and recto-vesical fasciæ; whilst the cord and testis have their special fasciæ to keep them in position.

5. DISEASES OF ENCAPSULED ORGANS.—A considerable number of organs, many important vessels, and a great variety of muscles are contained in distinct fascial capsules, sheaths, or envelopes, which will affect the course of the diseases of these structures in many ways—defining their limits, or determining the direction in which they spread, and thus influencing both their local and general phenomena. This has been already alluded to under the first head; but it is necessary to enumerate here the principal fasciæ that act in this way, namely:

(a) *Arterial sheaths:* of the carotid, subclavian, thoracic-aortic, and femoral. (b) *Muscular sheaths:* of the masseter, buccinator, psoas, iliacus, quadratus lumborum, erector spinæ, pectineus, rectus abdominis, levator ani, tensor vaginæ femoris, and the palmar and plantar muscles. (c) *Visceral capsules:* of the thyroid gland, parotid and submaxillary glands, tongue, prostate, penis, vagina, bladder, rectum, and mamma.

6. NEW-GROWTHS.—The direction, rapidity, and extent of spread of new-growths are considerably modified in certain situations by the relations of the fasciæ above described.

II. Pathological Relations of Fasciæ.—The principal diseases of fasciæ are: (1) Inflammation and its results; (2) Ossification; (3) Calcification; (4) Rheumatism; (5) Gout; (6) Syphilis; (7) Contraction; and (8) Extension. None of these conditions can be said to be common, or of serious importance.

1. INFLAMMATION.—Inflammation involving a fascia is usually secondary, having spread to it from the neighbouring structures, and especially from the muscle or organ of which the fascia may form the sheath. Even under these circumstances, a fascia rather resists than participates in the inflammatory process, as has been described above; and when it is involved, it tends to ulcerate on account of its feeble vitality, and to be discharged in the form of sloughs. The healing process is extremely slow in fasciæ, and after serious lesion their function is never completely restored.

2. OSSIFICATION.—Occasionally in aged persons portions of fasciæ are found transformed into bony tissue.

3. **CALCIFICATION.**—Calcification is very rare in fasciæ.

4. **RHEUMATISM.**—The condition known as muscular rheumatism, or according to its situation as lumbago, torticollis, &c., is believed by some authorities to involve the fibrous coverings or fasciæ of the affected parts. In the same way, many of the aches of some forms of 'chronic rheumatism' may possibly have their seat in fasciæ; and certain cases of neuralgia are probably to be referred to rheumatic inflammation of the nerve-sheath.

5. **GOUT.**—Amongst the pains of the gouty subject are some which are no doubt due to affections of fasciæ, such as pains in the heel and instep, and neuralgia of the sciatic, the anterior crural, and the brachial nerves.

6. **SYPHILIS.**—Syphilis may attack the fasciæ in the form of nodes, which in situations of low vascularity are apt to ulcerate, as, for example, at the inner aspect of the knee.

7. **CONTRACTION.**—Contraction is the most obvious of the morbid conditions of fasciæ, giving rise as it does to well-marked deformities. The fasciæ of the hand and foot are most liable to this change, with the result of unnatural flexion of the fingers and toes. Such contraction of the palmar and plantar fasciæ may be due to wounds, burns, or inflammation from any cause, or to gout or rheumatism; it is sometimes congenital; and it sometimes occurs without evident cause.

8. **EXTENSION.**—A fascia or sheath, though extremely inelastic, is liable to be stretched by swelling of the parts which it envelops; and, if the cause do not speedily disappear, may remain more or less permanently extended. The best instance of this condition is pendulous abdomen after pregnancy or other kind of abdominal enlargement.

J. MITCHELL BRUCE.

**FASCIOLA** (*fasciola*, a thin band).—A genus of trematode parasites of which the common liver-fluke forms a good type. This entozoon (*F. hepatica*) is characterised by the possession of a branched intestinal canal, thus differing from the flukes belonging to the genus *Distoma*, in which the canal is simple and bifurcated. The liver-fluke is of rare occurrence in man, though extremely abundant in, and destructive of, ruminating animals. See ENTOZOA.

T. S. COBBOLD.

**FASTING.**—The manifestation of vital activity implies consumption of material; and unless the supply of material in the form of food is equivalent to the loss occurring, a progressive wasting of the body and failure of power must ensue. Hence these phenomena constitute the necessary accompaniments of fasting; and with its prolongation the question resolves itself into one of time

—when the exhaustion of material shall have proceeded to such an extent as to render the continuance of life impossible.

**PATHOLOGY.**—To Chossat we are indebted for showing that the immediate cause of death from fasting is a reduction of the body temperature. At first there is a gradual, but not very extensive fall. Afterwards a more rapid decline occurs, until the reduction amounts to about 29° or 30° F. below the normal point, when death ensues. Chossat noticed that if, whilst in the state of torpor preceding death, the temperature of the animal experimented on was raised by exposure to artificial warmth, a restoration of consciousness and muscular power was induced; and some of his subjects of experiment which were thus rescued from impending death afterwards thoroughly revived on being supplied with food.

**SYMPTOMS.**—The most prominent symptoms arising from fasting are those due to the special sensations produced by the absence of food and fluid, and those attributable to a decline of the physical and mental powers. In the first place there is great uneasiness in the epigastrium. This is followed by a sense of sinking in the same region, accompanied by insatiable thirst; and if fluid be persistently withheld as well as food, the thirst becomes the chief source of distress. The countenance assumes a pale and cadaverous appearance, and a look of wildness is presented about the eyes. Emaciation becomes more and more marked, and with it there is a decline of the bodily strength. There is also failure of the mental power. Stupidity may advance to imbecility; and a state of maniacal delirium frequently supervenes. Life terminates either calmly by gradually increasing torpidity, or, it may be, suddenly in a convulsive paroxysm.

**DURATION OF LIFE.**—The usual duration of life under complete absence of food and drink may be said to be from eight to ten days. The special circumstances existing may, however, exert a modifying influence, and from the nature of these the period may be either diminished or increased. A stout person, as may be readily understood, has a chance of living longer than a thin one, on account of the store of combustible material which may be drawn upon being larger. Exposure to cold in conjunction with starvation very much hastens death. The presence of moisture in the atmosphere favours the prolongation of life, by diminishing the exhalation of fluid from the body. It may be assumed to be owing to the existence of warmth and moisture that persons buried in mines, or otherwise similarly placed, have been known to live considerably beyond the ordinary period.

The Welsh fasting girl, whose case caused so much sensational excitement in 1869, lived exactly eight days from the time she

was placed under systematic inspection to solve the problem of whether she could exist, as had been alleged by her parents, for an indefinite period without food. It appears that during the first part of the time she was cheerful and exhibited nothing extraordinary. Later on it was found that she could not be kept warm, and ultimately she sank into a state of torpor from which she could not be roused, and which speedily terminated in death.

In the Troedryhiw colliery near Pontypridd an inundation occurred in 1877, which led to the imprisonment of four men and a boy in one of the headings of the mine. The accident happened on Wednesday evening, the 11th of April. Efforts were at once made, by means of a cutting, to reach the chamber in which the imprisoned persons were confined, and to release them. This was not accomplished till the afternoon of Thursday, the 19th, when all were rescued alive and did well. They had been imprisoned in an atmosphere of compressed air nearly eight days, without food but within reach of water.

The performances of professional fasting men give a much longer time as that during which life can be sustained without food. Two feats of fasting were performed before the London public during 1890 by separate men. The first was announced to be for a period of forty, and the second for a period of forty-five days. Both were carried on for the specified time, and, through the payments received from the crowds of spectators admitted, must have proved lucrative undertakings. One of them subsequently became insane. Feats thus conducted for pecuniary gain, and supervised, as was here the case, by newspaper reporters as watchers, can hardly, it may be considered, be regarded as holding a sufficiently trustworthy position to command acceptance for the purposes of a medical work like this, whatever the amount of popular credence given to them at the time. In the case of the Welsh fasting girl watchings of considerable duration had been performed through which she had successfully passed, but the last watching was carried out by the experienced London nurses deputed to undertake the task with a strictness that defied deception, and with the result that death took place as stated.

In the case, again, of Ann Moore, of Tutbury, in Staffordshire, which, from the account published, must have attained great notoriety in the early part of the present century, watchings had been conducted in a manner to admit of testimony being given in support of her veracity; but ultimately a watching was so seriously undertaken, and with such zeal carried out, as to lead to her being brought by the ninth day into a sinking condition, when she took food, and afterwards made a written confession of the imposition that had been previously practised.

That a very small amount of food will suffice for sustaining life for a prolonged period is evidenced by what may be observed in some cases of constrictive disease affecting the œsophagus or cardiac orifice of the stomach.

**TREATMENT.**—Caution is required in the administration of food after prolonged fasting. Sudden transitions of all kinds are trying to the body; and, instead of allowing the rescued sufferer to gratify his desire to eat and drink according to his inclination after several days' abstinence, the supply of both food and drink should at first be limited, and afterwards gradually increased. There is reason to believe that the non-observance of this rule has upon some occasions been followed by disastrous consequences, which a different plan might have averted.

F. W. PAVY.

**FAT.**—Fat becomes morbid under a variety of circumstances:—

1. **Obesity.**—Fat may be found generally diffused in excessive quantity beneath the skin, beneath serous membranes, and in and upon various tissues and organs of the body. This condition constitutes what is known as obesity. See OBESITY.

2. **Partial Growth.**—A partial growth of fat sometimes occurs in paralysed muscles, and constitutes a characteristic feature of what is called pseudo-muscular hypertrophy. See PROGRESSIVE MUSCULAR DYSTROPHY.

3. **Fatty Interstitial Growths.**—Fat as fat-tissue more especially constitutes a disease when it is deposited upon and in the textures of organs, interfering with their structure and functions. This it does by pressing upon the histological elements of the organ invaded. Its effects are more clearly seen when it invades the muscular tissue of such an organ as the heart, the fibres of which, becoming more or less atrophied and distorted in their course and direction, are rendered inadequate for the performance of their functions. See HEART, Fatty Growth on.

4. **Fatty Tumours.**—Fat may also occur in isolated or circumscribed masses, constituting what are known as *fatty tumours* or *lipomata*. See TUMOURS.

5. **Fatty Infiltration.**—See FATTY INFILTRATION. RICHARD QUAIN.

**FATIGUE** (*fatigo*, *I weary*).—GENERAL REMARKS.—Fatigue is a regular and constantly returning symptom experienced by all persons. Periods of functional activity invariably alternate with periods of repose, during which the waste caused by the exercise of function is repaired. We are indebted to Sir James Paget for having pointed out that 'rhythmic nutrition is a law of nature,' and although the truth of this dogma is recognised on all hands, and may be said to be axiomatic, it has hardly

received that careful consideration at the hands of practical physicians which it deserves. Our whole life is composed of a series of vibrations—periods of tension alternating with periods of relaxation; and although the rapidity of these vibrations varies immensely, they are recognisable in all our acts, whether voluntary or otherwise. The vibrations of the heart are about seventy in a minute, those of the respiratory organs about sixteen. The whole body requires a certain period of absolute and continuous repose in each twenty-four hours (amounting to nearly one-third of the period), so that we may say its rate of vibration is once in the twenty-four hours. In like manner, the period of relaxation of the heart is about one-third of the total period of action, and this proportional correspondence between a local and a general condition is not a little interesting and suggestive. Again, it is universally ordained amongst civilised nations that once in every seven days there shall be a remission of labour and a change of occupation; and we recognise the fact that it is highly advisable for those who are occupied in one pursuit to break away from it at least once a year and indulge in that variety of work which we call amusement.

Fatigue occurs directly we attempt to alter the rhythm of our vital vibrations by prolonging the periods of tension at the expense of the period of relaxation, or by demanding for any length of time a quickening of the normal rate of vibration. We recognise the fact that athletes who over-train run risks of cardiac troubles and loss of wind; that the man who from any cause is unable to sleep runs a serious risk of permanent impairment of health; and when we find patients pursuing their avocations too zealously we know that, if such offence against the laws of nature be persisted in, general paralysis, or other forms of 'break-down,' are likely to be the result.

**DESCRIPTION.**—Fatigue may be either *general* or *local*, and both forms may be either acute or chronic. That fatigue in all its forms is due to impaired nutrition there can be little doubt, and we shall find that the symptoms of chronic fatigue are often the prelude of definite and recognisable degenerative changes.

**General Fatigue.**—General fatigue is recognised with ease both in its acute and chronic forms. There is a disability to perform either mental or physical work, and this disability is noticed first in work requiring attention or sustained effort, and last in those acts which have become automatic or secondarily automatic. The symptoms of general fatigue are usually referable to the nervous centres.

**Local Fatigue.**—Local fatigue is either acute or chronic, and the symptoms of it are referable usually to the muscles; but we

must always bear in mind that muscle and motor-nerve are practically one and indivisible, and that recent experiments have given great probability to the idea that every muscle is connected with a certain definite spot in the brain. When, therefore, we speak of a sense of fatigue, we must necessarily be in doubt, notwithstanding the fact that the symptoms are apparently located in the muscles, whether the brain, nerve, or muscle, one or all of them, be really at fault.

**Acute local fatigue.**—The first symptom of acute local fatigue is loss of power to a greater or less extent. By too frequent or too prolonged stimulation the irritability of the muscular tissue becomes exhausted, and it either refuses to respond, or responds but feebly to the stimulus of the will; whilst our power of adjusting the force of contraction to the act to be accomplished is lessened, and accuracy of movement and delicacy of co-ordination are destroyed. The second symptom is tremor, as everyone must have experienced who has been called upon for any unusual exertion. The third symptom is cramp-like contraction; and the fourth is pain, the pain being the pain of fatigue, and absolutely distinct from other varieties of pain. Fatigue is caused far sooner by prolonged muscular effort than by repetitions of short muscular efforts having due intervals of relaxation between them. Any one who has attempted to hold out a weight at arm's length knows the impossibility of continuing the effort for any length of time; and it is proverbially true that standing in one position is, to most people, far more tiring than walking, the reason being that in standing the muscles which support the body are subjected to a prolonged strain, while in walking we use the muscles on either side of the body alternately. The great increase of power which we obtain by using the muscles on either side of the body alternately would seem to be one of the chief reasons for the bilateral symmetry of the body. Not only is sustained effort a far more potent cause of fatigue than repeated effort, but we find that when fatigue supervenes, actions requiring sustained effort are the first to fail; and in this local fatigue resembles general fatigue. The last acts to be affected by fatigue are such as are automatic, and are accomplished without mental effort, and by the expenditure of the least possible amount of force. It is quite possible to exhaust a muscle by artificial stimulation, and if one of the small interossei muscles be continuously faradised, it will be found that in time its power of contraction to any form of stimulus may be absolutely abolished. It is tolerably certain that the brain can have no share in artificial fatigue thus produced, and there seems good reason to suppose that, in some people of energetic temperament, the irritability of a muscle may be exhausted, while

the power of mental stimulation remains almost unimpaired.

*Chronic local fatigue.*—This form of fatigue has causes and symptoms similar to those of acute local fatigue, and there can be little doubt that this condition is a common cause of many of those chronic maladies which seem to result from over-work, and are characterised by irregular muscular action. That some cases of writer's cramp (see WRITER'S CRAMP) are due to chronic fatigue of certain muscles employed in writing, and particularly of those subjected to prolonged effort, cannot be doubted. Some cases of torticollis seem due to the same cause. Duchenne and Brudenell-Carter have pointed out how, in cases of 'short sight,' the strain on the internal recti has caused troubles of vision, and even brain symptoms; and Mr. C. B. Taylor of Nottingham has shown reason for including in the category of fatigue-diseases a peculiar form of nystagmus, occurring amongst miners, who try their eyes by working in the dark. Dr. Augustus Waller, working at this subject from the physiological point of view, states that when a muscle is fatigued by over-stimulation of a motor nerve, it is the motor end-plate (the junction between nerve and muscle) which first fails to conduct the stimulus; and Dr. Reid, working in conjunction with Dr. Waller, has shown that when stimulation takes place from the brain, failure by conduction occurs first in the cord, where the ganglionic cell forms a junction between the fibre of the pyramidal tract and the fibre of the anterior nerve-root.

*TREATMENT.*—The treatment of fatigue in all its forms is *rest*, and the restoration of the proper rhythm of nutrition, if this be found perverted, by substituting rhythmical exercises for unrhythmical efforts.

G. V. POORE.

**FATTY DEGENERATION.**—SYNON.: Fr. *Dégénérescence graisseuse*; Ger. *Fettige Metamorphose*.

*DEFINITION.*—The process by which protein elements are converted into a granular fatty matter.

*SEATS OF OCCURRENCE.*—This change may occur in any of the component elements of the body, whether normal or abnormal.

*Physiologically.*—The production of milk from the protoplasm of the mammary cells, and of sebum from the cells of the sebaceous glands, are instances of fatty degeneration. The cells of the corpus luteum are partly in a condition of fatty degeneration; and it is by a similar change in the peripheral cells of the mature foetal portion of the placenta that normal involution of that organ is accomplished. Non-vascular structures, such as the cartilages, the cornea, and the intima of blood-vessels, frequently undergo fatty transformation of part of their substance. In a

less marked form, fatty degeneration occurs in the walls of the uterus and other muscular organs when returning to their ordinary size after temporary hypertrophy.

*Pathologically.*—As a purely morbid process, fatty change is most frequently met with in the muscular tissue of the heart, in the walls of capillaries, and in the urinary tubules; but it also occurs in the central nervous system, constituting the condition known as 'white softening'; in the liver; and in tubercular deposits, cancerous growths, infarcts, and inflammatory products in any situation whatsoever.

*ANATOMICAL CHARACTERS.*—*Physical characters.*—An organ that has undergone fatty degeneration presents the following physical characters. The bulk and weight are generally increased; the consistence is reduced—sometimes to a pulp, as in white softening of the brain; the colour is changed, either as a whole or in the affected portions of the organ, into a buffy or yellowish bloodless hue; and the resistance or firmness is diminished, so that the affected tissue is markedly flabby, and readily yields to pressure. The solid cut surface may appear compact and shining; and the section leaves a greasy stain upon the knife and fingers. When fatty degeneration is greatly advanced, as it may be seen, for example, in the liver, a portion of the organ thrown into water will float.

*Microscopical characters.*—In fatty degeneration the muscular tissue of the heart (see fig. 60) and the walls of capillaries are most suitable—especially those of the brain—for microscopical investigation. The earliest changes in the *cardiac muscle* in fatty degeneration are loss of sharpness of the individual stria, and the appearance of minute particles of oil between the elements. These changes, beginning near the nuclei, spread throughout the fibres in a longitudinal direction, while the particles increase in size, and assume the well-known characters of oil-globules. When the process is advanced, the whole of the sarcous substance is replaced by fatty particles contained within a delicate albuminous envelope. Finally the degenerated fibres either become atrophied by absorption of certain parts of the fat, and so disappear; or suffer rupture with discharge of their contents. The fatty nature of the change is proved by the solubility in ether of the particles that have escaped from the fibres.

Fatty degeneration of the *walls of vessels* is best seen in the capillaries and smallest arteries. The tunics first lose their normal translucency; minute granules appear in their substance; and these increase to form unmistakable oil-globules. Finally, the vessel gives way, and the oily particles and blood are discharged into the perivascular spaces.

In the other organs referred to, the microscopical characters agree with those just described, with certain differences dependent

upon the special structure of each. Thus fatty degeneration of a leucocyte leads to the formation of the body known as a *compound granular corpuscle*, in which the oil-drops finally replace the whole of the protoplasm. In 'white softening' of nervous tissue, the nerve-cells and probably all the nuclei of the part are converted in a great measure into granular corpuscles; and these breaking down into a fatty detritus, the whole constitutes a soft creamy-looking substance, which, as Virchow expresses it, 'is milk in the brain, instead of in the mammary gland.' 'Yellow tubercle' consists in part of cells and nuclei that have undergone fatty degeneration, and of fatty detritus. In the case of the uterus during involution the fat is probably rapidly absorbed, so that the appearances presented to the eye are those rather of atrophy than of replacement of the muscular substance by fat.

**PATHOLOGY.**—We have now to trace whence comes the fat that is found in this form of degeneration, and how. It is evident that in a number of instances—such as the production of milk and sebum—fatty degeneration is a truly physiological change, which is subservient to health when active, while its derangement or cessation constitutes disease. In other cases the process is essentially pathological, as, for example, in fatty degeneration of the heart and white softening of the brain; the functional activity of the part being impaired, or so abolished that the name of *necrobiosis*, or death-in-life, has been given to the condition. The fatty change in the two instances is, however, manifestly one and the same. The condition known as fatty degeneration had long been described, and it has always been a favourite subject with pathologists to discover its nature and its cause. It was generally assumed that the fatty matter present was introduced from without, being deposited from the blood as morbid material in place of the pre-existing tissues which were absorbed. Modern research has demonstrated that this is not so, and that fat is derived from a molecular change in the tissue or textures in which it is found. The subject is one of immense importance, involving the whole field of pathology; and it is but right to state that our acquaintance with the true nature of the process is almost entirely due to the investigations of Dr., now Sir Richard, Quain, which were published in the *Medico-Chirurgical Transactions* for 1850, and with reference to which Sir James Paget has remarked: 'Dr. Quain has candidly referred to many previous observers by whom similar changes were recognised; but the honour of the full proof, and of the right use of it, belongs to himself alone.'<sup>1</sup> Quain's conclusion, as then stated, was that the molecular fatty matter in the degene-

rated fibre was the result of a chemical or physical change in the composition of the tissue, occurring independently of those processes which we call vital. The arguments which he adduced in support of this view were the following: (1) That in the formation of the substance known as adipocere from albuminous material after death, the places of the muscular fibres, blood-vessels, and nerves are occupied by fatty matter which could not have existed in them as such during life. (2) That a true fatty degeneration may be artificially produced *post mortem*. (3) That masses of albuminous material deprived of nutrition in any part of the body, or the centre of non-vascular structures such as tubercle, undergo fatty degeneration to a marked degree. (4) That the circumstances under which fatty degeneration occurs in the living body exhibit impairment of general and local nutrition, such as blood-disorder, or disease of the nutrient vessels.

More than twenty years later (1871) these conclusions were experimentally confirmed in the living animal by the investigations of Professors Bauer and Voit, of Munich. On administering phosphorus to a starving dog, in which the amount of nitrogen (urea) daily excreted had become constant, these experimenters found that the amount of the excretion was thereby increased threefold; that this nitrogen was derived from the albumen of the tissues and not of the blood; and at the same time that three times the normal amount of oil had accumulated in the viscera. This oil could have its origin only in the transformed or decomposed albumen of the organism; the other product being the urea which had been excreted. The same results have been observed in poisoning by phosphorus in the human body.

Many other instances of the formation of a fatty from a nitrogenous body might be adduced if necessary, such as the ripening of cheese; the increased flow of milk on a meat diet; the formation of wax by bees from sugar and albumen; the production of fatty acids and their allies from proteid compounds in the process of pancreatic digestion; the increase of oil in olives by keeping; and the development of a rancid oil in the flake of salmon under similar circumstances. The numerous instances just adduced combine to strengthen the position—which was, however, sufficiently established by Sir Richard Quain—that in true fatty degeneration, the nitrogenous material of the tissues themselves, and not the blood, must be considered the source of the oily matter. It has been said that the circumstances under which fatty degeneration occurs are further confirmatory evidence in the same direction. These must now be considered.

**CONDITIONS OF OCCURRENCE.**—The circumstances under which fatty degeneration occurs are either such as affect the nutrition

<sup>1</sup> *Lectures on Surgical Pathology*, 1st ed. vol. i. p. 107, note.

of the whole system generally, or of a given organ, or portion of it, specially.

*General.*—When the amount of blood in the body is quickly reduced—for example, by severe but not actually sudden hæmorrhage, and in anæmia—death may occur from fatty degeneration of the heart, the voluntary muscles and the other viscera being likewise, but less seriously, affected. Again, general fatty degeneration is frequently due to depraved quality of blood, and especially to the presence in it of certain poisons, such as phosphorus, arsenic, antimony, and the more complex animal-poisons of the acute specific fevers.

*Local.*—Disease of the nutrient artery of a part is the morbid condition most frequently associated with localised fatty degeneration. A good instance of this is furnished by fatty degeneration of patches of the muscular tissue of the heart corresponding with degeneration, obstruction, or compression of a branch of a coronary artery. Another excellent example of the same is white softening of the brain from vascular degeneration. This is analogous to what occurs in dry gangrene, with the exception that decomposition takes place in the latter, probably from exposure to air.

*SUMMARY.*—When we review the circumstances under which fatty degeneration is found to occur, we discover that the condition that is common to them all is interference with nutrition, and especially with the process of oxidation. The red corpuscles are believed to be primarily affected in phosphorus-poisoning; they are numerically reduced in continued hæmorrhage; and they do not reach the tissues in sufficient numbers when the vessels are obstructed, or otherwise diseased. In the cases of the hypertrophied uterus and heart, of the placenta, and perhaps of the corpus luteum, the degeneration is probably due to the decline or cessation of functional activity, and the consequent decrease in the blood-supply to the large mass of protoplasmic structures.

With respect to the *intimate* or *essential* nature of fatty degeneration, it may be stated as highly probable, as far as our present knowledge extends, that the metabolism or decomposition that is constantly going on in living protoplasm is not simple or immediate; but that a primary decomposition occurs of albuminous substances into urea (or its allies) and fat, and a further or secondary decomposition of the fat into carbonic acid and water. If the amount of oxygen furnished by the blood is deficient, whether absolutely or relatively, the primary decomposition of the protoplasm alone may be effected; and the secondary decomposition, or the oxidation of fat into carbonic acid and water, may not occur. The result therefore of an absolute or relative deficiency of oxygen in protoplasmic tissues will be the accumulation of fat within them.

*EFFECTS.*—The physical effects of fatty degeneration of a tissue have been already described under the head of physical characters, being chiefly—change of colour, diminished consistence and resisting power, softening, rupture, dilatation and excavation, and alteration of size. The chief physiological effect is diminished functional power or activity, which is especially marked in muscular parts such as the heart, and in secreting organs, including the kidneys.

*TREATMENT.*—The subject of the treatment of fatty degeneration will be found discussed under the head of the diseases of the several organs which it may affect.

J. MITCHELL BRUCE.

**FATTY INFILTRATION.**—Fat in the form of oily particles is found to be present in excess in the cells of various secreting organs, constituting fatty infiltration. Thus it appears in the epithelium of the intestinal mucous membrane during digestion; in the cells of the liver and biliary passages; and in the kidneys of certain animals—for instance, the cat. When this condition becomes permanent it constitutes a disease. Glandular organs thus affected, as in the case of the liver, assume a buff or yellow colour, and they become softer and more friable than normal; whilst microscopically their cells are found to contain one or more large well-defined oil-globules, which tend to coalesce and occupy the cell. The quantity of oily matter in the cells may, however, vary from time to time, and the infiltration may be either of a transient or of a permanent character. In the one case, the function of the organ may not be materially interfered with; in the other case, the action of the cells may be so far affected as greatly to impair the secreting functions of the organ.

The causes of fatty infiltration are of two kinds—general and local. First, the *general* causes are (a) a superabundance of fatty matters in the blood, such as occurs in persons who indulge in rich food and in beverages containing alcohol; (b) imperfect oxidation, as in chronic tuberculosis of the lungs; and (c), according to some observers, the metastasis of fatty deposits from one part of the body to another.

Secondly, with respect to the *local* causes of fatty infiltration, one is a peculiar affinity or selective power of the cells of certain tissues, by virtue of which they incorporate with their substance oily or fatty matter. This facility has been explained, in the case of the cells of the liver and of the passages traversed by bile, by the presence of that fluid, which, as Virchow and others describe, is a powerfully determining cause of the infiltration of fat into protoplasm. Another local cause of the accumulation of oil in cells is their inactivity or imperfect power of

eliminating it, as is found to occur in the cartilages of the aged, and in inactive or unused muscles.

It must be said here, with respect to the appearance of fat in the last-named situations, that though, in some cases, it is derived unquestionably from the fat contained in the blood or chyle, it may in other instances be derived from the disintegration of the protein elements of the tissues. This subject, however, will be found discussed in the article upon FATTY DEGENERATION.

RICHARD QUAIN.

**FATUITY** (*fatuus*, silly).—Mental imbecility. See IMBECILITY.

**FAUCES, Diseases of.**—See THROAT, Diseases of.

**FAVUS** (Lat. *a honeycomb*).—SYNON.: *Tinea favosa*; *Dermato-mycosis achorina*; Honeycomb Ringworm; Fr. *Teigne faveuse*; Ger. *Erbgrind*.

**DEFINITION.**—A chronic, contagious, parasitic disease of the skin of man and some animals, caused by the growth of the Achorion fungus (discovered by Schönlein in 1839), and characterised by the formation of peculiar yellow crusts, and by the occurrence of inflammatory and often atrophic changes.

**ETIOLOGY.**—Favus is somewhat less contagious than ordinary ringworm, and individual predisposition has a marked influence in its spread. The young are most prone to attack, and children living amidst sordid surroundings. Propagation occurs by direct contact and inoculation, and transmission of the complaint has been observed to and from mice, to the rat, cat, dog, fowl, guinea-pig, rabbit, horse, and ox. In England, where it is uncommon, favus is seen mostly in foreign immigrants; but it occurs more frequently in Edinburgh and Glasgow. It is comparatively common in the southern provinces of France and Italy, and amongst the male Jewish population of Russia, Poland, Galizia, and the Levant; the Mohammedans of Turkey, Asia Minor, Syria, Persia, Egypt, Algiers, and Morocco; also in parts of India, China, and Brazil.

**SYMPTOMS.**—The fungus develops beneath the upper layers of the epidermis, around the hair, setting up some inflammation, and presently appears as a little yellowish elevation. In some weeks the latter forms a characteristic, sulphur-yellow, sub-epidermic, lentil-sized, umbilicated disc (the *godet favique*, *scutulum*, *favus*), lying in a red, moist, cup-like bed fashioned by the pressure of the growth, and traversed by a hair. Gradually the fungus invades the hair-sheaths and then the hair, growing perhaps three or four centimetres above the level of implantation. The bulb usually remains free, and indeed the hair in favus is not

invaded so quickly or abundantly as in trichophytosis, and, as a rule, is not nearly so disintegrated. The hairs become lustreless, dusty-looking, loosened, and finally atrophied and distorted. A characteristic mouldy smell is observable, which must be carefully distinguished from the stale scent of old dried-up pus. Favus may attack hairy regions, non-hairy parts, and the nails; but the scalp is far the most important site affected, on account of the intractability of the disease there, and the ensuing disfigurement. The characteristic cupuliform discs may attain to a diameter of about half an inch or more. They may remain isolated and disseminated, or be aggregated and more or less confluent. As they age, the colour fades or darkens by the admixture of exudations, and the consistence becomes more friable. They tend to lose their characteristic aspect, crumble and fall, to be replaced by others more or less imperfect. In course of time most irregular and atypical changes result from the effects of scratching and excoriation, and from pustulation. Secondary glandular inflammation can then arise. Finally, a disfiguring baldness may result; and as this occurs, the disease gradually dies out, but only after many years.

Regions of the trunk, extremities, or face, commonly spoken of as non-hairy, may be attacked primarily or often secondarily to the scalp. Here the favi are surrounded by inflammatory halos, or preceded by a ring-worm-like eruption.

One or several nails of the hands, or even of the feet, may be inoculated, and then the favi form beneath, and gradually erode the nail. At the same time the nutrition of the latter is much impaired, and the structure disorganised.

**DESCRIPTION OF THE FUNGUS.**—The mycelium is composed of flexuous, ramified, tubular, partitioned, and articulated filaments. When fructifying, the tubes are charged with granules, with nuclei, and with perfect nucleated gonidia or spores. Both the mycelium and the abundant gonidia are characterised by a great diversity of size and form. The gonidia are mostly rounded and oval, and may be isolated or united in chains. It is now established that the fungus can be distinguished in cultures; and when inoculated from animal to animal, and to man, it always gives rise to the characteristic clinical appearances. The *godets* consist of mycelial threads branching towards the central part, and then terminating in chains of gonidia and granules.

**DIAGNOSIS.**—In the various forms of favus where at some time or other more or less well-marked *godets* are observable, the diagnosis is clear; and the peculiar smell, the persistence of the malady, the condition of the hairs, and the occurrence of the disease in other members of the family, will attract

attention. In old-standing cases obscured by age or complications it is necessary to search thoroughly for the fungus, and to bear in mind the advice to be always on one's guard in the presence of a persistent, rebellious scalp-affection, simulating psoriasis, eczema, seborrhœa, and the *folliculites décalvantes*.

**TREATMENT.**—Appropriate measures are necessary to counteract any disturbance of the general health that may be present in favus. At the outset all favic and other crusts and accumulations should be thoroughly removed by the usual softening and cleansing methods, and the hair should be completely cut away, and kept so. Mild antiseptic dressings may then be applied, to subdue any inflammation, or heal excoriated surfaces. This accomplished, the further treatment is nearly identical with that for ordinary ring-worm (*see* TINEA TONSURANS). Cases are often most rebellious. Favus of non-hairy parts is much more amenable. When the nails are affected it is necessary to remove mechanically as much of the diseased part as possible, and then thoroughly rub in, or keep the part soaked with, parasitocides.

T. COLCOTT FOX.

### FEBRICULA (*febricula*, slight fever).—

**SYNON.**: Fr. *Fébricule*; Ger. *Febricula*.

**DEFINITION.**—Simple fever, of one (*ephemera*) or not more than a few days' duration; not preceded by any one known invariable antecedent; and not attended by any one definite organic lesion.

It may well be doubted, however, whether such a thing as simple fever, in the strict sense of the term, exists; anyhow it must be one of the rarer forms of disease. The conditions which, from our necessarily imperfect knowledge, it is convenient to call febricula are numerous and of great practical importance. They may be somewhat roughly grouped as follows:—

1. Abortive or incomplete forms of some one or other of the specific continued fevers—namely, typhus, typhoid, or relapsing. The writer's own experience leaves no doubt in his mind that such irregular forms are met with during epidemics of these diseases.

2. Instances of some of the exanthemata, especially scarlatina and modified variola, in which the usual rash is either absent or so slight or brief as to pass unnoticed.

3. Intermittent fevers, in which for some reason or other the paroxysms do not recur, or only at uncertain and distant intervals.

4. Cases in which the local symptoms usually attending certain forms of fever are very slight or very obscure, and therefore difficult, perhaps impossible, to detect. Instances of this occur in connexion with tonsillitis, erysipelas, rheumatic fever, influenza, and tubercular disease.

5. Cases in which considerable febrile

movement is present during the development of the primary as well as of the secondary symptoms of syphilis, and of which it is not easy to ascertain the real cause.

6. Fever as the consequence of exposure to a high external temperature—for instance, the *febris ardens* of tropical climates (Murchison); and of violent and prolonged muscular exertion.

7. Fever as the consequence of irritation of any organ or tissue, such as of the stomach by indigestible matter, of the colon by scybala; or of catarrh of a mucous surface—for example, urethral fever.

8. Certain ill-understood but not uncommon disorders of nervous centres, cerebral, spinal, or sympathetic, which are often followed by febrile movement.

**ANATOMICAL CHARACTERS.**—Fever, however caused, which runs high, produces congestions and tissue-changes in the viscera, especially in the lungs; but, in view of the short duration of febricula, it would in most cases be impossible to decide whether any pathological changes found after death were the consequence or the cause of the febrile movement. It is possible, but not yet proved, that there may exist some contagion capable of producing febricula, and febricula only.

**SYMPTOMS.**—Febricula is characterized by a rise of temperature, rarely exceeding 102.5° F., but sometimes, especially in cases due to exposure to heat, reaching 105° F. or even higher, although only for a short time. The access of fever may be gradual, or marked by slight rigors; and some or all of the common clinical symptoms of fever may be present in varying proportion and in greater or less degree, such as—general malaise; dry skin; frequent pulse, amounting to 100 or 120 per minute; tongue furred, with a more or less distinct, central, dry, reddish-brown streak; thirst, loss of appetite, and nausea; constipation; scanty, high-coloured urine; and headache, intolerance of light, slight deafness, restlessness, sleeplessness, and slight delirium at night.

**DIAGNOSIS.**—The diagnosis of febricula rests upon the exclusion of all the other recognised kinds of idiopathic or of symptomatic fever. As a matter of practical diagnosis at the bedside, almost every disease attended by rise of temperature is now and then, at its outset, mistaken for febricula.

**PROGNOSIS.**—The prognosis depends upon the degree and duration of the pyrexia, but in this country is almost always favourable.

**TREATMENT.**—In the absence of any special indication, rest in bed for a day or two, liquid food until the desire for solids returns, and, if constipation be a marked feature of the case, a moderate dose of some mild purgative, will be sufficient. It is, however, always prudent to remember that what seems to be febricula may be the beginning of some serious and perhaps highly infectious disease.

Cooling drinks, such as citrate of potassium in effervescence, liquor ammonii acetatis with a little spirit of nitrous ether; or nitrohydrochloric or dilute nitric acid (one drachm to a pint of water) with some fresh lemon-juice added, may be given according as the one or the other is grateful to the patient. Anything like active treatment, except sponging the skin or the use of the cold or tepid bath in cases of heat-fever, is unnecessary, rarely does good, and is almost always positively injurious.

J. ANDREW.

**FEBRIFUGES** (*febris*, a fever; and *fugo*, I drive away).—SYNON.: Antipyretics.

**DEFINITION.**—External applications or internal remedies which tend to lower the bodily temperature, when it has been abnormally raised by the processes of fever.

**ENUMERATION.**—The principal febrifuges, given in the order of their activity, are:—Cold Baths; Cold Affusion or Wet Pack; Alcohol; Diaphoretics; Salicylic Acid and its Salts, Quinine, Digitalis, and Aconite; Salicin, Phenazone, Acetanilide; and Water and Diluents generally.

**ACTION.**—Following Professor Binz, we may divide febrifuges into two classes:—(1) those which directly withdraw heat from the fevered organism; and (2) those which lessen its production.

1. In the first division we must give the foremost place to cold baths, the cold pack, and cold sponging, which powerfully abstract caloric from the surface of the body, and rapidly cool down the blood. Diaphoretics and alcohol act more feebly in the same direction, by dilating the cutaneous arterioles, and thus allowing the mass of the circulating fluid to be effectually exposed to the chilling influence of the air. See COLD, Therapeutics of; and DIAPHORETICS.

2. Secondly, we have to consider those drugs which actually check the febrile condition itself, by diminishing oxidation, or by lessening the rapidity of the circulation through those parts of the body in which the increased tissue-change is taking place. It is probable that we must explain the antipyretic properties of such drugs as quinine by their specific influence over the germs on which malaria depends; and perhaps the progress of science may enable us in the near future to nip febrile processes in the bud, by paralysing or destroying the bacilli which may start the chain of morbid processes which raise the temperature and depress the powers of life. The free use of water tends to promote excretion, and thus to remove the products of oxidation. Blisters possibly act by causing contraction of the vessels in the inflamed part.

**USES.**—Antipyretic treatment is not much adopted in this country as a matter of routine, holding as we do that temperatures raised within certain limits are not *per se*

elements of danger, and that, even although we may effectually cool down our patient, the progress of the disease may go on quite unchecked. But prolonged fever causes excessive tissue-change and fatty degeneration of the heart; and when the thermometer registers 105° F., and still tends upwards, we know that dangerous limits are reached, and that as a rule life is not long sustained above 107° F. It then becomes our duty to interfere; and this is best done by plunging our patient into a bath at 95°, and gradually cooling it down to 65°. When the temperature descends to within 4° or 5° of the normal we remove him to bed, remembering the dilatation of vessels which must follow the contracting effect of cold, and the consequent cooling process which must continue to go on. Here, as in all febrile conditions, the thermometer is our surest guide, and we must be directed by it as to when to resume the treatment, for frequent repetition may be needed, and on the Continent as many as 200 baths have been given in the course of a single illness. Along with this the Germans combine the use of large doses of quinine; but, notwithstanding the marked tolerance of the drug under pyrexial conditions, the danger of perilous depression from such free medication is no imaginary one; and, putting ague apart, we find this valuable drug most beneficial in such fevers as seem to owe their origin to septic poisoning. Digitalis is not a powerful antipyretic, and in large doses is too disturbing to the heart, and too apt to produce gastric derangement, to inspire much confidence; but phenazone causes profuse perspiration, and is much used in febrile disorders on account of its certain and rapid action, and acetanilide is effectual in smaller doses. The influence of salicylic acid over acute rheumatism is remarkable, as it seldom fails to reduce temperature and relieve pain in forty-eight hours; but in other feverish conditions its beneficial action is by no means so well-marked. Salicin also has numerous advocates, who prefer it to the salicylates as being less depressing to the circulation. Iron is of value in erysipelas, and exerts some controlling power over acute rheumatism. Aconite and diaphoretics are of undoubted service in aiding the defervescence of some of the minor febrile disorders.

R. FARQUHARSON.

**FEIGNED DISEASES.**—No insignificant part of the real difficulty in the practice of our profession depends on what we may call feigned diseases. The art of diagnosis consists in the power of recognising morbid conditions with skill and promptitude; and in proportion to the natural sharpness and well-digested experience of the medical man is his success in the discrimination of one symptom from another which resembles it more or less superficially. Many disorders

possess a strong family likeness in their very early stages, whilst others may prove deceptive throughout their whole course; and if to this we add the efforts at deception occasionally resorted to by impostors, we see the caution which must of necessity be adopted by those who exercise their calling within wide limits. In considering, therefore, the subject of feigned diseases, a greater amount of order may attend our studies if we adopt the following simple classification:—

1. Those diseases which naturally resemble one another, and in the deception attending the diagnosis of which the patient has no share.

2. Those diseases which are also difficult of diagnosis, but in which the patient involuntarily deceives under some morbid nervous impulse.

3. Cases in which the patient sets himself deliberately and elaborately to deceive those around him.

1. Under this heading we may perhaps include the exanthemata and other acute feverish affections, which are confessedly difficult of diagnosis before the eruption or other marked points of difference are fully established. Important though it may be, in the case of public schools or large bodies of men, to act promptly in the face of such an emergency, the medical man will often feel himself compelled to postpone his decision; but he should at the same time act on the defensive by the timely exercise of quarantine and hygienic precautions. Some diseases, again, are difficult to distinguish from one another, even after their prodromata have passed away, and among these we may include small-pox and pustular syphilis, which occasionally cause more than a shade of suspicion to pass over the mind; whilst mild variola and severe varicella must always have too many points in common to render them otherwise than stumbling-blocks even to the initiated. Various forms of roseola may closely simulate measles; scabies is often hardly to be picked out from amongst the eruptions which its irritation causes; whilst throat-affections may apparently overlap each other and engender the idea of diphtheria where nothing more than superficial or aphthous ulceration really exists. But it is when the ailment under which our patient labours resembles something else during its whole career that mistakes are naturally most likely to arise. We are frequently shocked with some scandal in which the innocent victim of brain- or other organic disease has been consigned to a police-cell, and where the plea of drunkenness has been attempted to be sustained by the guardians of the public peace. So difficult is it to make a really trustworthy diagnosis between the coma of alcohol, of uræmia, of opium, and of certain apoplectic conditions, that the truly cautious and well-informed practitioner would prefer

not to attempt to do so offhand. It is impossible to lay down any general rules, but we may remember that alcohol in poisonous doses lowers the temperature and dilates the pupil; that in uræmia an examination of the urine will put us in the right track; whereas opium will produce a contracted pupil; and in cerebral hæmorrhage some elevation of the body-heat may not improbably be observed.

But all these points may fail us from time to time, and we had best act at all times as though the case were really a serious one, and requiring treatment by all the best resources of the medical art.

A very little consideration will enable every experienced practitioner to recall other instances of this sort of natural mimicry: of the difficulty he must often have experienced in deciding between syphilitic and other brain-affections; of the close affinity between pulmonary consumption and dilatation of the bronchial tubes; of the resemblance between specific and malignant ulcerations; between various diseases of the testicle, the bladder, and the stomach, respectively. All these form part of the regular teaching of medicine and surgery, and will be found treated of elsewhere.

2. We must now consider the cases in which diseases are feigned, not by the direct action of the patient himself, but because he is unable to resist the vagaries of his weak and excitable nervous system.

Problems of the greatest complexity and difficulty are here presented to the medical man, and require for their due solution much tact and experience. Functional affections so closely simulate organic disease under these circumstances that suspicion is often completely disarmed, and treatment adopted the very opposite of that which would most probably prove curative. Hysteria, in its protean forms, supplies the greater number of these cases, and may very closely simulate a large variety, more especially, of neurotic conditions (*see* HYSTERIA). Paraplegia, incontinence of urine, joint-affections—in short, almost any disease which does not admit of palpable objective demonstration—may thus be feigned, and very severe treatment may even be adopted under the belief that real organic changes have to be met by the usually appropriate remedies. It is only necessary for us to refer thus briefly to these perplexing cases here; but it must always remain an interesting problem as to how the mechanism of causation here works, so to speak, and whether the patient actually suffers the acute pain of which she complains so forcibly. Sympathy, as we well know, however, is quite thrown away when dealing with these persons; and apparent roughness, with nerve tonics, and mental discipline, will often effect a cure, when the most elaborate combinations of other drugs ignominiously fail. A sudden shock, the pressure of poverty, or the absolute necessity for immediate exertion, will

often effectually and permanently arouse the bedridden hypochondriac of many years, and restore him to his friends as a useful member of society; and we need never despair of success even under apparently hopeless circumstances. And although in minor measure the hypochondriac may fancy that every organ in succession is the seat of disease, and may even succeed in thus imposing on the unwary, the experienced practitioner will speedily detect the fiction, and be able to relieve the unhappy sufferer from the weight of his woes. See NEURASTHENIA.

But let it not be forgotten that 'expectant attention,' or the constant direction of the mind to the supposed morbid condition of any particular organ, may actually catch the unconscious deceiver in his own net by converting mere functional disturbance into organic disease.

3. To the third division of our subject can the term Feigned Diseases perhaps alone strictly be applied. Here we are met face to face with deliberate and premeditated imposture; and there is nothing for it but to match our own wits against those of the deceiver, and to thwart his native cunning by the superior sharpness of science. Now, there is nothing in the history of medicine more remarkable than the elaborate expedients adopted, and the amount of actual discomfort and suffering endured, by persons who have been desirous of escaping military or other duty. The exhaustive works of Gavin and Marshall, and Boisseau and others, give us details no less ingenious than interesting of these devices; but it is curious to note in how limited a range the more traditional modes of imposture seem to run, and how the same old stories are made to do duty over and over again. Thus we read of blindness, and deafness, and epilepsy, and paralysis being carefully imitated, and can hardly withhold our admiration from the astonishing tenacity with which the apparent symptoms were duly maintained. Incontinence of urine, dysentery, hæmoptysis, jaundice, and insanity were among the most favourite rôles in the repertoire; and ingenious as were the preparations for duly sustaining the part, no less ingenious were the means for detection, which usually proved successful. However carefully the impostor had studied his character, some little point was usually omitted. The yellow conjunctiva of jaundice can hardly be feigned; the incontinence of urine was generally found to be attended by an expulsive effort; the blood apparently proceeding from the lungs was by no means intimately mixed with the pulmonary mucus; the blindness, or the deafness, or the paralysis was not proof against some sudden shock or mental impression. Most of the cases so carefully described by writers on military medicine are now mere matter of history, and are hardly likely to

occur again. And the reasons for this are twofold. First: the inducement for deception is practically gone. In former days, when the soldier's pay was small and his hygienic condition bad, discharge from the service as an invalid was eagerly prized as a means of escaping irksome duty; but matters have greatly changed for the better of late years. Not only are the emolument and the comfort of our army vastly increased, but short service and the reserve enable soldiers to retire early into civil life; whilst the abolition of bounty has removed the principal pecuniary inducement for frequent desertion and re-enlistment. It is now found much easier for a man simply to desert than to go through any elaborate process for the personification of disease. Again, the savage process of forcible impressment for naval service was naturally productive of many attempts to escape from the hard work and ferocious discipline of our men-of-war. Secondly: the progress of science and improved means of diagnosis have rendered the task of the impostor difficult, if not well-nigh hopeless. Feigned blindness can hardly resist the test of the ophthalmoscope; electricity will clear up many apparently anomalous nervous symptoms; the stethoscope and the sphygmograph will tell us the real condition of the heart; and careful observation will detect the rougher attempts to deceive. Again, malingering may often be exposed by examination under chloroform or ether. We are not likely now to be deceived by a piece of liver tied to the breast to simulate cancer, or by an artificial nasal polypus; but Mr. Gould records in the *Lancet* the case of a young woman sent to hospital for double buccal tumour, in whom on examination the swellings were found to consist of tightly wound balls of thread forcibly inserted between the teeth and the cheek. Mr. Anderson, of Richmond, fairly capped this in a subsequent number by narrating the vagaries of a schoolgirl who had produced a state bordering on gangrene of the hand by a ligature of ribbon resolutely applied to the forearm; and it is evident that although skin-diseases and ulcerations may be made or kept up by local applications, we only require a suspicion to cross our minds to put us on the right track for discovery.

We are not, however, to suppose that all attempts at deception have finally passed away, that feigned diseases are now things of the past. Anyone whose practice lies among prisoners, or soldiers, or schoolboys, or the sufferers from railway accidents, will very soon be convinced to the contrary (see RAILWAY INJURIES). Experience, however, will speedily show him who the schemers are, and enable him to circumvent their endeavours; and the range of symptoms simulated will soon be found to be singularly narrow. Subjective sensations are, of course,

very difficult to detect; and if a headache, or pain in the back or leg or arm, be complained of by the sufferer, real or assumed, we may often find it best for our own reputation to give him the benefit of the doubt. Whilst proceeding, therefore, with due caution, the practitioner must endeavour to hold the balance between an excess of suspicion and a too credulous attitude, remembering that the good of society and of the public service must be fairly considered, whilst all care must be taken not to confound the innocent with the guilty in dealing with disorders which ingenuity has occasionally been enabled to feign. ROBERT FARQUHARSON.

**FEIGNED INSANITY.**—See INSANITY, Varieties of.

**FESTER.**—A superficial suppuration, resulting from irritation of the skin; the pus being developed in vesicles of irregular figure and extent. The suppurating inflammation caused by a thorn or splinter of wood forced into the flesh is a common example of a fester.

**FEVER** (*ferveo*, I am hot).—SYNON.: Pyrexia; Fr. *Fièvre*; Ger. *Fieber*.

**DEFINITION.**—One of the most remarkable facts in connexion with disease is the rise of temperature which is attendant upon almost every disturbance to which the body is subjected. The rise of the temperature of the body, when it attains a certain height, and lasts a certain time, is called Fever; and is accompanied by derangement of function, attributable to the febrile condition itself, and in a measure independent of the initial cause.

**GENERAL CONSIDERATIONS.**—Fever plays so important a part in acute disease generally, is accepted so universally as a mark of the severity of the disease, and so often presents itself as apparently the chief antagonist with which the physician or surgeon has to contend, that the attempt to penetrate the secret of its essential nature has always been a favourite task, and every school in every age has had its theory of the febrile process. It is only, however, within comparatively few years that exact measurement of the body-heat by the clinical thermometer, combined with chemical examination of the various excretions at different temperatures, and aided by the experimental method of investigation, has furnished the data for such a theory (see THERMOMETER). A minute description of fever in the abstract, distinguishing, as would be required, between phenomena proper to fever and phenomena due to the condition or lesion on which the fever depended, would be lengthy, and so crowded with qualifications and exceptions as to be vague and unsatisfactory. The attempt, indeed, would have a more radical defect. Either some variety of fever must be taken as a type to which other forms are referred, which is vicious in principle; or all the phenomena of all febrile

conditions must be enumerated and classified, which would confound the accidental with the essential, and would result in a heterogeneous collection of facts without due relation among themselves.

A mere outline therefore will be given of the principal deviations from normal functional action observed in fever, and the space set apart for the subject will be reserved chiefly for an exposition of what is known of the nature of the febrile process.

**DESCRIPTION.**—In every attack of fever there are traceable the three stages of invasion, domination, and decline, with or without an antecedent period of incubation. They may all be run through in the course of a few hours, as in a paroxysm of ague, or they may extend over weeks.

The period of invasion is characterised by a rising internal temperature, while the surface may remain cold and pale, the patient feeling chilly and suffering from rigors or shivering; the pulse is frequent, but generally small and long, from contraction of the arteries. During the dominance of fever the temperature remains high, the skin is hot, and the shiverings are replaced by a subjective sense of heat; the pulse is now large and bounding from relaxation of the arterial wall. The decline is indicated by a falling temperature, a softer and less frequent pulse, and by a return towards normal conditions generally; it may be initiated or accompanied by a critical sweat or other evacuation. Death may take place at any period of the disease.

Taking the temperature as the index of the duration and character of each stage, we may find it in the first stage rising abruptly or gradually, continuously or with remissions. If the invasion extends over several days as in enteric fever, nocturnal exacerbations and morning remissions are, as a rule, observed. A rapid rise of temperature is usually continuous, or nearly so. When the opportunity occurs of making the observation, as in intermittent or relapsing fever, or when fever is experimentally induced in animals, or in man by surgical operation or accidental septic inoculation, the increased heat is found to be the initial phenomenon, preceding the rigors and all other symptoms.

The end of the period of invasion, and the setting in of the stage of dominance, is more distinctly marked by the change in the character of the pulse, and by the determination of blood to the surface, together with the substitution of the subjective sensation of heat for that of cold, than by the thermometer.

During the dominant stage the temperature remains at, or oscillates about, a given point, and the fever is considered to be moderate if the morning temperature is 102° F. or under, and the evening not above 103°; to be high when it ranges between 103° the morning and 104° in the evening; and to be

severe when these limits are exceeded; while, with rare exceptions, a temperature of  $106^{\circ}$  indicates great danger. As the stage advances, the heat may gradually rise or fall; the oscillations being slight or considerable, and at times irregular and extreme. Except when the fever is due to local inflammation, or to continual entry into the blood of morbid particles or fluids, the duration of the dominance is usually in proportion to the time occupied by the invasion.

The decline again is generally abrupt, and has the character of a *crisis* when the invasion has been rapid, and is protracted when it has been gradual.

A fatal termination may be ushered in by hyperpyrexia; more commonly the temperature falls below the normal point and there is collapse.

The pulse is always increased in frequency by fever, but while during the height of the disease there is usually some relation between the body-heat and the pulse-rate, the pulse is often extremely frequent before the temperature has reached its height during the invasion, and it does not in all cases fall with it *pari passu* in the decline. The different stages are marked rather by differences in the character of the pulse than in its rapidity; during the period of invasion the arteries are more or less in spasm, and the pulse is small and long; during the dominance, with certain exceptions, the arterial walls relax, the vessels are large, and the pulse sudden, short, and dicrotous; as the fever declines, the arteries are still further relaxed, but the action of the heart is less powerful, so that the pulse becomes softer.

Respiration is frequent, following as a rule the pulse; the amount of carbonic acid expired is greatly increased.

The tongue is generally more or less furred, its appearance varying with the degree and kind of fever and with its cause. It becomes brown and dry, or unnaturally red, in protracted and adynamic fever, when the teeth and lips may also be coated with *sordes*. There are almost always thirst and loss of appetite. The bowels are usually confined.

The secretions are all more or less modified. The perspiration may be greatly increased, as in acute rheumatism; more commonly it is checked, causing the skin to be dry and burning. The amount of urine will vary to a certain extent inversely with the amount of perspiration; but the tendency is to increase, and the solid organic matters—urea and other nitrogenised substances—are always considerably augmented in quantity. The chloride of sodium, on the contrary, is diminished.

The characteristic nervous phenomena of the stage of invasion are rigors, which may be slight, and represented only by shivering or chilly sensations, or, on the other hand, may be intensified to convulsion. Severe

headache is more common at this period than in the later stage, and there is usually considerable depression. When the fever has reached its height the rigors will have ceased, and there may be little or much delirium according to the severity of the attack, the idiosyncrasy of the patient, or the kind of disease giving rise to the fever.

**PATHOLOGY.**—The description of the febrile state has been abbreviated in order to leave place for a discussion of the nature and cause of the febrile process.

This will be facilitated by a brief reference to the production and regulation of the heat of the body in health, and would be comparatively easy had physiologists arrived at a complete and satisfactory solution of this problem. In the normal state the main source of animal heat is blood- and tissue-combustion. It may perhaps be well to say here once for all that the terms 'combustion' and 'oxidation' are employed as conveniently short expressions for the disintegration of complex chemical and structural molecules by which heat is liberated, and not to convey the idea of direct combination of oxygen with the carbon and hydrogen of the blood and tissues. Another very slight and unimportant cause will be obstructed motion of the blood in the capillaries: of direct conversion of nerve-force into heat we know nothing definite. The interesting and difficult part of the question is that which relates to the regulation of the temperature. It has been found that the changes which evolve heat are most active in muscle, in the nervous structures, and in the abdominal viscera; while in the lungs any combustion which may take place is not more than will counteract the loss of heat by evaporation and by the expired air. The skin, on the other hand, is the great cooling agent; there is little combustion of its structures, and it is continually losing heat by conduction when the surrounding temperature is low, but still more abundantly by evaporation under all conditions of external temperature. Heat is thus abstracted from the blood circulating in, and immediately beneath, the skin. At first sight, then, it would seem that the mechanism by which the temperature is regulated was extremely simple, and that it was to be found in the vaso-motor system of nerves. There being an internal heat-producing mass of tissue, and an external refrigerating surface, in order to raise the temperature, the arterioles of the skin are contracted, shutting off the blood, while those of the deeper structures are relaxed, allowing it to reach them in greater abundance; in this way a double influence is exerted—less heat is lost by the skin, and more is produced in the muscles and other internal parts. Conversely, the temperature would be lowered by flushing the skin with blood—which would thus be exposed to cooling influences—and divert-

ing it from the heat-forming, deep-seated structures. This explanation, however, is inadequate. It is true that the distribution of the blood, superficially or deeply, by means of the vaso-motor nervous system, contributes largely to the regulation of the temperature; but heat-production in muscle or gland is not directly proportionate simply to the amount of blood circulating through it. Tissue-combustion, and consequent evolution of heat, is excited or repressed by cerebro-spinal nerves which do not govern the arteries. The nervous stimulus which excites contraction in muscles determines in them at the same time evolution of heat, and this heat-production under nerve influence may be excited independently of contraction. The nervous system thus intervenes directly in heat-production chiefly by its action on the muscles, as well as indirectly through its influence on the circulation. This is not the place to discuss the question whether there are special thermal nerves and centres, but it seems probable that the muscles as thermogenic and as motor agents are under the control of the same central nervous mechanism.

Taking the increased heat as the characteristic of fever, the first question which arises is whether this is due to increased production of heat, or to diminished loss. While the diminished circulation in the skin, in the early stage, will obviously tend to retain heat within the body, there is now no room for doubt that there is increased heat-production. The temperature rises in spite of profuse perspiration, when, of course, heat is very rapidly lost, as in acute rheumatism, or when perspiration has been induced by jaborandi before a paroxysm of ague (Ringer); and it has been shown by direct experiment that in fever a man raises the temperature of a given quantity of water in which he is immersed more quickly, and to a higher point, than in health (Liebermeister). It is unnecessary to give other proofs or further refutation of the hypotheses which explain the heat of fever solely by diminished escape of heat from the body.

It may further be taken as certain that the immediate cause of the increased generation of heat is increased blood- and tissue-oxidation. This is shown by the increased products of combustion given off in the different excretions. The febrile elevation of temperature is attended at once by increase in the amount of carbonic acid expired. This is more marked during the rise than when the heat has attained its maximum, because the increasing temperature expels the gases of the blood, and the greater rapidity of the circulation sends the blood more freely and quickly through the lungs, and exposes it more to the air. At first there will thus be eliminated not only the carbonic acid formed under the influence of the febrile process, but that which was held in solution by the

cooler blood, and is driven off as its temperature rises; when the expulsion of dissolved carbonic acid is completed, the amount excreted will be diminished by so much, but it still remains larger than at the normal temperature.

A similar indication of increased tissue-combustion is furnished by the urine. The amount of urea is usually absolutely increased, notwithstanding a diminished consumption of nitrogenised food; or if the urea itself is not excreted in larger quantity, there is more nitrogenised waste in other forms. The total of nitrogenised matter contained in the urine is always augmented by fever.

While, however, the products of combustion show that rapid metabolism is going on, it is not clear that it is adequate to account for the whole of the heat evolved; and Dr. Ord has advanced the luminous hypothesis that the energy normally absorbed by the constructive operations taking place in the tissues is set free through the suspension of these processes by fever, and takes the form of heat.

The real difficulty of the problem arises when we inquire what is the cause of the increased tissue-combustion, and especially how the febrile heat is regulated. It has been already stated that the distribution of the blood to the deep structures and organs and to the skin respectively is not a sufficient explanation of the physiological balance of heat; but it might be supposed that the greater rapidity of the circulation in fever, renewing the supply of oxygenated blood within the structures more frequently and more freely, would account for the greater oxidation. The rise of temperature, however, is not in proportion to the flow of blood through the vessels, and hyperpyrexia is often coincident with a failing circulation, the heat of the body, indeed, apparently in some cases actually increasing after death.

Dr. Donald MacAlister has done much to elucidate the entire question in his *Gulstonian Lectures*, by bringing out more distinctly the thermogenic function of the muscles, and the thermotaxic or heat-regulating power of the nervous system, which operates through its influence upon thermogenesis in the muscles on the one hand, and upon the thermolytic action of the skin on the other.

One step towards the solution which may be considered certain is that the nervous system is concerned in the maintenance of the heat of fever. Each disease has its own characteristic range and variations of temperature; and this fact alone, that febrile heat is not vague and irregular, but that there is the substitution of a morbid for a normal balance, is evidence of nervous control. Numerous observed facts and experiments point to the same conclusion.

Another item of positive knowledge obtained by experiment is that pyrexia may be

excited by the introduction into the blood of septic or other matters, which, it is important to note, need not be particulate, but may be diffusible fluid. The increased heat may therefore be independent of capillary embolisms, and of bacteroid or other organisms.

Now in disease or after injuries we have almost always both causes in possible operation—an impression on the nervous system, and the entry of altered organic matters into the blood. In endeavouring to assign prominence to one or other, we have, on the one hand, such facts as the hyperpyrexia of cerebral lesions, which cannot be due to blood-contamination, and, on the other, the teachings of antiseptic surgery, which demonstrate that absorption of putrescent discharges is the great cause of surgical fever. It still remains to be determined whether the presence in the blood of foreign matters gives rise directly to increased activity of oxidation, or whether the poison, as we may call it, produces this result through its action on the nervous system, either by affecting the nerve-centres themselves, or by producing irritation in the capillaries, which is carried to the nerve-centres, and reflected along efferent nerves. In the present state of our knowledge this question cannot be definitely settled.

If a theory of the febrile process is to be formed, it must be based upon a theory of the relation between the nervous system and the processes of nutrition and oxidation, and especially the latter. Numerous facts of disease and of experiment point to the conclusion that the circulation of duly oxygenated blood through the tissues at the usual rate would, without some check to oxidation, result in more rapid tissue-change, and the production of a higher temperature than the established norm. The restraining power is supplied by the nervous system, the loss of this influence being illustrated in hyperpyrexia. The mode in which the nervous system acts may be represented as being through the tension maintained in the nerve-centres. All nervous actions have the character of phenomena of tension, and the tension generated in the cells is sustained in the nerve-fibres to their peripheral terminations, where they are merged in the structures, and so blended with them that all nutritive and oxidative changes are common to the nerve-endings and the tissues in which they end. If we suppose that the nerve-tension can modify chemical action, as can electrical tension or thermal conditions, and that, *vice versa*, the nutritive and oxidation changes in the tissues can influence the tension of the nervous structures, we can represent to ourselves the interaction between the nervous system and the blood and tissues in the febrile process. When from disease or injury of the great nerve-centres their power of maintaining tension is lessened or abolished,

and their influence proportionately diminished, so that the affinities of the blood and tissues have unrestricted play, the result is hyperoxidation and pyrexia. When, on the other hand, septic matters or other substances are introduced into the blood, acting as ferments or in some other way, they increase oxidation, and directly raise the temperature, overpowering the restraining influence of the nerves until this is reinforced, which may possibly occur through increased evolution of energy, resulting from the increased activity of metamorphosis. We cannot, however, here develop or fully elaborate this hypothesis.

TREATMENT.—The treatment of fever is of course primarily directed to the removal of the cause on which it depends, but together with the measures adapted to this end are usually employed means for the moderation of the febrile process as such, and these may at times take the first place. We can do little more than mention the more important of them, taking first what may be called the general methods, and premising that rest in bed, simple food, &c., are assumed to be understood. Venesection is now scarcely ever practised as a means of combating fever. Purgatives are often useful, as are also diaphoretic and diuretic salines, with abundance of water to drink, either alone or in the form of some agreeable *tisane*. Free action of the secretions, which is the object of these remedies, is of service in removing the increased products of oxidation, the water taken co-operating by acting as a solvent and vehicle; and it is possible that medicines which promote this activity may directly bring down the temperature. When, for example, perspiration has been induced, a coincident fall of temperature may be due more to some change antecedent to the perspiration, than to the loss of heat by transpiration and evaporation.

Of special measures for the reduction of febrile heat when this is becoming dangerous, either from its intensity or duration, the first to be mentioned is the cool or cold bath. This should be resorted to in all cases of hyperpyrexia, from whatever cause; its efficacy, first established in the high temperature of acute rheumatism and enteric fever, has been proved also in cases of septic hyperpyrexia after ovariectomy, and even in injuries to the brain. Here the water may be positively cold. When the bath is employed to control temperature, not dangerous from its height, but from its duration, as in enteric fever, the temperature need not be lower than 70° or 65° F. An ice-cap devised by Mr. Knowsley Thornton, for applying cold of 32° to the entire head, has been found useful in hyperpyrexia following ovariectomy.

Many substances have the property of reducing febrile temperatures when taken in large doses. Among the most powerful are the complex bodies recently introduced, phenazone, acetanilide, exalgin, and others of the

same class. Their influence on febrile heat is undoubted, and it may be exerted favourably in the early stage of sharp fever from whatever cause, but their continued use is attended with danger. The lowering of the temperature may be dearly purchased at the expense of the patient's strength. Another powerful agent often employed to combat fever is quinine. When given for this purpose, it is administered in doses of from ten to twenty or even thirty grains once in twenty-four or forty-eight hours; or three to six grains of the neutral sulphate may be injected under the skin. Salicylic acid has a remarkable influence on the temperature in acute rheumatism, and some effect, though far less marked, in fever from other causes. It may be added that when pericarditis has come on in rheumatic fever, this drug usually altogether fails to influence the temperature. The only other drug which need be specially mentioned is aconite, the mode of action of which is totally different from that of quinine, and of which it may almost be said that it antagonises the fever process rather than reduces temperature; its most marked influence being on the force of the heart and the contraction of the arteries. The opportunity for the manifestation of its powers occurs in the early stage of catarrhal fever, the result of chill. Given in frequent small doses (a drop or two of the tincture every five minutes till twenty minims or half a drachm has been taken) when the temperature is rising, the pulse frequent and hard, with headache and burning skin, the effects are often striking. When a local inflammation is established, it is no longer of much use; and when the fever is protracted, as in enteric fever, or when there is pneumonia, it may be dangerous, from its depressant influence on the heart. See ANTIPIRETICS.

W. H. BROADBENT.

**FIBRILLATION, Muscular.** — A localised quivering or flickering of muscular fibres. See MOTILITY, Disorders of.

**FIBRINOUS CLOT.**—See CLOT.

**FIBRINOUS CONCRETION.**—See CONCRETION.

**FIBROID DEGENERATION.**—A morbid change which consists in the substitution of a tissue somewhat resembling fibrous tissue for other structural elements; some pathologists consider this change to be of the nature of a degeneration. See DEGENERATION; and CIRRHOSIS.

**FIBROID PHTHISIS.**—A name given to certain cases of phthisis in which a considerable development of fibroid tissue occurs in the lungs. See PHTHISIS.

**FIBROMA.**—A form of tumour composed of fibrous tissue. See TUMOURS.

**FIBRO-PLASTIC GROWTH.**—A form of new-growth, composed of fibro-plastic elements. See TUMOURS.

**FICUS UNGUIUM** (*figus*, a fig; *unguis*, a nail).—A disease of the posterior wall of the nail. See NAILS, Diseases of.

**FIDERIS, in Switzerland.**—Chalybeate waters. See MINERAL WATERS.

**FIFTH NERVE, Diseases of.**—The fifth or trifacial nerve (*nervus trigeminus*), the largest of the cranial nerves, consists of a motor and sensory portion, the sensory fibres passing through the Gasserian ganglion, and being distributed to the face and a portion of the head. The motor portion, much the smaller, is physiologically independent of the ganglion, and supplies the pterygoid, masseter, buccinator, and temporal muscles. The two first divisions—the ophthalmic and superior maxillary—are entirely sensory, and proceed from the ganglion. The third or inferior maxillary division proceeds also from the ganglion, but receives besides the whole of the motor root. Lesions of this nerve cause disorders of sensation, motion, and nutrition or secretion, according to the anatomical position and extent of the injury.

The affections of the fifth nerve may be considered in the following order:—

1. **Trifacial Neuralgia.**—Neuralgia may affect one or all of the three divisions of the nerve. It is fully described under the head of tic-douloureux. See TIC-DOULOUREUX.

2. **Trifacial Anæsthesia.**—Anæsthesia of the trigeminus, usually unilateral, may be dependent either upon (a) *central lesion*; or upon (b) *peripheral lesion*.

(a) *Central lesion.*—Hemiplegia from hæmorrhage, softening from thrombosis, tumour, or other coarse affection of the intracranial nervous system, is usually accompanied by some anæsthesia in the district supplied by the trigeminus, arising probably from interference with the integrity of the fibres of origin of the nerve in their central course. The anæsthesia usually occurs on the same side of the body as the paralysis of motion, and therefore opposite to the seat of lesion. This is always the case when the lesion occupies a position above the pons Varolii. In disease of the pons Varolii, however, the loss of sensibility may involve both halves of the face, although it usually affects the same side as that upon which the limbs are paralysed, and opposite that upon which the portio dura and sixth nerves (when either or both of them are involved) are affected. In cases of apoplexy, the anæsthesia is usually very imperfect, and not sharply defined. It is short-lived, lasting from a few hours to days; but in certain cases it may continue, and even outlive the motor paralysis with which it is conjoined.

Intracranial tumours may produce more persistent anæsthesia, either by immediate destruction of sensory fibres, or, indirectly, by the cerebral enlargement, due to their growth, causing compression of the fifth nerve as it traverses the floor of the skull.

(b) *Peripheral lesion.*—Anæsthesia dependent upon lesion of the trigeminus in its peripheral course is a symptom of serious moment, which it is important to distinguish from that of central origin; and this may be accomplished by noting the following points: The degree of peripheral anæsthesia far exceeds that which obtains in cases owing their origin to a central cause. It is much more complete, and may involve, which the latter does not, trophic and vaso-motor complications. Its extent varies according as the trunk of the nerve, including the Gasserian ganglion, is involved; or only one or two of its branches. Should the main trunk be affected, there is more or less complete anæsthesia of one side of the face and part of the ear, conjunctiva, cornea, nostril, mouth, half the tongue, the gums on the same side, and a part of the palate. If the conjunctiva be touched with the finger, there is no reflex contraction of the eyelids. A glass from which the patient drinks seems to him as though it were broken, for he feels the material on the sound side and not on the affected side. The skin of the face is cool, and may be somewhat œdematous, and purplish in tint. After a few days, if the cause persists, the eye on that side looks dry, glazed, and congested; the cornea becomes cloudy, and in time sloughs and perforates, the contents of the eyeball escaping to a varying extent, so that the organ is destroyed. There is dryness of the nostril on the affected side, and irritant substances applied to it fail to produce sneezing. Taste is lost on that side of the tongue, except at the base, which is supplied by the glossopharyngeal nerve. The salivary secretion is diminished. In time there may be bleeding from the gums and ulceration of the mucous membrane. Should the lesion exist upon one of the three divisions of the trigeminus, the anæsthesia will be found sharply limited to the district supplied by that division.

The nature of the lesion must be determined by the examination of concomitant conditions. It may consist either in caries of the bone, tumour, aneurysm, or meningitis, acute or chronic. Whatever be the active cause by which the nerve is damaged, the effects will be the same: pressure upon, and disorganization of the nerve-fibres will result in the disorders described—sensory, motor, trophic, and vaso-motor. In such circumstances, one or more of the other cranial nerves are usually affected coincidentally. In tubercular meningitis, the fifth nerve is shown to be paralysed (along with others traversing the floor of the skull) by the conjunctivitis and corneitis so

often present in advanced stages of the disease. Should the condition accompany bulbar paralysis, the lesion must be referred to the nuclei of origin of the nerve in the medulla oblongata.

**TREATMENT.**—Syphilitic gummata on the floor of the skull, developed either in the membranes of the brain or in the nerve itself, are so frequently the cause of the disorganization of the fifth nerve which gives rise to anæsthesia, that in all cases it is right—unless some other explanation is evident beyond all doubt—to bear in mind the possibility of such a cause, and to prescribe accordingly without delay. From 10 to 20 grains, or more, of iodide of potassium should be administered every four hours. Should there be a gumma pressing upon the trunk of the nerve, this treatment will probably have the effect of bringing about a rapid amelioration, and, in many cases, supposing it has been applied early enough, a complete cure. It is evident that, as regards other causes, there is no particular indication for treatment, which must be adapted to the special circumstances of the case.

**3. Trifacial Hyperalgesia.**—Hyperalgesia may accompany or precede neuralgia of the fifth nerve. It may also precede facial anæsthesia when this is due to neuritis. There are varieties in the degree of this hyperalgesia. It is sometimes so severe that the slightest touch occasions pain. The face cannot then be washed in the ordinary way, but the patient has to take a piece of sponge or wetted rag and cautiously dab the skin with it. Sometimes it is described as a feeling of soreness only when the hand is passed over the face. In either case the condition is accompanied by diminution of the tactile discrimination in the part. In *mimetic spasm* of the portio dura, there is often hyperalgesia in the region of one or more divisions of the fifth, and the lesion is then doubtless connected with the deep origin of this nerve. In blepharospasm it will often be found, if the face be carefully examined, that pressure with the finger at some point will check the spasm. Subcutaneous division of a twig of the fifth (or afferent nerve) at this point will often bring about a cure of the affection. The supra-orbital or subcutaneous malar are the nerves most commonly in fault.

Photophobia is referable to hyperalgesia of the branches distributed to the conjunctiva.

**4. Motor Disorders.**—Affections of the motor root of the fifth nerve are either (a) of a *spasmodic*; or (b) of a *paralytic* character.

(a) *Spasm.*—Spasm of the muscles supplied by the trigeminal nerve may be tonic or clonic. In trismus, or 'locked jaw,' the teeth are clenched together by the tonic contraction of the masticatory muscles, which can be felt tense to the touch. According as the muscles are involved generally, or only partially, the lower jaw will be fixed in a

symmetrical position, or be pulled over to one side, or advanced or receded. Clonic spasm of the same muscles is observed in various convulsive disorders; and slower movements of a horizontal character constitute the grinding of teeth sometimes indicative of cerebral disease.

Trismus may either be one symptom of tetanus (see TETANUS), or it may occur by itself, and then it either arises from cold, or is of reflex origin, from irritation of the sensory portion of the nerve by decayed teeth, dentition, or disease of the jawbone. It may be due to the presence of a foreign body, possibly of very small size, lodged in the cicatrix of a wound upon the face, or even in some distant part of the body. Irritation from worms is a possible cause. It is still more commonly hysterical.

**TREATMENT.**—When arising from cold, the constant current should be applied to the contracted muscles. Any source of irritation, whether from decayed teeth, or otherwise, must be sought for, and, if possible, removed or remedied.

The removal of a foreign body will sometimes bring about an immediate cure. This failing, the hypodermic injection of morphia, in doses of gr.  $\frac{1}{8}$ , may be employed, and bromide of potassium given internally in doses of 20 grains. When the presence of worms is suspected, appropriate treatment must be employed. If the affection be hysterical, somewhat powerful faradic currents, directed to the muscles of the jaw, will scarcely ever fail to open the mouth and cure the ailment. Hysterical trismus cannot be mistaken for dislocation of the jaw, if it be remembered that in the latter accident the jaw is fixed with the mouth partly open.

(b) *Paralysis.*—Paralysis of the masticatory muscles is not very common, but may be observed sometimes in cases of bulbar paralysis, or it may accompany anaesthesia of the face, and depend upon tumour, abscess, aneurysm, or some such coarse disease encroaching upon the trunk of the nerve within the cranium. To test the state of the muscles, the patient should be asked to move his jaw to and fro laterally, as well as in opening and shutting the mouth. Any irregularity of movement will be evident to the eye, and defective strength or absence of contraction in the affected muscles may be felt by placing a hand on each cheek, whilst the patient performs movements of mastication. When the jaw is found to fail in being carried to one side in a munching movement, the fault of course lies with the pterygoid muscles of the *opposite* side. The affection is more often unilateral than bilateral. Its importance is bound up with that of the lesion which gives rise to it.

As in peripheral affections of the sensory portion of the nerve, especial attention should be paid to the causation, and if this probably

depends upon a tumour, the possibility of its syphilitic character should be borne in mind, and iodide of potassium administered in doses of from 10 to 20 grains, or more. If these measures fail, the question of surgical operation may have to be entertained. See NEURALGIA; and TIC-DOULOUREUX.

T. BUZZARD.

**FILARIA** (*filum*, a thread).—A genus of nematode worms, not very clearly defined, but which contains a variety of thread-like parasites whose body is of uniform thickness throughout, and at least fifty times longer than it is broad. Under this head are often included several human parasites, such as the *Dracunculus*, or Guinea-worm (*Filaria medinensis*), and the lung strongle (*F. bronchialis*), in addition to a variety of larval or sexually immature nematodes, whose genetic relations are only very imperfectly understood. In the latter category may be placed Bristowe and Raimey's entozoon (*F. trachealis*); von Nordmann's eye-worm (*F. oculi-humani* or *F. lentis*); the *loa*, infesting the eyes of the Angola coast and Gaboon negroes (*F. loa*); and, lastly, the nematode hæmatozoon (*F. sanguinis-hominis*) described by the late Dr. Timothy Lewis. It may be doubted if any of the above-mentioned parasites ought to be included in the genus *Filaria*, as understood by modern helminthologists, but, practically, it is still found convenient thus to speak of them. The *Dracunculus* will be found described under ENTOZOA; whilst the microscopic nematode infesting the blood will be found noticed under the articles CHYLURIA, and FILARIA SANGUINIS HOMINIS. T. S. COBOLD.

#### FILARIA SANGUINIS HOMINIS.

This name was applied by Lewis to the embryo filaria which he discovered in human blood in 1872; and so long as no other similar parasite was found to have its habitat in the circulation, the name was appropriate. Recently, however, the writer has pointed out that the blood of certain Africans is liable to be infested by the embryos of two additional species of filaria; it therefore becomes necessary to modify Lewis's original name. Observation has shown that the filaria of Lewis appears in the blood only at night, disappearing from it during the day; whereas, of the two recently discovered species, one of them appears in the blood during the day only, disappearing from it at night; the other, in those individuals in which it occurs, is present in the circulation both during the day and during the night. Taking these facts as a basis for nomenclature, the writer proposes to name the three species—

*Filaria sanguinis hominis diurna.*

*Filaria sanguinis hominis nocturna.*

*Filaria sanguinis hominis perstans.*

*Methods of demonstrating filaria embryos in the blood.*—Blood is obtained in

the usual way from a finger-tip; but in looking for filariæ, the respective habits of the different species must be borne in mind. Thus, if search is to be made for *filaria sanguinis hominis diurna*, the blood should be drawn between the hours of 11 A.M. and 6 P.M.; if for *filaria sanguinis hominis nocturna*, between 9 P.M. and 6 A.M.; if for *filaria sanguinis hominis perstans*, it may be drawn at any hour of the day or night.

To study the movements of the filariæ, ordinary preparations of freshly drawn blood must be used; but to warrant a positive opinion as to the absence or presence of embryos in the circulation, and for purposes of enumeration, a larger quantity of blood than can be dealt with rapidly in the ordinary way must be searched. The following is a good plan. Spread about half a drop of blood on a slide in a uniform layer over an area about 1" by 1", and allow it to dry. The slide may be stained and examined at once, or stored away till such time as may suit the observer's convenience. All that is necessary to demonstrate the filariæ in such slides, no matter how old they are, is to immerse them for two hours in a half-per-cent. solution of eosin, or a weak solution of fuchsin (one drop of the saturated alcoholic solution in an ounce of water), then wash them in one or two waters, and mount in Farrant's solution or glycerine jelly. So prepared, the filariæ and white blood-corpuscles are stained, whilst the colour of the red blood-corpuscles is discharged. To demonstrate the sheath, shape, and structure of the filariæ, make a thin film of blood by applying a clean slide over another slide having a droplet of fresh fluid blood on it; after allowing the blood to spread out between the slides, rapidly glide or pull them apart; immediately invert the slides over a capsule containing a few drops of acetic acid until they are dry; then immerse them in a half-per-cent. solution of eosin for two minutes, wash thoroughly, dry slowly, and mount in balsam. So prepared, the filariæ and leucocytes show pearly-white, whilst the red blood-cells are deeply, and the liquor sanguinis is lightly, stained by the eosin.

No matter how prepared, the slides ought to be scrutinised in the first instance with a very low objective—an inch is sufficient—and every part of them examined. A quick-travelling mechanical stage is of great assistance in this, and ensures thoroughness. When a filaria is found it can be centred and examined with a quarter-inch or higher objective; but it must not be forgotten that, in the first instance, only very low powers are suitable for searching.

The three species of embryo filariæ sanguinis hominis closely resemble each other. As seen in recently drawn blood, they are long, slender, gracefully formed, transparent, colourless, snake-shaped organisms, which

for hours continue in a state of great activity, wriggling and twisting about on the slide like a wounded earth-worm, or an eel on a hook. After a time movement gradually slows down; but by a little management in preventing drying of the slide, the parasites can be kept alive for five or six days. In none of them can anything like an alimentary canal, or any definite internal structure, be made out.

As far as is at present known, the points of contrast in the three species are as follows:—

1. *Filaria sanguinis hominis diurna* (fig. 51).—The free hæmatozoal embryo of a species of filaria, the mature form of which is still unknown. Excepting that it enters the circulation during the day, this blood-worm resembles very closely in size, form, and habit the now familiar *filaria sanguinis hominis nocturna* of Lewis. The peculiarity in periodicity referred to is, doubtless, an adaptation to the habits of its unknown intermediary host. There is some reason for supposing that the filaria loa—a parasite living in the connective tissues, and sometimes showing itself underneath the conjunctiva in negroes—is the mature form of *filaria sanguinis hominis diurna*; for the geographical distribution of both, as far as our knowledge of this extends, is the same, and the undoubted embryo of *filaria loa* resembles *filaria sanguinis hominis diurna* in shape, and possibly in size; and, moreover, a patient of the writer's, who had at one time a filaria loa under his conjunctiva, has now the *filaria sanguinis hominis diurna* in abundance in his blood. The parasite seems to be confined to the tropical zone of Africa, more especially to the west coast. Future observation is likely to show that a large proportion of the inhabitants of these districts are affected with these worms, and it is likely that in a proportion of instances some pathological condition may be traced to them. As yet our information on these points is much too fragmentary to warrant even speculation.

2. *Filaria sanguinis hominis nocturna* (fig. 52).—The free hæmatozoal embryo of *filaria Bancrofti*. We owe the first notice of this parasite to Demarquay, who, in 1863, found numerous specimens of what, considering the circumstances of the case, must have been *filaria sanguinis hominis nocturna*, in the contents of a lymphous tumour of the thigh.<sup>1</sup> Later, in 1869, Wucherer in Bahia, and, quite independently, in 1870, Lewis in Calcutta, found the same parasite in chylous urine. It was not, however, until 1872, when Lewis showed that the normal habitat of this embryo worm was the human blood, that the discovery excited much interest. Since that date the significance of this parasite as a factor in tropical pathology has gradually come to be recognised, and many interesting

<sup>1</sup> *Gaz. Med.* 1863, p. 665.

points of a complicated life-history have been worked out.

In the first issue of this Dictionary Lewis described the *filaria sanguinis hominis nocturna* as follows: 'Its average length is  $\frac{1}{75}$ " (= 0.34 mm.); its breadth  $\frac{1}{3500}$ " (= 0.007 mm.), or about equal to the diameter of a red blood-corpuscle. It is enclosed in a transparent tubular sac, within which it can be seen to alternately contract and elongate itself. This sac is extremely delicate and translucent, and may sometimes, when the worm has shortened

itself more than usual, be seen collapsed and folded like a ribbon, and the next moment be instantaneously straightened again, by the extension of the filaria to its ordinary length. After death the worm may occupy either the entire length of the tubular sac, or be so contracted as to leave the tube empty at one or both ends, as may be observed in the woodcut (fig. 54).'

As no sign of growth or of reproductive organs is distinguishable in this the hæmatozoal stage of the parasite, it is evident—and

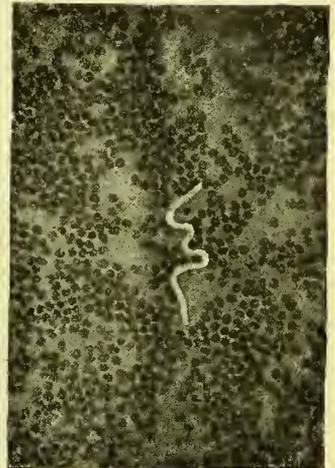
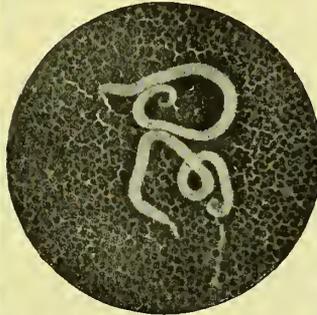
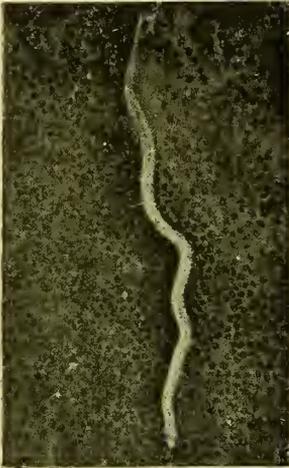


FIG. 52 — *Filaria sanguinis hominis nocturna* × 180.—1. Measures  $\frac{1}{80}$  in. by  $\frac{1}{3500}$  in., or thereabouts. 2. Is provided with a coarser sheath. 3. Caudal end tapers gradually for about one-fifth of the entire length of the animal, and terminates in a sharp, or nearly sharp, point. 4. Cephalic end is rounded off, and has obscure oral movements of a pouting character. 5. No tongue-like organ visible. 6. Appears in the blood at night, disappearing during the day. 7. Has a wriggling, but no locomotive, movement. 8. Many specimens have a well-marked granular-looking aggregation about the middle of the body.

FIG. 51. — *Filaria sanguinis hominis nocturna* × 180 (this fig. and figs. 52 and 53 from microphotographs by C. Detmold).—1. Measures  $\frac{1}{80}$  in. by  $\frac{1}{3500}$  in., or thereabouts. 2. Is provided with a very delicate sheath. 3. Caudal end tapers gradually for about one-fifth of the entire length of the animal, and terminates in a sharp, or nearly sharp, point. 4. Cephalic end is rounded off, and has distinct oral movements of a pouting character. 5. No tongue-like organ visible. 6. Appears in the blood during the day, disappearing during the night. 7. Has a wriggling, but no locomotive, movement. 8. A few specimens have a faintly marked granular-looking aggregation about the middle of the body, but in none is it very well marked.

FIG. 53. — *Filaria sanguinis hominis nocturna* × 180.—1. Measures  $\frac{1}{125}$  in. by  $\frac{1}{5500}$  in., or thereabouts. 2. Has no sheath. 3. Caudal end tapers more gradually for two-thirds of the entire length of the animal, and is abruptly truncated where it has tapered to about one-third of the diameter of the thickest part of the body. 4. Cephalic end is either conical or truncated, passing from one form to the other rapidly by a peculiar jerking, extruding and retracting, movement. 5. From time to time, a minute tongue-like organ is rapidly protruded and withdrawn at the extremity of the cephalic end. 6. Observes no periodicity. 7. Has a locomotive as well as a wriggling movement. 8. No granular appearance.

this was early recognised—that these blood-worms are only the immature young of parasites which must exist in a sexually mature form elsewhere in the tissues of the human host, and in intimate relationship with the circulation. Lewis made diligent search for the parental form soon after his discovery of the embryo, but it was not until 1877 that he found two living specimens—a male and a female—in the diseased tissues of a nævoid elephantiasis of the scrotum. Late in the previous year Bancroft, in Brisbane, found

several mature females in a 'lymphatic abscess' of the arm, and also in 'an encysted hydrocele of the cord.' The latter were carefully examined by Cobbold, and the parasite was named by him *filaria Bancrofti*. Since that time the mature filaria Bancrofti has been found in India, China, and Brazil. It is a long, thread-like worm, about the thickness of a horse-hair, of a white colour and firm consistence, and exhibits considerable activity, and a remarkable tendency to coil. Cobbold described the female as follows:

'Body capillary, smooth, uniform in thickness. Head with a simple circular mouth, destitute of papillæ (fig. 55). Neck narrow,



FIG. 54.—*Filaria sanguinis hominis nocturna*.  $\times 300$ . (Traced from a micro-photograph.)



FIG. 55.—Anterior end of mature *filaria sanguinis hominis nocturna*.—Mature form.  $\times 100$  diameters.

about one-third of the width of the body. Tail of female simple, bluntly pointed; reproductive outlet close to the head; anus

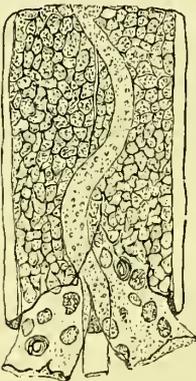


FIG. 56.—A portion of the mature *filaria sanguinis hominis nocturna*, showing uterine tubules filled with ova in various stages of development; also the intestinal tube.  $\times 100$  diameters.

immediately above the tip of the tail. Length of largest females  $3\frac{1}{2}$  in., breadth  $\frac{1}{90}$ '''. In the female the uterine tubes are packed with ova in all stages of development (figs. 56 and

57). Lewis gives the measurement of these, in which the embryos are distinctly visible, as  $\frac{1}{66}$ '' by  $\frac{1}{90}$ '''. In a fresh specimen of the filaria examined by the writer the largest ova gave slightly greater measurements, and

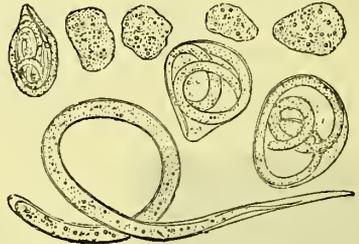


FIG. 57.—Ova and embryos of *filaria sanguinis hominis nocturna*.  $\times 300$  diameters.

he saw that towards the vaginal end of the uterine horns the embryos lay outstretched, and that in the vagina they had very much the appearance they have in the blood, proving that under normal circumstances the parasite is viviparous. Lewis gives the breadth of the fragment of a male he examined as  $\frac{1}{180}$ ''.

It is now known that the parent worms live together in the lymphatics; that the embryo while *in utero*, by dint of vigorous movements, stretches its chorionic envelope to form the long tubular sheath in which it lies extended, as we see it in the blood and lymph, and that, after this stretching of the chorion is complete, the embryo is born into the lymph-stream, which carries it through the glands, along the thoracic duct, and thus into the blood. Under ordinary circumstances of health and habit the hæmatozoon cannot be found in the blood during the day, but at evening it appears there. At first only a very few can be found; as night advances their numbers gradually increase; about midnight they attain their maximum—in some cases as many as 200 may be found in a single drop—after this they gradually diminish up to about 8 or 9 A.M., when they disappear for the day. An attack of fever may disturb this periodicity, and Dr. Stephen Mackenzie<sup>1</sup> has shown—and his observations have been abundantly confirmed—that by causing the subject of filaria infection to sleep during the day and keep awake during the night, the habit of the embryo parasite in his blood is correspondingly changed: it then appears, contrary to its normal habit, in the blood during the day, and disappears from it at night. See NEGRO LETHARGY.

This singular periodicity is, apparently, an adaptation to the nocturnal habits of the female of a particular species of mosquito, which imbibes with the blood the young filaria, and acts as its intermediary host.

<sup>1</sup> *Trans. Path. Soc.* 1882, vol. xxxiii.

Transferred to the stomach of the mosquito, the filaria first casts its chorionic envelope, and then bores its way into the thorax and thoracic muscles of the insect. Here it undergoes a remarkable metamorphosis, eventuating in its becoming possessed of an alimentary canal, a circumcaudal circle of papillæ, and greatly increased size, power, and activity. This metamorphosis takes about six days to complete. At the end of this time the mosquito dies, after depositing her eggs on the surface of the water to which she resorts for this purpose. It is presumed that the filaria then escapes from the body of the insect to the water, into which the latter has probably fallen. Through this medium, it is believed, it gains access to the human body, being swallowed in drinking-water.<sup>1</sup>

**GEOGRAPHICAL DISTRIBUTION.**—The filaria sanguinis hominis nocturna has been found in most tropical and subtropical countries in which it has been properly looked for. Its exact geographical distribution as an endemic parasite is doubtless in keeping with the geographical distribution of the particular species of mosquito subserving its interests as intermediary host; but, as the filaria may live for many years in the tissues of its human host, it is frequently brought by travellers from tropical and other countries to colder latitudes; and there is at least one well-authenticated instance in which it was found in a patient who had never quitted England.

**PATHOLOGICAL RELATIONS.**—Chyluria, œvoid elephantiasis or lymph-scrotum, chylous dropsy of the tunica vaginalis (variously termed chylocele, lymphocele, galactocele), varicose and indurated inguinal glands, chylous ascites, recurring orchitis, certain kinds of abscess occurring in the limbs—especially in the thighs and groins, lymphangitis, and lymphatic fever, are almost invariably accompanied or preceded by the presence of the filaria in the blood or in the lymph, or in both. The association is so constant and so intimate, that in 99 cases out of 100 these diseases may be confidently attributed to the filaria, at all events when they are of tropical origin.

Tropical elephantiasis arabum is also attributed to the filaria, although the parasite is by no means always found in the fully developed cases. Theoretically, all elephantoid diseases are produced by some mechanical or other physical influence operating by obstructing or irritating the lymphatic vessels. In tropical elephantiasis it is maintained that this influence is the filaria nocturna; and on the following grounds. Cases are on record of lymph-scrotum—a disease certainly

attributable to the filaria—passing into true elephantiasis arabum; many cases of true elephantiasis arabum scroti have a history of antecedent lymph-scrotum; lymph-scrotum has supervened in flaps left after removal of an elephantiasis scroti by operation; elephantiasis of the leg has followed on the removal of a lymph-scrotum; lymph-scrotum and elephantiasis of the leg sometimes co-exist in the same individual; a case is on record of lymphorrhagia in an elephantoid leg co-existing with varicose inguinal glands and filariæ, and in which chyluria subsequently developed; an instance is recorded in which a son developed a lymph-scrotum, and the mother—living in the same house—an elephantiasis of the leg; where elephantiasis is endemic, there too chyluria and lymph-scrotum are found. It is unreasonable to suppose that the combinations and concurrences mentioned were, in every instance, simply coincidences; much more likely the same cause produced them all. This being proved to be the filaria in chyluria, lymph-scrotum, and varicose inguinal glands, it follows that it must be the filaria also that produces true tropical elephantiasis arabum. Furthermore, as far as known, the geographical distribution of elephantiasis is co-extensive with that of the filaria.

Because the filaria is not always found in elephantiasis, it has been maintained by some that this parasite cannot be the cause of this disease. The fact is, the filaria is mortal and may die before its host; nevertheless, the ill it has wrought may not disappear at its death. It sometimes happens that in chyluria and in lymph-scrotum no filariæ can be found in the blood, urine, or lymph, although at one period they must have been present; sometimes it can be found in the lymph only, and not in the blood; and instances are on record in which the parasite disappeared from the blood and lymphous discharges whilst the cases were under observation, yet the characteristic morbid discharges persisted. In pathology, as in other things, a transient influence may produce a permanent lesion. It must be admitted, however, that the reason of the very frequent absence of the embryos of the filaria from the blood in developed elephantiasis is not known, and further observations bearing on this point are much wanted.

From the fact that in many countries where the filaria is endemic quite 10 per cent. of the adult population harbour it, yet only a small proportion have any of the diseases enumerated, it is evident that the parasite does not necessarily give rise to disease. It would appear that so long as the parasite is healthy and normally situated it is innocuous. Should it die, it may act as a foreign body and cause abscess; the dead filaria has actually been found in lymphatic abscesses. But the precise way in which the

<sup>1</sup> For a detailed description of the metamorphosis of the filaria in the mosquito, see *Trans. Linnæan Soc. of London*, vol. ii. part 10; 2nd ser. 'Zoology,' April 1884.

parasite usually brings about pathological obstruction of the lymphatics is, as yet, to some extent a matter of conjecture. Of two ways in which it so acts there is good evidence. (1) The parent worm may cause lymphatic stasis by operating at a point high up the thoracic duct, either itself plugging that vessel, or giving rise to inflammatory thickening in or around its walls, ending in stricture. (2) As mentioned, there are instances on record in which, though the filaria was present in the chylous urine, or in the lymph drawn from a lymph-scrutum, or from varicose inguinal glands—yet, after the most careful and prolonged search, it could not be discovered in the blood. It is evident that in these cases there must have been complete obstruction of a lymphatic area, otherwise the filaria embryos must have appeared in the blood, as in ordinary examples of filarial infection. The obstruction could not possibly have been in the thoracic duct, for, if complete in this situation, it must have speedily brought about the death of the patient. It is safe, therefore, to infer that it must have been somewhere on the distal side of the receptaculum chyli. In two such cases the ova of the parasite have been found in the lymph; and it is believed that in this circumstance we have the explanation of the site and character of the lymphatic obstruction in a large proportion of cases. Normally, the parasite is viviparous, and the long, slender, active embryo has no difficulty in finding its way along the lymphatics and through the glands into the circulation. But should the embryo be born prematurely, as in the two cases referred to, before the chorionic envelope has undergone the stretching process already described—that is, should ova (seven or eight times the diameter of the outstretched young) be prematurely launched into the lymph-stream, they act as emboli, and, being very numerous, effectually plug the glands connected, directly or by anastomosis, with the lymphatic vessel in which the aborting parent lies. The location of the worm; the degree of obstruction, whether caused by embolism, pressure, or stricture; pressure by enlarged glands interfering with the compensating drainage of the affected areas by the veins; traumatic irritation of the congested lymphatic area; the position of the part involved as regards dependence or elevation, a most important factor in elephantiasis; the health of the patient; and other circumstances, easily understood, will determine the site and exact character of the resulting disease.

**TREATMENT.**—(a) *Curative.*—No means of killing the filaria have been discovered. The indications for treatment, when disease has developed, are supplied by the pathology. Rest, elevation of the affected parts, elastic bandaging, and other means to facilitate the flow of lymph through the damaged lymph-

atics, are of great benefit. See CHYLURIA; and ELEPHANTIASIS ARABUM.

(b) *Preventive.*—The fact that the mosquito acts as intermediary host indicates the direction that preventive measures should take. Drinking-water, in districts where the filaria is endemic, ought to be boiled and filtered; wells, cisterns, and receptacles for drinking-water ought to be covered with fine wire-gauze, to prevent the access of the mosquito; and persons known to harbour the parasite ought to sleep under properly constructed mosquito-curtains. By any or all of these means this parasite, in the course of a single generation, could be exterminated, and the diseases it produces made things of the past.

3. *Filaria sanguinis hominis perstans* (fig. 53).—The free hæmatozoal embryo of a species of nematode, the mature form of which is unknown. Hitherto this hæmatozoon has been found only in the blood of negroes from the West Coast of Africa, and it is probable that as an endemic cause it is restricted to this part of the world. Twice it has been found in company with the filaria sanguinis hominis diurna; in several other cases it was not so associated. We may therefore infer that the association of filaria perstans and filaria diurna in the two instances referred to was merely accidental.

Attention to the points of contrast between filaria perstans and the other blood-worms, as set forth in the table in page 697, will lead to its recognition. In addition to what has been stated in this table, it may be mentioned that the body of this parasite is possessed in a high degree of powers of elongation and contraction, and that the measurements given in the table referred to are those of the animal when it is dead or moribund. When travelling actively among the blood-corpuscles it sometimes so extends itself as to appear a mere thread, the elongation concurring with a corresponding attenuation; at other times, and when not actively travelling, it assumes the proportions given. A very striking habit which it has is that of coiling itself up like a rope, and remaining motionless in this attitude for a few seconds before resuming its usual movements. Another peculiarity worth mentioning is the result, apparently, of the locomotive habits of this parasite: not infrequently it is seen fixed to one spot on the slide, vainly endeavouring to break away, and constricted by a delicate rope of fibrin. It would seem that in moving about the slide it pushes the strings of fibrin in front of it, and at times becomes entangled in a rope of them.

The filaria sanguinis hominis perstans is probably very common in the endemic region; for, of ten negroes from the West Coast of Africa whose blood was searched, it was found in five.

That in certain cases it gives rise to pathological conditions is probable; but of what

these are we are as yet ignorant. In one case the parasite was associated with the peculiar disease called Negro Lethargy or the Sleeping Sickness of the Congo; in another with insanity; but in the other three instances in which it was found there was no concomitant disease. There may be some grounds for associating it with a peculiar African disease called *Craw-craw*, a kind of itch, apparently induced by a filaria-like parasite, and not the *acarus scabiei*; as yet this supposition is little more than hypothetical.

The presence of a beak, the absence of a sheath, the marked locomotor habits, the extensibility and pliancy of its body, and also the non-observance of any periodicity in its entrances and exits from the circulation, suggest that the embryo *filaria sanguinis hominis* perstans gains its freedom and enters its particular intermediary host by dint of its own efforts, and that it is not, as is the case with *filaria sanguinis hominis nocturna*, and probably *diurna*, dependent on the good offices of some species of mosquito or other bloodsucker.

PATRICK MANSON.

**FISSURE** (*findo*, I cleave).—A narrow and superficial crack or solution of continuity, observed on the skin and mucous membranes, and especially near the line of junction of these structures, as on the lips and within the anus. See ANUS, Diseases of; and CHAPS.

**FISTULA** (*fistula*, a pipe).—A narrow track or canal leading from a free surface, and extending more or less deeply to some seat of local irritation; or it may be constituting an abnormal communication between two or more cavities, as in the case of vesico-vaginal or recto-vaginal fistula. See ABSCESS.

**FISTULA IN ANO**.—See RECTUM, Diseases of.

**FIT**.—A popular synonym for a sudden seizure characterised by loss or disturbance of consciousness from any cause, with or without convulsions (see CONVULSIONS; EPILEPSY; HYSTERIA; and SYNCOPE). The term is also applied to a sudden or acute seizure of certain diseases, such as gout, asthma, and ague.

**FLATULENCE** (*flatus*, a puff of wind). SYN.: Fr. *Flatulence*; Ger. *Flatulenz*.

**DEFINITION**.—The undue generation of gases in the stomach and intestines.

**ÆTIOLGY**.—The principal cause of flatulence is fermentation or decomposition of the contents of the stomach and bowels—a condition usually induced by embarrassment of function. Hence it is a common symptom in dyspepsia—especially the atonic forms as met with in the debilitated and the aged, constipation, gastritis, enteritis, hepatic disorders, and intestinal obstruction. When flatus is generated too rapidly to be accounted

for by fermentation, as in hysteria, hypochondriasis, and other forms of nervous debility, it has been ascribed to secretion of gases from the mucous membrane.

**SYMPTOMS**.—The clinical phenomena vary as flatus is retained or discharged; and with the seat of its formation, whether chiefly in the stomach or intestines. In the former the concomitant symptoms are those of dyspepsia; in the latter there is usually constipation. As a rule, however, flatulence pervades at the same time more or less all the hollow viscera, and indicates torpor of the digestive organs. It is apt to lead to these further evils:—*(a) Pain* from distension or from irregular and forcible contractions of the walls—hence gastrodynia and colic are apt to arise; *(b) arrest of the normal movements* of the stomach and intestines, and consequent accumulation within them of fermentable matters, with further generation of gases, leading to paralytic distension: hence dilatation of the stomach and colon, tympanites or meteorism, and aggravation of pre-existing dyspepsia or constipation may ensue; and *(c) pressure on adjacent organs*, such as the heart and lungs, inducing palpitation and irregular action of the heart, precordial anxiety, faintness, vertigo, dyspnoea, or even asphyxia.

In tympanites there is a rapid generation of flatus, which overpowers the contractility of the hollow viscera; and the abdomen is round, tense, and tympanitic. See TYMPANITES.

**TREATMENT**.—*(a) Imprisoned flatus should be dislodged* by friction of the abdomen with stimulating liniments, and gentle kneading of the most distended parts; large draughts of hot water; spirit and hot water; ammonia, ether, or spirit of chloroform; aromatic stimulants—ginger, capsicum, cloves, mint, anise, cajuput, camphor, or cascarrilla. When flatulence is chiefly intestinal, enemata containing laudanum with asafetida, turpentine, or rue; and the compound asafetida pill with extract of nux vomica, and an aperient, are the most useful measures.

*(b) The generation of flatus should be arrested*. Fermentation may be checked by sulphite or sulpho-carbolate of sodium, sulphurous acid, carbolic acid, creasote, terebene, salol, or charcoal—from the poplar or vegetable ivory, immediately after food; and by correcting and toning the digestive organs. Repeated fractional doses of calomel will also frequently arrest fermentative flatulence. Food likely to ferment or lodge, such as starch, sugar, fruits or green vegetables, and warm liquids—especially tea and soups—should be avoided; the meals should be well masticated and solid throughout, and liquids should only be taken sparingly at the close or an hour after. In some cases, however, flatulence is connected with an insufficient supply of fluids, and should be met by

increasing it; then also a tumblingful of hot water night and morning and an hour before each meal, which should consist of solids, may prove useful. Alkalis—carbonate of magnesium and bicarbonate of sodium—and bitters, especially strychnine, are often useful in the flatulence of hysteria, hypochondriasis, of the very nervous and the aged; but perhaps the best results follow alkalis with nux vomica and bismuth an hour before, and hydrochloric acid alone or with some reliable preparation of pepsin after food. It is also essential to see that the action of the liver is healthy.

GEORGE OLIVER.

**FLEXION** (*flecto*, I bend).—A bending. This term is applied either to the act of more or less forcible flexion, as in some methods of treatment, for example in the cure of aneurysm or the reduction of dislocations; or to the condition in which parts are bent, as the result of disease or of disorder, as when the limbs or certain internal organs are bent upon themselves. See WOMB, Diseases of.

**FLINSBERG, in Silesia.**—Chalybeate waters. See MINERAL WATERS.

**FLOODING.**—A popular term for excessive discharge of blood from the womb. See MENSTRUATION, Disorders of; and PREGNANCY, Disorders of.

**FLUCTUATION** (*fluctus*, a wave).—A physical sign consisting in a wave-like or undulating sensation. It is elicited by a peculiar mode of palpation with the one hand, while percussion is made with the fingers of the other; and is due to the presence of a fluid in a natural cavity, such as the peritoneum, or in an abnormal closed space such as a cyst (see PHYSICAL EXAMINATION). The term 'fluctuation' as used by the surgeon has a somewhat different signification, being applied to the sensation of the presence of a fluid which may be felt when alternate pressure with the fingers is made, as over the seat of an abscess. See ABSCESS.

**FLUKE.**—See ENTOZOA.

**FLUOR** }  
**FLUX** }.—A flow or excessive discharge from a mucous surface, through any of the natural passages, of serum, blood, mucus, pus, or the various secretions. As illustrations of fluxes may be mentioned salivation, bronchorrhœa, biliary flux, diarrhœa, dysentery or bloody flux, cholera, and leucorrhœa or white flux (*fluor albus*).

**FŒTUS, Diseases of the.**—Two classes of abnormal conditions are seen in the fœtus, namely: Those which depend upon some interference with the process of development, such as malformations and monstrosities; and those which are the result

of disease. This article treats of the latter only.

1. *Amputation.*—Amputation, partial or complete, of fœtal limbs may take place, from constriction of the limb by a band of the amnion. An attempt at reproduction of the lost limb is sometimes seen, in the shape of rudimentary fingers and toes, projecting from the stump. That such a stump is the result of amputation is proved by the fact that the part cut off has been found *in utero*.

2. *Spontaneous fractures and dislocations.* Fractures and dislocations occur *in utero*, the latter being the more rare. They are due to some condition of the bones and ligaments respectively, leading to undue fragility of those structures; for they are always multiple, and are not accompanied with bruising of the adjacent soft parts.

3. *Tumours.*—New-growths are met with in the fœtus—cysts of various kinds, fibroma, lipoma, &c. That most special to the fœtus is a tumour situated over the coccyx, which may be as large as a fetal head. Such tumours are spheroidal or ovoidal in shape, elastic in consistence, and present rounded inequalities on the surface. On section they are found to consist of strong fibrous trabeculae, in the meshes of which are numerous small cysts lined with epithelium. It is thought by many that they originate in Luschka's gland. Another special kind of tumour is that known as a *fœtal inclusion*—a swelling usually on the lower part of the trunk, and containing some part of another fœtus, more or less imperfectly developed.

4. *Inflammation of serous membranes.* This form of disease may occur in the fœtus, such as pleurisy and peritonitis. The morbid anatomy of these changes does not differ from that in the adult. Peritonitis is often found associated with syphilis; and it appears to be almost always fatal to the fœtus.

5. *Visceral inflammations.*—Inflammation of the lungs has been met with, in the form of grey or white lobular hepatisation. It is most frequent in syphilis. Enteritis has also been described. Certain malformations of the heart which are met with can be explained by supposing the occurrence of endocarditis during fœtal life; but there is no proof that the fœtus is subject to rheumatism.

6. *Specific fevers.*—The morbid changes of *enteric fever* have been found in the fœtus. In pregnant women suffering from *intermittent fever*, paroxysms of convulsive movements of the child have been felt to occur as regularly as the attacks of ague in the mother; and the child when born has been found to have a large spleen. Children have also been born with skin-eruptions thought to resemble those of *measles*, *scarlatina*, and *small-pox*. The facts as to the last-named disease are the most numerous and probable. The eruption of variola in the fœtus differs some-

what from that seen after birth, because, the skin of the fœtus being bathed with fluid, no crusts form, and the pustules run a course like those on mucous membranes.

7. *Diseases of the skin.*—The fœtus is subject to skin-diseases. Pustules of ecthyma; patches of erythematous redness; ulceration of the skin, and syphilitic eruptions, have been seen. Intra-uterine ichthyosis is met with. Children have been born jaundiced, but only by mothers themselves suffering from that condition. But women with jaundice do not always bear jaundiced children. Jaundice is not necessarily fatal to the fœtus.

8. *Syphilis.*—Syphilis leads to various lesions in the fœtus; and while it usually proves fatal, the subjects of it that may survive till birth are feeble and badly nourished. Flat tubercles occur on different parts of the skin, especially round the mucous orifices; and pemphigus may be seen, affecting chiefly the palms of the hands and the soles of the feet. The occurrence of peritonitis has already been mentioned. Yellow indurated nodules, of varying size and number, may be found in the liver, as well as in the lungs. A peculiar change has been described in the thymus gland, in which this structure externally appears healthy, but when cut into and compressed exudes a whitish puriform fluid. Other changes have been recorded, affecting the spleen, pancreas, and suprarenal capsules, but they are not distinctive. Changes in the bones have also been described, consisting of an osteochondritis, affecting the ends of the long bones, most frequently the lower end of the femur.

9. *Rachitis.*—Fœtal rickets is a disease somewhat different from that seen after birth. The limbs are short, very fat, the fat being thrown into rolls separated by transverse constrictions; the trunk, especially the abdomen, is large; the head often hydrocephalic. The bones are short, often fractured and bent; there is swelling, softening, and delayed ossification of the epiphyses. It may begin early, and cease before delivery; or the child may be born with the disease in progress. Nothing is known of its causes.

10. *Tuberculosis.*—Tuberculosis sometimes commences in the fœtus, tubercles having been found in the mesentery and in the lungs.

11. *Dropsies.*—Dropsy is met with in the fetus, sometimes of the serous cavities, of which hydrocephalus is the most common. It is often associated with rickets. Next in frequency comes ascites; and lastly hydrothorax, which is very rare. These affections may destroy fetal life *in utero*; but they more often lead to death because they render destructive operations necessary before delivery can be accomplished. General anasarca is also met with, and there is reason to believe that it depends upon disease of the

placenta, impairing the excretory function of that organ. It is always fatal, if not before birth, within a few hours after it.

12. *Visceral diseases.*—Hypertrophy of the liver, spleen, or kidneys may occur in the fetus. Cystic disease of the kidneys may be met with, the organs being converted into a mass of cysts containing no trace of secreting structure. Both kidneys are usually affected, and the disease is generally associated with some malformation elsewhere. Hydronephrosis, single or double, along with dilatation of the ureter, or ureters and bladder, has been seen, dependent upon impermeability of some part of the urinary passages. Any of these visceral diseases may form a tumour so large as to impede delivery. None of them can be diagnosed before birth. Concretions of uric acid and urates are not uncommon.

#### The Causes of Death of the Fœtus.

The various causes of death of the fœtus *in utero* may now be briefly considered. The first of these is *injury*, as when the mother receives a blow upon the abdomen, or has a fall. Such occurrences rarely injure the fœtus directly, although this has been known to happen. When they prove fatal to the fetus, they do so by leading to hæmorrhage into, or separation of, a portion of placenta, and consequent disturbance of the fetal circulation. *Poisons* in the mother's blood, such as lead, urea, bile, or carbonic acid (as in the case of heart-disease with cyanosis), may lead to death of the fœtus. *Syphilis* has already been referred to. *Epileptiform convulsions* may destroy fetal life, either by asphyxia, or by leading to hæmorrhage into the placenta. Extreme *anæmia* and the *cancerous cachexia* are among the causes of fetal death. Any kind of disease attended with *pyrexia* will also destroy fetal life if the temperature rise high enough. A temperature exceeding 105° F. appears to be invariably fatal to the fœtus. Certain *diseases of its appendages* may lead to death of the fœtus, such as hæmorrhages into the placenta, old organised clots forming what are called placental 'infarcts'; obliteration of the umbilical vessels; or interruption of the circulation in the cord by knots in it, or pressure upon it. It is believed that there are some *diseases of the mother's uterus* which lead to death of the fœtus; but what they are is not known. It is said that some women acquire a habit of having dead children. This means that there are cases in which an apparently healthy woman will repeatedly have dead children, the cause of whose death a skilled observer cannot find out. In other words, there are causes of fœtal death as yet unknown.

**The Consequences of Death of the Fœtus.**—A dead fœtus while retained *in utero*, and thus protected from the air, does not putrefy, but undergoes a process of

maceration. The whole body becomes soft and flaccid, its tissues being infiltrated with fluid; but it has no putrid odour. The skin presents bullæ filled with reddish serum, and the epidermis is readily detached with slight friction. The surface is of a cyanotic colour, which after exposure to the air becomes of a more or less bright red; it is not greenish as is seen in putrefaction. The cellular tissue is infiltrated with bloody serum. The viscera have lost their distinctive tints, and become of a reddish-brown colour. The cranial bones are abnormally mobile, overlapping one another to a greater extent than normal; and the periosteum may be stripped off them. These appearances are much the same, whatever be the cause of death, but they vary in degree according to the length of time which has elapsed since death.

Besides the diseases of the fœtus described above, there are others which are not fatal, and the chief interest of which lies in their course and treatment after birth. These are discussed in other articles, and need not be mentioned here. G. E. HERMAN.

**FOLIE CIRCULAIRE** (Fr.)—This term is applied by the French psychological physicians to a variety of insanity characterised by alternations of excitement and depression. The patient passes through an attack of mania of perhaps an ordinary character, but when he appears to have recovered he sinks into melancholia, and thence emerges again to become maniacal and excited. The duration of each stage may vary from weeks to months. Sometimes one state will follow the other immediately; in other cases a period of convalescence will intervene, during which the patient appears well, and can hardly be considered insane. Yet the prognosis is extremely unfavourable in all such cases; and it is of great importance in estimating the extent of recovery of a patient, that it should be clearly ascertained that the attack is not one of a series following one another in the manner mentioned.

G. F. BLANDFORD.

**FOLLICLES, Diseases of** (*folliculus*, dim. of *follics*, a bag).—The name 'follicle' has been applied to a great variety of different structures, which have in common the shape of a bag or sac, whether circular or elongated in outline; for example, the Graafian follicles, the lymphatic (Peyerian) follicles of the intestine, and the follicles of the mucous membrane of the stomach, intestine, and uterus. The name has been further extended to include glands somewhat more complex in structure, such as the sudoriparous glands or sweat-follicles, the sebaceous follicles, and the tonsils; as well as the saccular depressions in which the hair and the teeth take their origin. The name *synovial follicles* is sometimes given to processes of synovial

membrane invaginated in the capsule of a joint.

For an account of the diseases of these various structures, the reader is referred to the several articles under which they are discussed; but so far as the true follicles are concerned, the following may be said to be the principal morbid changes to which they are liable: atrophy; hypertrophy; obstruction, and distension; inflammation; ulceration; cystic disease; new-growths; tubercle; acute specific processes, such as the typhoid; and parasitic disease.

**FOMENTATION** (*foveo*, I keep warm).

SYNON.: Fr. *Fomentation*; Ger. *Bähung*.

DEFINITION.—Fomentation is the application to the surface of the body of flannels, cloths, or sponges moistened with hot water, either pure or containing some medicinal substance in solution.

ACTION.—The action of a simple fomentation is the same as that of a poultice. By its warmth and moisture it tends to relax the muscular fibres of the skin and soften the cuticle, thus relieving tension, and diminishing pain and irritation. In the early stages of inflammation it favours resolution, by maintaining the temperature, and promoting active circulation through the area which has suffered from the injurious influence which has started the process. In the later stages it promotes and hastens suppuration, by causing dilatation of the vessels, and encouraging exudation. A fomentation is superior to a poultice in lightness and cleanliness, but unless care be taken it loses its heat more quickly. This disadvantage may be overcome by covering the fomentation with a thick layer of cotton-wool.

APPLICATION AND USES.—A fomentation is thus applied: A piece of coarse flannel, sufficiently large to cover the affected part when folded into two layers, is put into a basin, and boiling water is poured upon it. It is then lifted from the basin with a pair of tongs or some convenient instrument, and dropped on the wringer. This is a stout piece of towelling with a stick attached to each end. The sticks then being twisted in opposite directions, as much water as possible is squeezed out of the flannel. It is then immediately placed on the affected part, and covered with a large piece of oiled silk or indiarubber sheet extending at least one inch on each side of it. Over this may be placed a thick layer of cotton-wool, and a bandage. If the flannel be not squeezed sufficiently dry, it will wet the bed or clothing. If not sufficiently covered with oiled silk and wool, it soon becomes cold. Whatever means may be taken to retain the heat of a fomentation, it can be kept above the temperature of the body only for a few minutes. If, therefore, the full effect of fomentation is desired to be obtained, the flannels must be changed every

twenty minutes. In many parts a sponge, or a piece of spongio-piline, wrung out of boiling water, forms a most convenient form of fomentation. When the fore-arm or hand is affected, a bath of hot water may be substituted for fomentations. The temperature of the water must be maintained by the repeated addition of small quantities of boiling water.

Fomentations are especially useful in all cases of erysipelas and diffuse cellulitis, and in boils. In peritonitis they are borne more easily than poultices, on account of their greater lightness. Whenever they can be employed they are superior to poultices, on account of their cleanliness. They are not applicable to cases in which there is a discharging wound or abscess, as under such conditions the cloths become foul.

**VARIETIES.**—If it is desired to add some slight counter-irritation to the warmth and moisture, the fomentation may be sprinkled with turpentine before it is applied. This forms the ordinary *turpentine-stupe*. The sedative action of the fomentation may be increased by sprinkling it with laudanum. The ordinary poppy-fomentation is often used with the same intention. It is thus prepared: Half a pound of poppy-heads with the seeds taken out is boiled for ten minutes in four pints of water, and the liquid then strained off. The decoction is kept warm over a fire, and the flannels dipped in it and applied as before described about every half-hour. The term 'dry fomentation' is sometimes applied to bags of hot salt, bran, or chamomile-flowers; or pieces of flannel toasted before a fire and laid on hot. These often give relief in cases of intestinal, renal, or biliary colic.

MARCUS BECK.

**FOMITES** (*fomes*, touch-wood, tinder).—Substances capable of retaining contagium-particulae, and thus of being the means of propagating any infectious disease. The most important fomites are bed-clothes, bedding, woollen garments, carpets, curtains, and letters. See **CONTAGION**.

**FOOD.**—See **ALIMENT**; and **DIET**.

**FOOD, Ætiology of.**—See **DISEASE**, Causes of; and **DIGESTION**, Disorders of.

**FORAMEN OVALE, Patency of.**—See **HEART**, Malformations of.

**FORCIBLE FEEDING.**—In the treatment of insanity, and sometimes under other circumstances, it happens that we are compelled to administer food by force to patients who, for one reason or another, refuse to take it. The majority of these persons are melancholics who think they ought not to eat, or try to commit suicide by starvation, and many are in a feeble bodily

condition when they commence this refusal. Such persons must be fed by force. Strong patients may be allowed a longer time, for many refuse from whim or obstinacy, and hunger will overcome their disinclination. Some patients in a state of acute delirium will take no food; they must be fed at once, and it is important that they should be fed without a protracted and exhausting struggle, for they will resist desperately unless completely overmastered. In almost every case feeding should be done early: the sooner it is done, the shorter will be the period during which it will be required. The various plans to be adopted range from merely feeding with a spoon, as one feeds a child, to sending food down the œsophagus with a tube passed through the mouth or nose, the patient being restrained in a chair or on a bed by attendants or by mechanical means. The mode of feeding varies according to the resistance, and no one method is applicable to every case. There is no need to pass an œsophageal tube down the throat of a man whose resistance is passive and easily overcome; on the other hand, we occasionally find patients of great muscular strength and indomitable will, who can hardly be fed with safety in any way except by the stomach-tube. Of these we may speak first.

In what position are they to be fed, sitting or lying down? If they are to be held by attendants, no doubt the recumbent posture is the one in which the latter can exercise most power. But when a man is so strong that, as we are told, five attendants must hold him, a struggle will not be unattended with danger, for the five are not all acting together, and he gets loose now a leg and now an arm, to the great discomposure of the operator; moreover, this struggle repeated three times a day, soon renders him a mass of bruises. He should be placed in a strong wooden chair, and by sheets wound round his body, arms, and legs, he can be fastened to the chair so completely that he is quite incapable of movement; yet he gets no bruise, and the operator acts upon him free from all inconvenience. Some writers advocate feeding by the nose, and prefer this method to passing a tube through the mouth. It is well fitted for quiet patients, who do not make a desperate resistance and only close their teeth, but it has many disadvantages. A long tube may enter the larynx, or may curl forwards and be caught by the teeth. And if the food is passed into the nose by a short tube or funnel, it is often ejected, and a long time is spent in the operation. The time consumed in feeding by the nose is one of the great objections, and much exhaustion is often thus produced. The only advantage of feeding in this way is that we are not compelled to force open the mouth. This is a work of difficulty, if the patient is strong and his

teeth perfect, unless we are provided with the screw-key invented for this purpose: with this there is little difficulty, and only bungling will injure the teeth or gums, if the patient is properly secured and the head held by an attendant, not between his knees, but in his hands. If the œsophageal tube be of good size it cannot enter the larynx. The wooden termination of the tube must be short, so as to allow of its curving. The operator, standing in front, passes the tube through the hole of the gag, inclining it to the patient's left, so as to avoid the vertebræ. It may be held by the tongue, but at the first inspiration the hold is relaxed, and it glides down the œsophagus. The food may be poured down it by a funnel or pumped down by the pump.

The next class of patients, making less resistance, may be fed by Paley's feeder, a glass vessel with a flattened spout which goes over the tongue; the food is emitted, a little at a time, by means of a spring, and finds its way down the gullet. A certain number of patients may be fed by getting a funnel behind the teeth and pouring food into it; and others by holding the mouth open by means of two spoons, and then pouring food in. The objection to the latter method, and to its many modifications, is the time taken up in the operation, whereby great fatigue and exhaustion are produced.

The food must be in a more or less fluid form, and should consist of soups, milk, eggs, and the like. *See* DIET.

G. F. BLANDFORD.

**FORMICATION** (*formica*, an ant).—An abnormal subjective sensation referred to the skin, which is described as of a 'creeping' character, and as resembling the crawling of ants upon the surface. *See* SENSATION, Disorders of.

**FOURTH NERVE, Diseases of.**—Morbid states of the fourth nerve are shown by *spasm* or *paralysis* of the superior oblique muscle, which it supplies.

*Spasm.*—Little is known of over-action of this muscle, except that clonic spasm in it is the cause of rotatory nystagmus when the quick movement of the upper end of the vertical axis is inwards.

*Paralysis.*—Paralysis is not uncommon. Its usual causes are inflammation of the nerve-sheath from cold; hæmorrhage into the nerve; syphilitic affections of the nerve or of its membranes; cerebral tumours, &c., pressing on or injuring the nerve at its origin from the valve of Vieussens, or in its course around the crus; aneurysm; exostoses or growths in the orbit; and degeneration of the nucleus, in common with the nuclei of the other nerves of the ocular muscles, from which it is occasionally paralysed in tabes, in 'progressive external ophthalmoplegia,' in the rare acute inflammation of the nuclei,

and in sudden lesions in this part of the mid-brain. It has also been known to be involved in the rare affection of the muscles of the eyeball in diphtheritic paralysis.

*SYMPTOMS.*—Even in complete paralysis of the superior oblique muscle there is little obvious deviation of the affected eye. Movement downwards is, however, defective, and therefore diplopia exists when the eye is moved below a line which runs obliquely downwards from the healthy to the paralysed side, through the point of mid-fixation. The uncertainty which results from 'defective projection' of the image formed by the affected eye occurs chiefly in looking down, and in the corresponding downward position of the head. It is common for the first discovery of the defect to be that the patient becomes giddy when he goes downstairs, and sees two flights of stairs before him instead of one. The chief visible defect in movement of the affected eye (examined alone) is downwards and inwards, because it is when the eyeball is moved in this direction that the superior oblique has most influence on the vertical position of the eyeball. In the direct downward movement there is slight convergent strabismus, because the inferior rectus lacks the counteraction to its inward traction, so that its tendency to rotate the globe (lower end of vertical axis inwards) is also unopposed and perceptible. The diplopia which exists when both eyes look down is homonymous—that is, the image formed by the affected eye is on the same side as that eye. The left eye (if affected) being higher than the right, its image (the left) appears lower than the right image. The action of the superior oblique being to move the upper end of the vertical axis of the eye inwards, there is, in its paralysis, an abnormal divergence of the upper ends of the vertical axes, and the double images (being always inverted) will converge; their upper ends being nearer together than the lower. This is due to the obliquity of the false image (*e.g.* the left), and this obliquity is greatest when the eyeball is moved to the left and downwards, because in this position the rotatory power of the superior oblique is greater, and the obliquity is least in looking inwards and downwards. Thus the convergence of the images is greatest when the difference in height is least, and *vice versâ*. When the paralysis of the superior oblique has existed for some time, a secondary contraction of the inferior oblique may cause crossed diplopia on looking upwards.

*TREATMENT.*—The treatment of paralysis of the fourth nerve is in the main that of its cause. When due, as it very commonly is, to syphilis, iodide of potassium in full doses, with or without mercury, is necessary. Smaller doses of iodide with quinine or iodide of iron are also useful for rheumatic paralysis. Blisters to the temple are bene-

ficial in the early stages. A little good, but not much, may be effected by applying the constant current from the eyelid to the forehead (Benedikt), a few cells only being used.

W. R. GOWERS.

**FRAGILITAS CRINIUM** (*fragilitas*, brittleness; *crinis*, the hair).—See **HAIR**, Diseases of.

**FRAGILITAS OSSIUM**.—A diseased condition of the bones, in which they are extremely fragile, so that they are liable to fracture from very slight causes. See **BONE**, Diseases of.

**FRAMBŒSIA** (*framboise*, a raspberry).  
SYNON.: Yaws; Fr. and Ger. *Pian*.

DEFINITION.—An exanthem consisting of an eruption of reddish-yellow tubercles, which gradually develop into moist fungous masses, without constitutional symptoms, or with only such debility as results from ulceration and prolonged discharge.

ÆTIOLOGY AND PATHOLOGY.—Yaws is epidemic among the African race, both in their native country and in the West Indies, especially during childhood—then also premonitory symptoms and pyrexia are more distinct. The disease is contagious and inoculable, and presumably due to a microbe; it has no relationship to syphilis, as was once supposed from a likeness to condyloma. It is not hereditary, and does not recur. ‘Parangi’ in Ceylon, and ‘Button Scurvy’ which occurred in Ireland prior to 1851, were diseases closely similar if not identical; and filthy water and excess of vegetable food appeared to promote them.

SYMPTOMS.—There is a period of incubation of from three to ten weeks in frambœsia, and pains in the limbs and *malaise* always precede the outbreak. The tubercles begin with little or no hyperæmia or tenderness, and range in size from that of a pin’s head to a prominent mass two inches in diameter. Some subside without breaking the cuticle or destroying the derma; others become fungous, somewhat spheroidal masses, pinkish in colour, and with a dirty-yellow, sticky, fœtid secretion. Later on the fungus shrinks and is converted into a brownish scab, or ulceration extends deeply and widely into the tissues. The subsidence of the fungus is succeeded by a pigmented stain, and the healing of the ulcer by a dark depressed cicatrix.

Frambœsia selects by preference the face and neck, the joints, the feet, and the genital regions; and often forms a fringe of tubercles or a prominent ridge around the mouth, the nostrils, or the anus. The mucous membrane near these outlets may be affected in like manner. The lymphatic glands in the vicinity become large and tender.

DURATION AND RESULTS.—The duration of frambœsia averages three months, but may be prolonged to one or more years. Spon-

taneous cure is usual; but in cachectic subjects and when its development is irregular the constitution suffers, the excretions become offensive, and there is extreme prostration. Then the joints swell; ulcers with excessive discharge form around them; and the patient may be crippled for life, or may perish from septicæmia or asthenia.

TREATMENT.—The curative measures of yaws advised by the best authorities are cleanliness, generous diet, saline eliminants, and the local use of carbolic-acid lotions, diluted mercurial ointments, or iodoform. In the later stages constitutional remedies, such as mercury in minute doses, iodide of potassium, and tonics, are indicated. As regards prevention, every hygienic improvement is potent. In some parts of Africa and in Fiji a habit of inoculating the young prevails, so as to procure immunity in adult age, when frambœsia is much more severe.

ERASMUS WILSON. E. D. MAPOTHER.

**FRANCE**, South of.—The eastern part (Mediterranean coast) is dry and bracing, with a very clear atmosphere. The chief resorts in it are CANNES, MENTONE, HYÈRES, and NICE. The western part is moist and mild but variable, the principal places in it being ARCAÇON, BIARRITZ, and PAU. See **CLIMATE**, Treatment of Disease by.

**FRANZENSBAD**, in Austria.—Alkaline sulphated waters. See **MINERAL WATERS**.

**FRECKLES**.—SYNON.: *Lentigines*; *lenticele*; Fr. *Ephérides*; Ger. *Sommersprossen*, *Sommerflecken*.—A freckle is a pigmentary discoloration of the skin, which has received its Latin or technical name from a resemblance in colour, figure, and size to a lentil. It varies in tint from yellow to olive, from brown to black; and is met with on the exposed parts of the skin, particularly the face, neck, and hands, and occasionally on the covered parts of the body (‘cold freckles’). It is usually found in children and women in whom the skin is sensitive and delicate, and has obtained its German synonyms from its greater frequency in the summer season. See **PIGMENTARY DISEASES OF THE SKIN**.

ERASMUS WILSON.

**FRÉMISSEMENT CATAIRE** (*frémissement*, purring; *cataire*, connected with a cat).—A physical sign felt on applying the hand over the region of the heart or great vessels in certain morbid conditions, and compared to the sensation conveyed to the hand by the purring of a cat. This sign is more commonly known as ‘thrill’ or ‘purring tremor.’ See **PHYSICAL EXAMINATION**.

**FREMITUS** (*fremitus*, a low noise).—A group of physical signs, elicited by placing the hand over the respiratory organs, while the patient speaks (*vocal fremitus*), or coughs

(*tussive fremitus*); or in certain morbid conditions when the patient simply breathes (*rhonchal* and *friction fremitus*). A fremitus may sometimes also be felt over the cardiac region in connexion with the movements of the heart, when the surfaces of the pericardium are much roughened. Another form of fremitus is a peculiar sensation called *hydatid fremitus*, which may be elicited by a special mode of percussio over hydatid tumours in some cases. See PHYSICAL EXAMINATION.

**FRICITION.**—SYNON.: Rubbing; Fr. *Friction*; Ger. *Reibung*.

**DEFINITION.**—By friction we mean surface-rubbing, as distinguished from *shampooing*, or, as it is sometimes called, *medical rubbing*, a process of manipulation by which deep pressure is made upon the muscles.

**APPLICATIONS AND USES.**—Friction is usefully employed over the surface of a limb, or over the trunk, for a variety of purposes. It is especially useful when the circulation is enfeebled, either by the external application of cold, amounting, when in a severe degree, to frostbite, or in cases of paralysis. The effect is still further increased by the use of various stimulating liniments, more especially when it is desirable to excite a certain amount of counter-irritation over a large cutaneous surface, for the relief of congestion or inflammation of internal organs.

Another object with which friction is largely employed in medicine, is to facilitate the absorption and introduction into the system of various remedial agents applied externally. By this means gastric irritation and disturbance are avoided, and the effects of the remedies upon the system can be more closely watched and regulated. In this way mercury is frequently introduced into the system by the process commonly spoken of as *rubbing in*, and salivation can be more easily avoided or checked at its commencement than when mercury is administered by the mouth. The part of the body selected for this purpose is that along the inner side of the thigh up to the groin; and mercury, in the form of ointment, rubbed in every night and morning, will generally affect the system in a few days.

Another instance of friction is to be found in the fattening of children by the process of rubbing in oil—fresh neat's-foot oil is the best—every night and morning, over the chest, abdomen, arms, and thighs. Emaciated children, thus treated, gain in weight by the absorption of the oil; and not only do they fatten, but their general nutrition and health are improved, often with the diminution of glandular swellings and the disappearance of coughs.

WILLIAM ADAMS.

**FRICITION-FREMITS.**—The form of fremitus produced by the rubbing together of surfaces roughened by various morbid conditions, as of the pleura in breathing, or of the pericardium from the movements of the heart. See FREMITUS; and PHYSICAL EXAMINATION.

**FRICITION-SOUND.**—A physical sign, heard on auscultation, and due to the rubbing against each other of serous surfaces that have lost their natural smoothness and moistness from any cause. See PHYSICAL EXAMINATION.

**FRIEDRICHSHALL**, in Germany. Sulphated waters. See MINERAL WATERS.

**FROSTBITES.**—DEFINITION.—The local effects of severe or extreme cold upon any exposed parts or tissues of the body, more serious than those described as 'chilblains.'

The parts of the body most frequently affected by frostbites are the toes and neighbouring parts of the lower extremities, the fingers and hands, the nose, and the ears, all because of their especial liability to exposure, and the first named because also of their remoteness from the heart, and other disadvantages in their blood-supply.

By far the largest proportion of cases occur among men in or about the prime of life: first, because from the nature of their occupations they are most liable to exposure; and secondly, because from constitutional vigour they better withstand any immediately fatal general effects of exposure, and so survive with local damage.

**SIGNS AND SYMPTOMS.**—The signs and symptoms of frostbite vary in manifestation, in rapidity of occurrence and in succession, with the degree of cold, the medium and mode of application, and the duration of exposure, as also with the general condition of the sufferer. They may, however, be thus described: Transient redness of the part affected, accompanied by hyperæsthesia and tingling sensations, is more or less quickly followed by purplish lividity with diminished sensibility. Blanching, with numbness, supervenes. Next, congelation takes place, signalled by whiteness, hardness, and absolute insensibility. Lastly, the freezing process may extend more and more deeply, and be continued until all the tissues are literally 'frozen to death,' and their recovery is impossible. 'Dry gangrene' is then established, and the parts affected shrivel, dry up, and blacken. If the last stage indicated has not been reached, more or less complete recovery may be effected sooner or later. But 'moist gangrene' is liable to supervene, from inflammation after partial restoration of the circulation in the more severe cases; and other, though less serious trophic lesions, from similar cause, frequently occur in the milder

cases. Parts that have not been actually frozen, but have been subject to repeated or long exposure, may become gangrenous, or may suffer from more or less permanent impairment of nutrition and function, such effects being due to organic damage to the nerves and blood-vessels. See GANGRENE.

**TREATMENT.**—The treatment of a frost-bitten part essentially consists in such measures as are calculated to restore warmth, and re-establish the circulation. The greatest care, judgment, patience, and perseverance are requisite, or the attempts made may only render the mischief worse. The process must be gradual and slow to be safe.

The patient must not be brought at once into a warm room or near a fire, but should be kept in a moderately cold atmosphere, and in a recumbent position. The part affected should be somewhat raised, and gently rubbed with snow or bathed with ice-cold water for some time. Very gradually the temperature of the water may be raised, or oil may be substituted. Later, dry friction may be employed. The frictions should be carried considerably above the part obviously affected. From time to time the affected parts may be lightly enveloped in fur or wool, and the frictions repeated at intervals. After apparent restoration has been effected, treatment by friction, with or without some mildly stimulating liniment, should be persevered with day by day for some considerable period, and the part should be warmly clad. Consecutive inflammation of the skin, with vesication or superficial sloughing, occurs in some cases; in other cases the deeper structures also suffer, and moist gangrene results. Such conditions are especially liable to occur in cases in which the change from cold to warmth has been too rapid. They are to be treated *secundum artem*.

If, in spite of all efforts, restoration cannot be effected, and any part remains cold, dead, and gangrenous, or if from consecutive inflammation moist gangrene should supervene, it is better to wait for complete or almost complete separation of the dead structures before proceeding to amputation or other operative measures. The adjoining parts remain for very long in such depressed vital condition that, if cut through, they are little likely to heal favourably, and very often slough after fresh injury inflicted by operation wounds.

Parts that have been severely frostbitten, even if preserved, are liable to suffer permanently from more or less serious impairment of nutrition, due probably to damage done to the trophic nerves as well as to the blood-vessels. The skin of such parts is often discoloured, and in some cases is hyperæsthetic or painful, and in others more or less numb. It is prone to become inflamed and to ulcerate; and if ulcerated or wounded, healing takes place very slowly as a rule.

Protection against exposure to cold and injury should be carefully and constantly afforded.

For the constitutional and general treatment of the frostbitten, see COLD, Effects of Severe or Extreme.

ARTHUR E. DURHAM.

**FUMIGATION** (*fumigo*, I smoke).—**SYNON.**: Fr. *Fumigation*; Ger. *Beräucherung*.

**DEFINITION.**—A mode of employing certain medicinal agents, which are capable of being volatilised by heat, the vapour being allowed to escape into an apartment, or to come in contact with articles of clothing and other objects, for purposes of disinfection; or being allowed to act upon the surface of the body for therapeutic purposes. The chief agents which are thus used are sulphur and mercury.

**MODE OF APPLICATION AND USES.**—*Sulphur.*—The mode of using sulphur as a disinfectant is explained in the article **DISINFECTATION**. The sulphurous acid may also be brought into contact with the body, sulphur being burnt in a suitable apparatus. It is sometimes thus used for the cure of scabies and other affections.

*Mercury.*—Mercurial fumigation, general or local, has long been employed in the treatment of syphilis, and many different preparations of mercury have been used in this way.

*General mercurial fumigation* is now usually effected by exposing the body to the fumes of calomel mixed with steam, the steam being added with the twofold object of inducing gentle perspiration, and of rendering the mercurial fumes less irritating to the respiratory passages. The best form of mercurial vapour-bath is that recommended by Mr. Henry Lee. The apparatus consists of a spirit-lamp which is enclosed in a case of wire gauze, on the top of which is a small plate surrounded by a porcelain trough. One ounce of water is poured into the trough, and the lamp is then lighted. When the water begins to boil, from 20 to 30 grains of resublimed calomel are spread on the plate, and the apparatus is placed between the patient's legs as he sits on a chair undressed, but surrounded by a moleskin cloak long enough to reach the ground. The cloak is tied round the neck, but kept away from the rest of the body by a cane hoop. If it be thought desirable that the vapour should be inhaled, the slit of the cloak may be opened slightly from time to time. The patient should not be alone during the bath, as it sometimes causes faintness. When the calomel is exhausted, which usually takes about a quarter of an hour, the patient goes to bed wearing the cloak as a nightdress. The bath may be repeated every night, or less often, according to the case and the effect

produced. The state of the gums must be closely watched, and the dose of calomel regulated according to circumstances.

General mercurial fumigation has been employed in all stages of syphilis, but is probably best adapted for the secondary manifestations, especially the widely spread dry eruptions. It may be tried whenever mercury is not well borne by the stomach. See SYPHILIS.

*Local* mercurial fumigation has been found serviceable in treating obstinate affections of the skin and mucous membrane, in the later as well as in the earlier stages of syphilis. From 3 to 5 grains of calomel is the usual quantity for each fumigation.

GEO. G. GASCOYEN. ARTHUR COOPER.

**FUNCTIONAL DISEASES.**—A class of diseases in which no anatomical or structural change can be detected to account for their presence. See DISEASE, Classification of.

**FUNGI, Diseases due to.**—See PARASITES, Vegetable; and MUSHROOMS, Poisoning by.

**FUNGOID** (*fungus*, a mushroom).—A term applied to superficial granulations and morbid growths, especially those of a malignant nature, when they sprout rapidly and assume an appearance somewhat like a mushroom. See CANCER; and ULCER AND ULCERATION.

**FUNGUS-DISEASE OF INDIA.**  
SYNON.: Madura Foot; Mycetoma; *Morbus Tuberculosis Pedis*; Fr. *Dégénérescence Endémique des Os du Pied*; *Pérical*.

**DEFINITION.**—A diseased condition of the hands and feet, occurring in India, characterised by enlargement and distortion of the affected extremity, due to thickening of the cutaneous tissues, with degeneration and subsequent fracture of the osseous structures. Two forms of the malady are described—the one, the pale or ochroid form, characterised by the presence of minute globular fatty particles like fish-roe, and, though very rarely, by the existence of minute pink concretions not unlike red-pepper granules; the other, the melanoid or dark form, characterised by the existence of black or dark brown masses, varying in size from that of a grain of gunpowder to a walnut, and composed of fungoid filaments, cells, and pigmentary deposit.

**DESCRIPTION.**—This remarkable disease had not been observed beyond the limits of Hindostan, until a solitary case of it was recorded within the last few years in Italy. In India it has rarely been seen to affect any but natives. No case of a European or half-caste has been recorded as suffering from a typical form of the malady. The foot has been observed to be affected more often than the hand; hence it was common formerly to find

the malady referred to as one peculiar to the foot. It has been recognised as a distinctive disease in India for more than thirty years, and was described by Goodfrey, of Madras, in the *Lancet* in 1846, and by Eyre in the *Indian Annals of Medical Science* in 1860. It is to Dr. Vandyke Carter, however, that we are chiefly indebted for what is known of the malady, clinically and pathologically, and his writings date as far back as 1860. His published memoir on the disease (*Mycetoma, or the Fungus-disease of India*, 1874) contains a summary of all that had been written regarding it up to the period of publication. The foot or hand affected with the disease presents appearances not unlike those observed in some forms of caries—especially caries of scrofulous origin. When the foot is affected, it is found to be considerably increased in circumference, the enlargement seldom extending far beyond the ankle; the foot is prone to run in a line with the leg, and may be everted or inverted. It is not, however, in the aspect presented by the limb that the leading peculiarity consists, but in the character of the discharges from the sinuses, the openings of which are scattered all over the surface of the affected tissue. It is this peculiarity which led Dr. Carter to separate the disease into two forms—(1) the 'pale' or 'ochroid,' the discharge of which consists of whitish-yellow roe-like bodies of about the size of millet-seed; and (2) the 'dark' or 'melanoid,' so called from the dark brown or even black granular bodies that constantly escape through the sinuses, not unlike grains of coarse gunpowder. The first form may be said to present two or three varieties, according to the modified character of the discharges; these will be referred to more definitely farther on.

The malady would appear to occur more frequently in Madras, Bombay, and the more westerly and north-westerly parts of India than in Bengal proper. This, however, seems to apply more especially to the dark variety; for, whilst no well-authenticated case of this form has been recorded as having manifestly originated in the last-named province, cases of the pale variety are not infrequent. So far as the foot is concerned, the pale form is apparently the one most commonly met with all over India. As regards the hand, however, this would not seem to be the case, for, whilst the writers have had the opportunity of examining two or three hands affected with the dark variety, they have not seen one affected with the pale; nor can they find any account of such a case having been witnessed. The distortion of the hand affected in this manner is very peculiar—it is shortened and thickened, owing to the destruction of the carpus and metacarpus, and the consequent irregular tension of the extensor and flexor tendons.

**ANATOMICAL CHARACTERS.**—On our laying open a characteristic specimen of the dis-

ease, the bones are found to be extremely softened, so that they can readily be divided by means of a common knife. The interior of the hand or foot is occupied by a series of sharply defined cavities, some quite isolated, but the majority communicating with one another and with the exterior, by a series of complex channels, or sinuses, containing glairy fluid and solid concretions in various proportions. Both cavities and channels are lined by a dense glistening membrane, composed of white fibrous and elastic tissues. The surrounding tissues are generally in a very fatty condition, and, where the disease is of long standing, are more or less completely blended into an indistinguishable mass. So far a common description is applicable to both forms of the disease; but on proceeding to the consideration of the contents of the cavities, great differences present themselves.

*Pale form.*—The pale or ochroid form is capable of subdivision into several varieties, according to the nature of its morbid products. In the commonest and most characteristic variety the cavities and channels contain masses of spherical bodies like fish-roe, of a pinkish yellow or white colour, surrounded by gelatinous glairy matter. In certain cases, however, the roe-like bodies are almost or entirely absent, and the gelatinous matter and liquid oil are generally diffused throughout the tissues. In a third and very rare variety the section looks as though besprinkled with grains of red-pepper, from the presence of innumerable minute concretions of a bright red hue.

*Dark form.*—The appearances presented in the dark form of the disease are strikingly different. Here, in place of the roe-like bodies of the previous form, the cavities and channels contain masses of a dark brown or black colour. These masses vary greatly in size, some not being larger than the normal fat-lobules surrounding them, others attaining to the size of a small orange. The larger masses greatly exceed any of the roe-like masses of the pale variety in size, and their consistence is also much firmer than that of the latter. They are tuberculated on the surface, and closely resemble truffles in appearance. On section, they present a more or less distinctly radiating structure, and the interior is generally somewhat lighter in colour than the tuberculated exterior coating. In some cases they are tightly fitted into the cavities in which they lie, but in others they lie loose, and are surrounded by a certain amount of gelatinous matter. The amount of the latter present is, however, much less than in the pale form.

The masses of morbid material in both forms are primarily situated in spaces normally abounding in fat. Long series of them are frequently interpolated among the loculi in the subcutaneous tissue, between healthy

fat-lobules; others occupy the interior of the bones; and a third series are developed in the pads of fat lying around muscles and tendons. The muscles and tendons in such cases may frequently be found quite intact, although surrounded by masses of the morbid material. Due to this persistence, fracture and crushing of the softened bones often occur, and it is on this that the distortion of the affected part is in many cases in great measure dependent.

*Minute characters of the morbid products.* The roe-like particles are composed of a nucleus of granular, waxy consistence, surrounded by a fringe of radiating crystals. They appear to be almost entirely composed of fatty matter, and no traces of the presence of parasitic organisms of any kind can be detected in them. The bright red particles occurring in certain cases of the ochroid form are concretions, consisting in great part of phosphates and carbonates, and containing a considerable proportion of iron.

The dark masses present in the other form of the disease are of much more complex structure. In all, or almost all, cases they contain septate fungoid filaments in greater or less proportion. These are sometimes difficult to distinguish, but may generally be detected by allowing portions of the material to soak for some days in liquor potassæ. The proportion which the filaments bear to the entire mass, when thus separated, is in any case very small, and in some cases extremely so, for on the completion of the soaking only a very small quantity of colourless flocculi, consisting of masses of branched filaments mixed with empty cyst-like cells, is left behind in the fluid. The latter has assumed a brown colour from the solution of the dark mass. The filaments and cysts (*see the accompanying figure*), in so far as tests have yet determined,

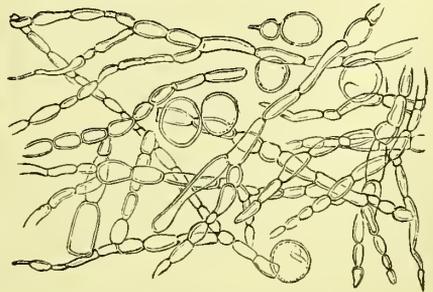


FIG. 58.—Fungoid Filaments and Capsules obtained after prolonged maceration in caustic potash of the dark masses of fungus-disease.  $\times 500$ .

are indistinguishable from undoubted fungal elements. They are, as a rule, quite empty, and show no signs of growth, or indeed of life. The basis in which they are imbedded

varies greatly in different cases. In some instances it is soft and contains much oily matter, but in the more advanced cases this is almost absent. It is then only soluble by means of alkalis. The ash consists mainly of calcium phosphate, and is red, from the presence of oxide of iron.

The fungoid filaments have never yet been shown to be capable of any further development. All attempts at cultivation have failed in causing them to assume any form by which their true nature and relationships may be determined. They have never given any unequivocal signs of life external to the body, even when exposed to conditions favourable to the growth of fungi, as demonstrated by the development of various extraneous moulds upon the surface of the black masses themselves, or on the media in which they were immersed.<sup>1</sup>

**SYMPTOMS.**—Dr. Carter writes: 'As a rule the local indications of this disease are the same for all its forms; for commonly it is not possible to discriminate the several varieties by simple inspection or bare clinical history of the case.' The statements made by the patients as to the mode of origin and progress of these complaints are very various; but, taken generally, they seem to imply that the symptoms are analogous to those usually observed in deep-seated disease of the osseous and adjoining tissues. Eventually a more or less hard lump is felt in the sole of the foot or palm of the hand, or in several places. Generally one or more abscess-like formations occur; and several sinuses are ultimately established, the latter, as a rule, presenting a peculiar mammillated appearance—the 'tubercles,' apparently, of earlier writers. Along with these changes, enlargement and distortion of the affected member take place, but unaccompanied with severe pain. Discharges set in, more or less offensive, according to the nature of the subjacent degeneration, and the limb becomes not only useless, but a burden to its owner. In this manner the sufferer may go on for from one to fifteen or more years, unless relief be sought in a surgical operation.

**COURSE AND DURATION.**—Both forms of fungus-disease run a very chronic course, and often without very materially affecting the general health of the patient; in some cases, however, great emaciation accompanies the disease. With regard to the duration of the malady, it may be stated that cases have been recorded as having existed for various periods up to twenty-six or even thirty years.

**PATHOLOGY.**—The occurrence of the fungoid filaments in the products of the dark variety of the disease has caused many authors to

regard them as the essential cause of it. There are, however, good grounds for rejecting such a conclusion. Had the dark form of the disease been the only one with which we were acquainted, there might have been some cause to regard it as due to parasitic agency. When, however, we find that the pale form, whilst causing all the important lesions present in the other, shows a total absence of all fungoid elements in its products, we are forced to regard such elements as of secondary importance. The only means of overcoming this objection would be a demonstration that the products of the pale form are due to a degeneration of the black matter, in the course of which the fungoid elements disappear. No such demonstration has been given, and, on the contrary, it has been shown that each form is capable of running an entirely independent course, the gradual transformation of the normal fat having been traced in the one case to the production of the roe-like particles, and in the other to that of the black masses in which the fungoid elements are imbedded. Were the pale form the only one known, the disease might be described as a mere degeneration of the fatty tissues, with the results consequent on the presence of the morbid products of the process in the surrounding parts; but this explanation, although so far applicable to the dark form, throws no light on the source of the fungoid elements. They are present in masses which are entirely isolated in the tissues, having no communication with one another or with the exterior. There is no evidence of their passage from one cavity to another—on the contrary, they are absolutely limited to the contents of the cavities, the membranous walls of the latter and the intervening tissues never showing any traces of a spreading mycelium, or of any other fungal elements.

Assuming the filaments to be of undoubted fungal origin, the facts point rather to their simultaneous and independent development in multiple centres, than to their spread from one to another. The fact of the necessity of a suitable soil or nidus, in addition to the mere presence of germs, in order to secure the development of organisms, is generally accepted. That germs of most various kinds must constantly be introduced into the blood, is a self-evident fact. Putting their introduction by means of the lungs out of the question, their constant introduction from the intestinal canal can hardly be denied. It can be demonstrated that the intestinal contents abound in vegetable organisms—spores, bacteria, &c.—in a living condition. As it is an ascertained fact that solid particles of inorganic matter of far larger size than many of these germs can enter the circulation, it can hardly be denied that the latter may, and indeed must, enter also. So long as such bodies do not meet with con-

<sup>1</sup> The later researches of Dr. Carter and others (*Lancet*, July 2, 1892, p. 18) indicate that Fungus-Disease is identical with Actinomycosis. See ACTINOMYCOSIS.—EPITOR.

ditions favourable to development, they are no doubt destroyed and utilised by the living matter of the blood and other tissues. If, however, they are deposited in a medium favourable to them, they will grow and undergo such development as they are capable of. The morbid products of the disease here described are practically dead material, external and extraneous to the body, and it has been experimentally demonstrated that, when removed from the body, they form a basis capable of supporting the growth of fungal organisms. Given these two conditions—the constant presence of germs in the circulation, and the possession of a suitable soil for fungi, and the difficulty of accounting for the presence of fungal elements in the latter appears in great part to be removed.<sup>1</sup>

**TREATMENT.**—There are no instances recorded of a spontaneous cure having been effected, nor have remedial applications proved of material permanent value in either form of the disease. Removal of all the diseased tissue, by amputation of the affected extremity, is the only remedy which meets

with general approval. The subsequent treatment resolves itself into that of an ordinary surgical operation.

D. D. CUNNINGHAM. T. R. LEWIS.

**FUNGUS HÆMATODES** (*fungus*, a mushroom; *αἷμα*, blood; and *εἶδος*, form).—A synonym for soft malignant growths, which are exuberant and highly vascular, and therefore peculiarly liable to bleed. See **CANCER**; and **TUMOURS**.

**FURED, in Hungary.**—Weak alkaline saline waters. See **MINERAL WATERS**.

**FURFUR** (Lat. bran).—A bran-like desquamation of the skin, met with in several cutaneous diseases, more especially pityriasis, psoriasis, and ichthyosis: scurf; dandruff. See **SKIN, Diseases of**.

**FURFURACEOUS** (*furfur*, bran).—A term applied to conditions in which the epidermis is shed in the form of bran-like scales. See **FURFUR**.

**FURUNCULUS** (Lat. a little rascal, dimin. of *fur*, a thief).—A synonym for boil. See **BOIL**.

## G

**GADFLY.**—The popular name for a genus of insects whose larvæ infest man and the lower animals. See **CÆSTRUS**.

**GALACTAGOGUES** (*γάλα*, milk; and *ἄγω*, I move).

**DEFINITION.**—Agencies which increase the secretion of the mammary gland.

**ENUMERATION.**—The most common galactagogues are: Mental Emotions; Local Nervous Stimulation; Warmth; good Food; Alcohol; Jaborandi; the fresh leaves of the Castor-Oil plant; Tonics; and Electricity.

**ACTION.**—When we consider how powerfully mental processes may affect the activity of nerves supplying the secreting structure of glands, we can understand how these may influence the secretion of milk, as of the sweat, the saliva, and the tears. The maternal feelings of joy, as well as the reflex stimulation of the infant's lips, act most rapidly in developing the functions of the breast. Warmth and good diet also play their part in the process. Alcohol in the form of malt liquors, or malt-extract, is a useful adjunct; and so are such tonics as iron, which counteract in some measure the severe drain on the constitutional resources. Little use has yet been made in actual practice of those drugs

which are specially credited with galactagogue properties; but we are told on good authority that a poultice made of the fresh leaves of the castor-oil plant, aided by teaspoonful doses of a fluid extract prepared from the same, has a markedly stimulating influence on the mammary secretion. Belladonna is well known to dry up the milk; and jaborandi, which is its antagonist in so many respects, has been shown to have here also a directly opposite effect, and to be a drug of which further use may yet be made when we wish to excite or re-establish the proper functions of the mammary gland.

ROBERT FARQUHARSON.

**GALACTIDROSIS** (*γάλα*, milk; and *ἰδρῶς*, perspiration).—A term signifying milky perspiration. See **SUDORIPAROUS GLANDS, Disorders of**.

**GALACTORRHŒA** (*γάλα*, milk; and *ῥέω*, I flow).—An excessive flow of milk. See **LACTATION, Disorders of**.

**GALL-BLADDER AND GALL-DUCTS, Diseases of.**—These affections may be considered in the following order: (1) Catarrh; (2) Inflammation and its Results; (3) Stricture; (4) Dilatation; (5) Cancer; (6) Foreign Bodies; (7) Enlargement

<sup>1</sup> See footnote on preceding page.—EDITOR.

of the Gall-bladder; (8) Perforation; and (9) Biliary Fistula.

1. **Catarrh of the Bile-passages.**—This disease very rarely gives opportunities for examination after death. At the period after death at which it is customary to make *post-mortem* examinations in this country, all redness of the duct has usually disappeared; and there are left only swelling and pallor of the mucous membrane, which is covered with a tenacious glassy or purulent secretion. By this swelling and secretion the bore of the duct is often greatly narrowed; and it can be seen that no bile has passed over it for some days, as all colour has disappeared from the affected part of the tube. In judging of this, however, no pressure must have been made upon the gall-bladder during the earlier part of the examination. These appearances are most pronounced in the common duct and the gall-bladder; they are gradually lost in the hepatic duct and its branches in the liver. The process seems most intense at the duodenal end of the gall-duct, and the orifice of the papilla itself is often found plugged by mucus, an appearance which certainly favours the notion that the catarrh is propagated from the stomach and duodenum. This is believed to be the commonest source of catarrh of the bile-ducts. It is also seen in nutmeg-liver and cirrhosis; and a tendency to chronic catarrh is set up by the presence of foreign bodies in the ducts, such as gall-stones.

**SYMPTOMS.**—Jaundice is often the first symptom which draws the attention of the patient to his health in a case of catarrh of the bile-ducts, although in a certain number of cases this is preceded by symptoms of gastric disorder, such as vomiting or sense of sickness, loss of appetite, and furred tongue; or, on the other hand, by diarrhoea. The jaundice lasts usually about three weeks, sometimes as much as six or eight weeks. After this, suspicion should be aroused whether something more than a simple catarrh be not present.

**DIAGNOSIS.**—The diagnosis depends upon the absence of any physical signs indicating organic change in the liver; and on the presence of gastric symptoms. Thus nearly all cases of simple jaundice are diagnosed by some physicians as cases of catarrh of the bile-ducts. As the greater number of the patients recover, very few opportunities are given for verifying this diagnosis; but in those which have been examined, plugs of mucus in the ducts have not infrequently been found. The catarrh caused by gall-stones is lost in the jaundice and pain associated therewith.

**TREATMENT.**—The treatment should at first be directed to the gastric symptoms, beginning with a purgative, followed by a course of effervescent alkaline medicines, and restricted diet. Later on, dilute nitro-hydro-

chloric acid taken before meals is often very useful. Daily enemata of one or two quarts of water, at a temperature of from 60° to 90° F., to be retained as long as possible, have been recommended.

2. **Inflammation and its Results.**—In some cases of typhus and typhoid fever, and in other typhoid states, the gall-duct and gall-bladder become *ulcerated*, or filled with purulent fluid, or covered with *croupous exudation*. The same thing may happen when gall-stones are impacted in the ducts. The gall-ducts are sometimes *obliterated* by fibrous bands passing over them. Sometimes they suffer a congenital obliteration by the over-growth of the fibrous tissue around them.

3. **Stricture.**—Stricture of the gall-duct is occasionally met with. Usually it is associated with the presence of gall-stones. It would seem most probable that it arises from inflammation and ulceration of the duct produced by the transit or impaction of calculi. On the other hand, it is possible, though not probable, that it may be primary, and by impeding the escape of bile give rise to gall-stones. Stricture of the gall-duct causes dilatation of the ducts or gall-bladder behind.

4. **Dilatation.**—The gall-ducts and gall-bladder become dilated whenever there exists an obstruction, either pressing on the ducts from the outside, or formed within them. The first result is dilatation of the ducts behind the obstruction. The gall-bladder becomes much dilated, often filled with a thick green bile. If the obstruction last long, the coloured part of the bile is absorbed, and its place taken by a colourless fluid, either viscid or limpid. This fluid contains neither bile-pigment nor bile-acids, is often albuminous, and contains abundance of mucus. The ducts outside the liver may be enormously distended. It is common to see them as big as the middle finger. Within the liver they are also dilated, but not to so great a degree; and they are more dilated on the left than on the right side. The dilatation of the ducts may become cystic, and sometimes moniliform. The writer (J. W. L.) has always been able to find columnar epithelium in these dilated ducts. In some cases of long-continued obstruction, the contents of the bile-ducts become colourless; in other cases, purulent; and small abscesses form around the bile-ducts, and open into them. These abscesses may be multiple; or, more commonly, only a single large one is formed. The abscess or the dilated gall-ducts may rupture into the peritoneum, and cause fatal peritonitis.

5. **Cancer.**—Primary cancer of the gall-ducts and gall-bladder is sometimes met with; or they may be affected secondarily.

6. **Foreign Bodies.**—Foreign bodies are occasionally met with in the gall-ducts. The most common of all are, of course, *gall-stones*. Much less common are entozoa,

such as the *Distoma hepaticum*, *hydatids*, or the two kinds of *ascarides*, especially lumbrici.

**SYMPTOMS.**—In all these different morbid states it is usually only possible to say at the bedside that the large bile-ducts are obstructed; a more complete diagnosis is commonly impossible. Jaundice is an important symptom, as without it disease of the bile-ducts cannot be diagnosed. It is commonly very intense, the urine being deeply coloured, and the fæces quite colourless. The enlargement of the liver, if present, is commonly uniform, the surface being smooth, and the edges well-defined. The gall-bladder may often be felt at the edge of the right lobe as a rounded tumour; this is then a sure sign of the obstruction of the gall-ducts. In simple diseases of the gall-ducts there is an absence of splenic tumour, of ascites, and of other symptoms of portal obstruction. In many cases, however, diseases of the liver and of the gall-ducts are so intimately bound up together, that they cannot, during life, be separated.

**PROGNOSIS.**—The prognosis, if simple catarrh of the gall-ducts and gall-stones can be excluded, is unfavourable.

**TREATMENT.**—The treatment must be conducted on general principles.

**7. Enlargement of the Gall-bladder.** The gall-bladder cannot be felt in health during life. But it may often readily enough be made out where there exists any obstruction in the common or cystic duct, so that it becomes distended with fluid. It may also be felt when the walls become fibrous or calcified, or the seat of cancer; or when its cavity is filled with gall-stones. A tumour may then be felt under the border of the right lobe of the liver, in the situation of the gall-bladder. When filled with fluid, a rounded, sometimes oblong, sometimes pear-shaped tumour is felt; in other cases it has an irregular shape, or a somewhat rounded outline. The gall-bladder usually enlarges in a direction obliquely downwards and forwards in a line which, drawn from the tenth costal cartilage, crosses the middle line a little below the umbilicus (Mayo Robson). A greatly distended gall-bladder has been mistaken for ascites, and tapped. The diagnosis depends chiefly on the situation of the swelling, and even then the distended gall-bladder may be mistaken for hydatid disease of the liver-substance or of the omentum, or for a tumour of a neighbouring organ, especially the right kidney, which has pressed against the liver. The difficulty of the diagnosis is much increased if the liver be moved from its natural place, for then the position of the gall-bladder becomes uncertain. Bamberger says he has often mistaken a softened cancerous nodule of the liver for a distended gall-bladder. If it be certain that a fluctuating tumour be the gall-bladder, and no jaun-

dice be present, a diagnosis may safely be made of *hydrops cystidis felleæ*, or dropsy of the gall-bladder; but if jaundice be present, or if the tumour do not fluctuate but appear solid, there are then no definite rules for diagnosis: all depends upon the surrounding facts of the case. Dropsy of the gall-bladder is not a dangerous disorder, and requires no treatment; while the prognosis and treatment of the other states depend entirely on their respective causes. Surgical treatment is now extensively employed.

**8. Perforation.**—Perforation of the gall-bladder or of the gall-ducts is generally the result of ulceration, due to gall-stones, inflammation, and other causes. Fatal peritonitis ensues if the perforation occur into the abdominal cavity. Frequently, however, previous adhesions have been formed between the biliary reservoir or duct and the neighbouring organs, as the intestine, or the abdominal wall, and the result of this is—

**9. Biliary Fistula.**—This may exist between the gall-bladder or gall-ducts and the surface of the body, the stomach, colon, or duodenum. Very rarely gall-stones find their way into the urinary tract.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

**GALL-STONES.** — **SYNON.**: Hepatic Calculi; Cholelithiasis; Fr. *Calculs biliaires*; Ger. *Gallensteine*.

**DESCRIPTION.**—Gall-stones are seen in man and most of the vertebrate animals, and in some molluses. They are especially common in oxen. They are found in the biliary passages; most usually in the gall-bladder, or the cystic and common duct; more rarely in the hepatic duct, and in its branches within the liver. They vary in size from fine gravel to concretions five inches long. The largest are commonly single, and then they are rounded or oval in shape. The smaller calculi are usually numerous, being then tetrahedral or wedge-shaped, showing the facets or plane surfaces caused by mutual pressure or friction. They are never lighter than water when first removed from the body. Only after drying do they float. Their consistence when raised to the ordinary temperature of the body becomes much less, so that they can be moulded by the fingers. Their colour varies from white to almost black; most commonly it is brown.

Dr. Thudichum thinks that the nuclei of gall-stones are mostly formed of casts of the hepatic ducts. There is rarely more than one nucleus. Its chemical composition is a compound of lime and bile-pigment, or traces of mucus and phosphatic earths. The chief chemical constituent of human gall-stones is cholesterin; some gall-stones are wholly composed of this substance; most contain 70 or 80 per cent. Other constituents of gall-stones are the bile-pigments, either by themselves or in combination with lime. Very

small quantities of the bile-acids are found, and these are also in combination with lime. It is rare to find gall-stones with any large amounts of carbonate or phosphate of lime, though the ash of nearly all gall-stones shows a large amount of carbonate of lime, the product of the combustion. Traces of copper, iron, and manganese are found in nearly all gall-stones. Lime-salts of the fatty acids are likewise found.

**ÆTIOLOGY.**—Age has an apparent influence in the production of gall-stones: they are exceedingly rare in infancy and childhood, their frequency increases after the age of puberty, and they become still more common after thirty. Women are thought to be more liable than men to gall-stones. In cancer of the liver, gall-stones are certainly very commonly found, while on the other hand in cirrhosis they are scarcely ever seen. Want of physical exercise and indulgence in rich diet seem to favour their production.

**PATHOLOGY.**—What is the cause of the first formation of a gall-stone? It is not simply concentration of the bile, since the cholesterin and pigment remain in solution so long as the bile is unchanged; but the beginning of decomposition of the bile-acids causes a precipitation. The cholesterin is likewise thrown down when the reaction of the bile changes from alkaline to acid. Gorup-Besanez and Dr. Thudichum have kept bile several months, and found the reaction at the end of that time acid, with an abundant sediment. It is thus probable that the retention of bile in the gall-bladder or gall-ducts favours the formation of these concretions. It is also probable that gall-stones may be dissolved spontaneously, as erosions are sometimes seen on them; or they may break up, and thus pass out.

**SYMPTOMS.**—Gall-stones while still in the gall-bladder rarely give any signs of their presence. They are frequently found in the gall-bladders of persons who during life had no symptoms which could be referred to the liver. It is when they begin to leave the gall-bladder, and escape into the cystic and common duct, that symptoms arise of gall-stone colic. They often begin with a dull pain near the liver, with vomiting, rigors, and elevation of temperature; or, quite suddenly, a severe pain in the right hypochondrium comes on, described as shooting, stabbing, burning, &c. The pain extends into the epigastrium, rarely to the left hypochondrium, to the right shoulder, and, according to some, even into the extremities. The pain is very intense, and may give rise to delirium and convulsions in nervous persons, or to hysterical attacks in women. Vomiting is usually present; and, as the attacks most often come on after eating, at first only the food taken is thrown up, and then a colourless mucus. The right hypochondrium is usually very tender, and the

muscles are rigid. The pulse, as a rule, is not increased in frequency, being indeed rather below than above the natural number. In violent attacks the pulse becomes very frequent and small, or almost imperceptible; the eyes are surrounded with dark marks; the nose is pointed; the breath is cool; and cold sweats break out over the body. In this state death may occur, but it is a rare event. A few hours after the attack, the conjunctivæ may show a yellow tinge, which will gradually spread from the upper part of the trunk all over the body. The jaundice is more or less intense according to the shape of the gall-stone—whether completely obstructing the duct, or merely causing a hindrance to the passage of the bile. In some cases jaundice may be altogether wanting, as when the stone is in the cystic duct. The jaundice may last an indefinite time. The duration of the attack of colic itself varies: usually not lasting more than a few hours, it may extend over several days. As soon as the gall-stone reaches the duodenum the attacks are over, the stools become dark, and the jaundice begins to disappear. When the gall-stones reach the intestine, they are commonly evacuated with the fæces; some cases have, however, been recorded in which they were so large that symptoms of intestinal obstruction were caused and death resulted. A gall-stone ulcerating its way into the intestine may give rise to hæmorrhage from the bowels. Other rare symptoms are occasionally associated with gall-stones, as intermittent pyrexia, glycosuria, and enlargement of the spleen.

**DIAGNOSIS.**—The diagnosis of gall-stones is often more or less difficult. Some physicians think that the diagnosis should not be made unless the concretions be found in the stools; and the search for them should be made by passing the fæces through a sieve. It is thought by many that it cannot be made if there be no jaundice present. On the other hand, it has been proved that severe and even fatal attacks have occurred without the occurrence of jaundice or discoloration of the fæces. Recurrent attacks of severe 'spasm' in the right hypochondrium or epigastrium should awaken a suspicion of gall-stones, even if no jaundice follow the attacks. Persistent jaundice is characteristic of cancer rather than of gall-stones, though the two may co-exist. Cancer of the head of the pancreas may readily be mistaken for gall-stones in the common duct.

**PROGNOSIS.**—It is almost impossible to make a trustworthy prognosis in these cases. The physician can never speak confidently, or feel quite happy when treating a case which he looks upon as one of gall-stones.

**TREATMENT.**—The treatment of gall-stones may be discussed under two heads: during the paroxysm of the colic; and between the attacks.

During the *paroxysm* the object is to relieve pain. This may best be done by full doses of morphine; and if this be rejected by vomiting, it may be administered hypodermically. Belladonna may also be used. The patient may be put in a warm bath and kept there, the heat being maintained by the renewal of the warm water. Should these means fail, chloroform or ether may be inhaled.

Between the attacks of biliary colic dieting is of great importance. Limiting, or practically excluding, saccharine and fatty matters sometimes leads to the cessation of attacks, which were previously frequent. A great number of remedies have been proposed: the most popular is Durande's, which consists of three parts of ether and two parts of turpentine; the best plan is to give 10 to 20 minims of this mixture three times a day, enclosed in capsules or pearls. The German physicians have great confidence in the alkaline mineral waters, especially Carlsbad. Some think the benefit is due simply to the large amount of water daily ingested, causing a large flow of bile. Lately, an old plan of treatment has been revived—namely, the administration of large doses of olive oil, as much as six to twelve ounces being given for a dose. Following the administration of the oil, numerous concretions have been found in the stools, which have been mistaken for gall-stones. They have, however, been proved to consist of neutral fats and free fatty acids, with a small proportion of cholesterin. Though the oil does not soften and bring away gall-stones as imagined, yet undoubted relief is sometimes afforded, with diminution in the size of the swollen liver. Dr. George Harley advocates extrusion of gall-stones blocking the ducts by digital manipulation. The obstruction has, in some cases, been overcome by this means, but it is treatment which should be carried out only with great care and gentleness, as the gall-duct, inflamed, softened, and ulcerated by the presence of a stone, might be ruptured by too forcible manipulation. In cases of enlarged gall-bladder from gall-stones, or repeated attacks of severe pain in the hepatic region, with or without jaundice or suppuration of the gall-ducts, the aid of a surgeon should be obtained, with a view to an exploratory operation or cholecystotomy. The cases recorded where this and other operations on the gall-bladder and ducts have been performed justify a hope that in future many lives will be saved which would without operative interference be lost. Other kinds of treatment recommended are by purgatives, as castor oil, or by taraxacum or diluted nitro-hydrochloric acid. Emetics have been employed, but they are dangerous on account of the straining which they cause, and which may lead to the rupture of a vessel.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

## GALLOPING CONSUMPTION.

A popular name for phthisis when it runs an acute or rapid course. See PHTHISIS.

**GALVANISM, Uses of.**—See ELECTRICITY IN MEDICINE.

**GANGLION** (γάγγλιον, a hard gathering).—This term is applied to a variety of somewhat different affections, including: 1. The *simple* ganglion. This is a cystic tumour formed in connexion with the sheath of a tendon. 2. The *compound* or *diffuse* ganglion, which consists of a chronic effusion into the common sheath of a group of tendons, giving rise to a fluctuating swelling. One variety of this contains the so-called melon-seed-like bodies. 3. The term is extended by some writers to enlargements of the bursæ mucosæ. See BURSAE, Diseases of.

**1. Simple Ganglion.**—DESCRIPTION.—The simple ganglion forms a rounded tumour, occasionally lobulated, in the immediate neighbourhood of some tendon. The most common situations are the dorsum of the hand, the dorsum of the foot, the palm of the hand at the root of a finger, and behind the outer or the inner malleolus. The tumour varies in size from a pea to a pigeon's egg. It may fluctuate distinctly, or be so tense as to seem solid. It is not adherent to the skin or to the tendon with which it is in relation. It is painless, but often gives rise to a sense of weakness in the affected part. The wall is composed of a more or less delicate fibrous tissue, fusing with the surrounding areolar tissue, and lined by an imperfect layer of endothelial cells. Its contents are most usually semi-solid, like apple-jelly, but sometimes fluid; they are said to be neither albuminous nor gelatinous, but colloid in character. As to the exact nature of the tumour opinions differ, and probably it is not always the same. It is said to arise in the following ways: 1st. By a hernial protrusion from the sheath of a tendon, the neck of which becomes gradually contracted and finally closed, so giving rise to a cyst in intimate connexion with the sheath. 2nd. Gosselin has described small follicles or sub-synovial crypts, which he believes may become dilated, so as to form ganglia. 3rd. The tumour may be a cyst of entirely new formation.

**TREATMENT.**—Painting with iodine is of little or no use. Forcible rupture of the cyst by a blow or pressure sometimes effects a cure. The best treatment is to puncture the tumour with a clean grooved needle, and to squeeze out the contents, afterwards applying pressure or a blister over the collapsed cyst. This treatment may require to be repeated more than once.

**2. Compound or Diffuse Ganglion.**—DESCRIPTION.—This disease is almost exclusively confined to the sheath of the common flexors of the fingers. It may consist of

a simple dropsy of this sheath, forming an hourglass-shaped swelling in the front of the wrist, the constriction being caused by the annular ligament; or, in other cases, the tumour may contain melon-seed-like bodies, which give rise to a sense of soft crackling when it is manipulated. These melon-seed bodies are smooth, oval and flattish in shape, and of a pearly-white colour. They are of almost cartilaginous toughness, and on section present an appearance of concentric lamination. Under the microscope they are found to be composed of very imperfect fibroid tissue. Their origin is somewhat doubtful. They have been supposed to be due, first, to hypertrophy of the fringes normally found on a synovial membrane, the pedunculated projections so formed being ultimately broken loose by the movement of the tendons; secondly, to the formation of pedunculated warty outgrowths on the synovial membrane, which become free in the same way; thirdly, to fibrinous deposits taking place from the fluid in the ganglion; and, fourthly, to the results of accidental hæmorrhage. When these bodies are abundant, the fluid is usually scanty.

**ÆTIOLOGY.**—Recent observations have shown that in the great majority of cases, if not in all, the compound ganglion of the wrist is a chronic tubercular affection of the synovial membrane. Many cases have been recorded in which it was associated with tubercular disease of the carpus, but which was the primary affection is not certain.

**TREATMENT.**—The treatment of compound ganglion is unsatisfactory. Iodine is useless. Aspiration followed by the injection of iodine has occasionally been of service. An incision made into each end of the tumour, followed by drainage under antiseptic dressing, is often of use. In extreme cases Syme recommended laying the whole cavity open, and allowing it to granulate. This always left much stiffness in the tendons. When melon-seed bodies are present, they must be removed by incision, and the case treated antiseptically, as septic suppuration would almost certainly lead to implication of the wrist-joint.

MARCUS BECK.

**GANGLIONIC NERVOUS SYSTEM, Diseases of.**—See SYMPATHETIC SYSTEM, Diseases of.

**GANGRENE** (*γραινω*, I corrode).—SYNON.: Mortification; Sphacelus; Fr. *Gangrène*; Ger. *der Brand*.

**DEFINITION.**—Gangrene is the arrest of the functions of organic life in a circumscribed portion of the soft parts of the body, leading to the complete death of the same. It implies destruction of a considerable area of tissue *en masse*, as distinguished from molecular disintegration or ulceration.

The whole process of death is included

under the term 'gangrene'; the result being mortification, sloughing, or necrosis of the invaded tissue, organ, or limb.

**CLASSIFICATION.**—*Clinically*, gangrene varies greatly according to its position, cause, extent, the powers of the patient, and the existence of complications. The disease may also be classified *ætiologically* and *pathologically* into *spontaneous* or *traumatic*, *dry* or *moist*, *chronic* or *acute* *gangrene*, *senile gangrene*, and *hospital gangrene*. The division into dry and moist gangrene, although convenient, has little pathological significance, being founded on physical characters. Both dry and moist gangrene may arise from similar causes, and coexist even, in different parts of the invaded structures; and it cannot always be predetermined whether a gangrene shall be of the dry or of the moist form. Dry gangrene is usually dependent on senile changes, chiefly arterial degeneration; and is more limited in extent, and chronic in progress. The parts first affected, having time to dry up as the disease invades those adjacent, become mummified, hard, and black, resembling the knuckle of a Spanish ham. Moist gangrene occurs from venous obstruction; and when a large area of living tissue suddenly mortifies, more especially if it have been previously inflamed or gorged with fluid, the dead parts become rapidly transformed into a deliquescent putrid mass, infiltrated with gas. To this type belong many different forms of gangrene—for instance, that following the obliteration of the main artery or vein of a limb, or their becoming plugged by embolus; or the form called *spreading traumatic gangrene*; or gangrene affecting an internal organ.

**ÆTIOLOGY.**—The causes of gangrene may be local and constitutional, or a combination of both. Amongst *local* causes we find intense inflammation of some organ or tissue, such as occurs in pneumonia, noma, cellulitis, anthrax, or phlegmonous erysipelas. Injuries and mechanical violence of various kinds, caustics, and extremes of heat or cold, as seen in burns and scalds or in frostbite, also lead to gangrene. Gangrene may be produced either as the direct consequence of the long-continued application of cold, or as the result of the subsequent inflammatory reaction. Local arrest of the circulation, as in a limb whose main blood-current is arrested by injury, the application of a ligature to an artery or embolic plugging, a strangulated hernia, an intussusception, or internal strangulation, a ligatured hæmorrhoid, a too tightly bandaged limb, or severe paraphimosis, may lead to gangrene. It may also occur from extravasation of urine or feces. Continued pressure produces a variety of gangrene called *bed-sore*, especially in lowly vitalised parts, and in persons suffering from chronic diseases, or from certain lesions of the nervous centres. See ULCER AND ULCERATION.

One of the most frequent constitutional or general causes of gangrene is deficient blood-supply to the part, dependent upon chronic disease of the arteries, the result of arteritis or degenerative senile changes, whence the term *senile gangrene*. Gangrene may occur in the course of certain diseases, as diabetes, typhus, typhoid, measles, and scarlatina; embolism being in many cases the immediate cause of the complication. The gangrene due to ergotism is probably induced by the effect of the poison on the blood-vessels. A peculiar form of gangrene sometimes met with in the extremities—the *gangrène symétrique* of the French—is one in which no more definite cause can be found than anæmia and an extremely feeble circulation. Loss of nerve-influence is also a cause of gangrene. Acute spreading traumatic gangrene is a manifestation of a hyperacute form of septic infection. And, finally, there is *hospital gangrene—pourriture d'hôpital*—a disease seldom met with now except amongst wounded soldiers accumulated in great numbers in foul hospitals near a battle-field, contagious in nature, and due to a specific septic agency. See RAYNAUD'S DISEASE.

**SYMPTOMS.**—The symptoms of gangrene vary with the species, the extent, and the stage of the disease, and with the rapidity of its progress; and also according as it is dry or moist, incipient or complete. The importance of the tissue or organ invaded, and the presence or absence of complications, such as renal or cardiac disease, or of fever, also create differences.

The earliest symptoms are those of diminished circulation, sensibility, and temperature, together with change of colour in the part affected. Pain is one of the primary symptoms, and often precedes the others. In dry, senile, or chronic gangrene there may in some cases be few symptoms beyond moderate local pain and discomfort, though oftentimes the pain is intense; whilst, when the type is of the moist or acute variety, the symptoms are usually more severe, and have a tendency to lapse into those called typhoid or septicæmic. Between these two forms there is every intermediate grade of severity. The amount of fever varies at the outset, but it is usually considerable. The patient generally suffers much from pain, restlessness, and want of sleep; the appetite is bad; the tongue is loaded; and the pulse is feeble or intermittent. When the disease becomes arrested, the dead portion of tissue slowly separates and drops off, or is removed; very often, however, the disease steadily progresses, and the patient dies from exhaustion, or more rapidly from the absorption of septic matter.

Such being the principal symptoms of gangrene in general, we may next describe the symptoms of the leading varieties of the disease.

*Dry or Chronic Senile Gangrene.*—In the dry form of senile gangrene the part is gradually starved to death by reason of the diminished blood-supply. Embolic plugging may also produce the dry variety. The arterial current is suddenly arrested, while the veins empty the blood from the limb. The limb becomes whitish-mottled and shrunken, then brown and black. Senile gangrene generally occurs in the toes, very rarely in the fingers; and is either spontaneous or excited by some trivial cause. A discoloured patch appears on one of the toes, of a dusky reddish-brown colour, which soon becomes dry and black in the centre. The disease spreads very slowly to the adjacent parts. First, a zone of discoloration appears, from the blood stagnating in them; then they become livid, red, and inflamed; afterwards darker; and, finally, black and dry. One or several toes may be involved, or the foot in whole or in part. The patient generally dies sooner or later, exhausted by pain, and from general feebleness of the vital powers. Sometimes a line of demarcation appears, the dead parts separate, and recovery follows; but as a rule relapses take place.

*Moist or Acute Senile Gangrene.*—This form of senile gangrene is more active in its progress, and more rapidly fatal. It is often ascribed to some slight local irritation or injury, such as an inflamed corn or nail. The affected toe becomes dusky-red, swollen, and extremely painful; soon after the dark hue of absolutely dead tissue appears, following closely the zone of inflammation; and this gradually spreads onwards, till the whole foot or possibly the leg, if the patient survive, becomes implicated. Beyond the gangrenous area the skin is mottled, and presents all the appearances due to impeded circulation. In this type of gangrene, stasis of the blood and coagulation first take place in the capillaries, and extend thence to the smaller arteries. It occurs in elderly persons with feeble circulation, due to fatty heart and atheromatous arteries. Diabetic gangrene is commonly of this form.

*Gangrene from Arterial or Venous Obstruction.*—The most common form of moist gangrene is that due to sudden arterial obstruction of a large vessel, when the collateral supply cannot establish itself with sufficient rapidity. It is acute; and a large portion of the body being at once engaged, the tissues do not, as in dry gangrene, have time to desiccate *pari passu* with the spread of the disease. It may happen after ligature of a main trunk for aneurysm; or by the impaction of a fibrinous clot formed upon the roughened wall of an atheromatous vessel, and consequent plugging of the artery, or by embolism. This form of gangrene often assumes the dry character. An entire extremity may be thus affected. Venous obstruction does not produce gangrene unless

complete. Collateral venous channels usually take the place of obstructed ones with ease. In cases due to embolism—a form rare in the upper limbs, but common in the lower extremities, and more especially at the bifurcation of the tibial arteries—sudden and severe pain marks the onset of the disease. The temperature of the part rapidly falls; livid discoloration and loss of sensibility ensue; the blood stagnates in the veins; the skin mottles; bullæ form, filled with turbid fluid; the colour changes from whitish-grey to green, olive, or black; and the affected tissue at length becomes a deliquescent putrid mass, often crepitating from gas imprisoned in the meshes of the cellular tissue. The tendons, blood-vessels, nerves, and bone resist the disintegrating process longest.

*Gangrene from Inflammation.*—Gangrene as the result of intense inflammation is likewise of the moist type. When it supervenes, the tension, pain, and swelling of the inflamed area subside; the part becomes soft and doughy; the colour changes into dusky violet, brown, or black; and all the evidences of rapid decomposition, varying with the part affected and the acuteness of the inflammation, make their appearance.

*Gangrene as a Complication of General Disease.*—Gangrene is occasionally met with in the later stages of typhoid fever, or even during convalescence; and usually occurs in the lower limbs, though sometimes in the face or in the lungs. It is of vascular origin, and is a most grave complication. In form it may be either dry or moist, and may be of either of two kinds. One is of early occurrence in the progress of the disease, and generally affects the toes symmetrically; the other occurs later, or during convalescence, and is of embolic origin.

Measles is prone in some cases to occasion gangrene, generally affecting the face, the vulva, or the lungs. Scarletina and some forms of erysipelas are also occasionally followed by extensive gangrene. The disease has been also observed during some epidemics of cholera. Gangrene produced by the prolonged consumption of diseased rye-bread chiefly occurs in the male adult, and very rarely in women or children. Other symptoms of ergotism will then be present, gangrene being a late occurrence. Gangrene from ergotism precisely resembles ordinary senile gangrene. It almost invariably appears in the lower limbs; and when a line of demarcation forms, this generally seems to correspond with the nearest joint. In diabetic gangrene no special vascular deficiency can be traced. The vitality of the tissues seems impaired by the glycosuric condition, and some slight accidental cause provokes a gangrenous inflammation.

*Hospital Gangrene.*—Hospital gangrene presupposes a wound. The lesion on invasion ceases to discharge, and becomes covered

with a grey tenacious slough extending from the centre towards the edges, which become much inflamed and everted. The patient rapidly lapses into a typhoid condition; and the issue is generally fatal.

*Acute Traumatic or Spreading Gangrene.* This form also follows upon an injury, which may be either slight or severe. It is generally seen in one of the extremities, originating in a wound and rapidly spreading towards the trunk—*gangrène foudroyante*. It is in many cases intimately associated with, and probably induced by, septic infection of the system. It is usually fatal, and scarcely amenable to any treatment short of very early amputation.

*Visceral Gangrene.*—The gangrene of internal organs will be found described in connexion with the diseases of the several organs.

**PROGRESS.**—The progress of gangrene dependent on constitutional causes is too often from bad to worse; and a fatal issue may supervene without any attempt at repair. The gangrenous inflammation, preceding the actual death of the part, continually invades fresh tissues; the fever increases; a sallow sunken countenance, with rapid thready pulse, sordes, muttering delirium, tympanites, hiccough, subsultus, scanty loaded urine or even suppression, supervene; the weakness increases; and death, preceded by collapse, takes place.

In other cases, especially those dependent on local causes, there is a tendency to limitation; and the process by which this is accomplished is the same for all kinds of gangrene, in every part of the body. In superficial structures a red band of healthy inflammation will be observed at the junction of the living with the dead part, called the *line of demarcation*; active cell-infiltration takes place; and a separating wall of fibrin and young cells is established in the layer of living tissue nearest to the dead structures. This becomes by degrees converted into an ordinary granulating surface, and the dead tissue is detached and cast off. The time required for this separation varies with the bulk of the dead portion, the nature of the tissue, and the vitality of the individual. The risk of septicæmic poisoning is present throughout the entire course of the disease.

**DIAGNOSIS.**—The diagnosis of gangrene of external parts cannot be attended with any difficulty; the evidence of its presence being readily appreciated when the symptoms already described make their appearance.

**PROGNOSIS.**—The prognosis of a case of gangrene will mainly depend on the likelihood of the disease becoming limited, the strength of the patient, and the conditions in which he is placed; which are all at first difficult to determine. The cause of the gangrene, and the presence or absence of organic mischief elsewhere, also exercise a great influence. When the malady is dependent on

a constitutional cause, there will be little tendency to delimitation and a bad prospect of recovery. When the cause is local, and the destruction of tissue neither extensive nor invading a vital part, the prognosis will be favourable. But a local injury, when the conditions are unfavourable, may be followed by a severe and extensive gangrene, or some essential part of the body may become implicated.

**TREATMENT.**—When gangrene is imminent, our first care should be to adopt means to support the vital warmth of the part, and to encourage and relieve the embarrassed circulation. When the disease is fully developed, our attention must be directed to control as far as practicable the spread of the disease; to favour the separation of the dead tissue; and to keep the parts as clean and harmless as possible. We must also anticipate complications, or combat them when they occur; and support the patient's strength by good food, stimulants, and fresh air, together with opiates sufficient to allay pain. A limited traumatic gangrene is to be treated as an ordinary sloughing wound. If a complete portion of a limb be involved, amputation should be performed as soon as the line of demarcation is established. In gangrene due to arterial obstruction, the extremity should be raised to assist the return of venous blood; antiseptic dressings and cotton-wool applied locally; and plenty of bland food administered at frequent intervals. When the whole limb is affected, nature may be assisted by completing the amputation of the part as soon as the limiting line is distinct. In gangrene caused by the ligature or rupture of a main artery, or the pressure of an aneurysm, amputation close to the seat of the lesion may at once be performed, as the gangrene will not extend higher than this point; but even under these circumstances it may often be safer to await indications of a commencing demarcation than to amputate previously. In spreading traumatic gangrene early amputation of the affected limb has been strongly recommended; but as this cannot remove the already poisoned condition of the blood, upon which in all probability the gangrene depends, it is generally a useless measure, as the gangrenous action would invade the stump. It affords, however, the only possible chance. In senile gangrene amputation is sometimes indicated, and the dead parts may be removed near to the demarcating line; but an amputation higher up, as in the thigh, has been advocated, where the prospects of repair due to the more vigorous circulation will be presumably better. In cases where traumatic gangrene is localised and dependent merely upon the violence of the injury, the vitality of the individual will be sufficient to neutralise the mischief, unless it be very extensive. A line of demarcation will appear, and then, but not before,

will it be proper to amputate, unless the part be otherwise hopelessly injured, or a large articulation opened, when immediate amputation is indicated.

For the treatment of gangrene occurring in internal organs the reader is referred to the articles on the diseases of the several viscera.

WILLIAM MAC CORMAC.

**GARGLE** (*γαργαρίζω*, I wash the throat).—**SYNON.**: Fr. *Gargarisme*; Ger. *Gurgelmittel*.

**DEFINITION.**—Gargles are liquids employed for the production of local effects on the throat and pharynx.

**MODE OF GARGLING.**—Gargling consists in taking about a tablespoonful, more or less, of the gargle into the mouth, throwing back the head, and agitating the liquid by the air expelled through the larynx. With some persons the gargle goes little beyond the uvula and base of the tongue; but if the head be thrown well back, the fluid can be made to pass into the cavity of the pharynx, and may even reach the larynx and vocal cords. The tension of the muscles, in thus throwing back the head, is apt to provoke efforts at deglutition, so that sometimes small portions of the gargle may be swallowed; and occasionally the effort terminates abruptly in the patient jerking his head forwards, and expelling the gargle forcibly through the nose.

Dr. H. Guinier, of Caunterets, has demonstrated a method of gargling the laryngeal cavity. His instructions are as follows: (1) Slightly to raise the head; (2) to open the mouth moderately; (3) to protrude the chin and lower jaw; (4) to emit the sound of the double vowel *æ*. These four movements open largely the back of the mouth, lift the velum palati and uvula, separate the base of the tongue from the posterior wall, and allow the liquid used for gargling to gravitate into the cavity of the larynx. One expiratory act is the only respiratory movement that is now possible, deglutition is under control, and the gargle bathes the pharynx and supraglottic portion of the larynx. When it is desirable to gargle the back of the pharynx and posterior nares, the patient must take a mouthful of the gargle, and use it as he lies flat on his back with his tongue drawn forward.

**USES AND COMPOSITION.**—The use of gargles is contra-indicated in parenchymatous inflammations of the tonsils, and in all cases where movement of the fauces causes severe pain; and where, as in some persons, an inability exists to retain liquid beyond the anterior pillars of the fauces: for these it is more convenient to apply the fluid to the fauces and pharynx, either by injection, or in the form of a medicated spray, or else by aid of a brush or sponge.

Gargles vary in composition according to their object. If prescribed with the view of exercising the muscles of the soft palate and

pharynx, and thereby increasing their tone, cold or iced water is generally sufficient. If with the view of reducing and allaying local inflammatory conditions of the throat, solutions of chlorate or nitrate of potassium or of borax (two drachms to eight ounces), or of solution of acetate of ammonium (one part in four), combined with infusion of linseed, thin gruel, or water, and used warm, are the best. When it is desirable to excite the mucous membrane and neighbouring glands to further secretion, and thus reduce local congestion, stimulant gargles are useful, such as the tinctures of capsicum (half a drachm to eight ounces), arnica, myrrh, pyrethrum, and eucalyptus rostrata (two drachms to eight ounces); this class of gargles often relieving the deafness caused by obstruction of the Eustachian tubes, by increasing the pharyngeal secretion. To check excessive secretion, astringents are advisable in the form of the salts of iron and zinc, iron-alum or alum (forty grains to eight ounces), tannin (half a drachm to eight ounces), or infusion of rhatany. If we require to check ulceration, or to dilute and purify foul or putrid secretions from the throat and tonsils, antiseptic gargles will answer the purpose, composed of one or two drachms of liquor potassii permanganatis to a pint of water, or one drachm of solution of chlorinated soda to half a pint of water, or sulphurous acid one part to four, or glycerine of carbolic acid (half an ounce to eight ounces). In cases of syphilitic sore-throat, a gargle of perchloride of mercury (three grains to eight ounces) has been recommended. In many cases combinations of different kinds of gargles are beneficial. Water, barley-water, rose or orange-flower water sweetened with a little honey, glycerine, or syrup, constitute the bases of most gargles. JOHN C. THOROWGOOD.

**GASTEIN**, in the Austrian Alps. Simple thermal waters. See MINERAL WATERS.

**GASTRALGIA** (*γαστήρ*, the stomach; and *ἄλγος*, pain).—SYNON.: *Gastrodynia*; Fr. *Gastralgie*; Ger. *Gastralgie*; *Magenschmerz*.

**DEFINITION**.—Pain in the stomach, which occurs in various disorders of that organ and related parts.

**SYMPTOMS**.—In acute erythematous gastritis a burning painful sensation affecting the stomach, and not infrequently extending up the œsophagus, is felt almost immediately after taking food or a stimulant liquid. In acute catarrhal gastritis pain is seldom complained of. In ordinary chronic gastritis it is usually absent; or, at most, only a slight heaviness or fullness after food forms a symptom of the malady. In what the writer has named 'eczema of the stomach' the pain is very severe, comes on two or three hours after food, and is temporarily relieved by eating. The same kind of pain may be ob-

served whenever there is profuse mucous secretion, but it is less severe than in cases where gastric disturbance replaces eruptions of the skin. The pain accompanying pyrosis is of a spasmodic character, and is relieved by the ejection of a tasteless fluid. Severe pain is a common accompaniment of atonic dyspepsia occurring in nervous or hysterical persons. It comes on when the stomach is empty, but is often aggravated directly after food, and is relieved by the escape of flatus, and by stimulants. The most severe gastralgia is that accompanying ulceration of the stomach. In this disorder it is referred to one spot, and is likewise often felt in the back. The mere fact that a pain in the epigastric region is confined to one small spot should induce the practitioner to view the case with suspicion. The pain in ulceration is usually absent when the stomach is empty, but comes on from two to ten minutes after food. In other instances a longer period, such as half an hour, elapses; but it is this definite relation to the digestive process that makes the pain of ulceration characteristic of the disease. It is usually said that when the pain comes on directly after food the cardiac region is the seat of the ulcer; and where a longer interval takes place the sore will be found near the pylorus. This, if true as a general rule, is, nevertheless, open to many exceptions; for, as a patient observed, the pain occurred later after taking food in proportion as she improved in health, and the first evidence of a relapse was the shortness of the interval of ease after eating. In cancer the pain is more continuous than in simple ulcer; it is less influenced by the digestive process, and is more diffused. It is, however, no uncommon circumstance to find cancer of the stomach without any complaint of pain. The softer kinds of malignant growth seem to the writer least apt to cause suffering, probably because the peritoneum is less liable in such cases, than in the other forms, to be affected with inflammatory action.

Neuralgia of the stomach is a favourite disease with some authors. There is no doubt that the normal sensibility of the stomach is vastly exaggerated under some conditions, but the writer's observation leads him to believe that neuralgic pain affecting the stomach *independently of other disease*, is a much rarer complaint than is generally supposed (see STOMACH, Diseases of). The writer has met with three different forms of pain in the region of the stomach ascribed to neuralgia, which are evidently of a different nature: (1) Where severe pain has come on at some period of the day at a certain hour, at a considerable period after the commencement of digestion. In many of these cases it has been evident on inquiry that the apparent periodicity was the result of the punctual disposal of the chief meal of the day. They were, in fact, cases of chronic catarrhal gas-

tritis attended with considerable secretion; most of them occurred in gouty men, and might be classed under the head of eczema, rather than of neuralgia of the stomach.

(2) Cases such as the following: A young lady had suffered for many months from agonising pain in the left hypochondrium, coming on at each menstrual period, and resisting all methods of treatment. In this and other similar cases, it will be found that the seat of the pain is really external, in the intercostal nerve, and not in the stomach itself.

(3) Severe attacks of pain in the left hypochondrium and epigastrium in females of a nervous temperament, who had either been born, or had passed most of their lives, in tropical countries. The real seat of the pain in such cases appears to be the colon; there is always constipation; and a mild aperient and tonic treatment is usually effectual in removing or ameliorating the complaint.

S. FENWICK.

**GASTRIC FEVER** (*γαστήρ*, the stomach).—A popular name for a febrile condition attended with prominent gastric symptoms; as well as for typhoid fever. *See* TYPHOID FEVER.

**GASTRIC GLANDS**, Diseases of (*γαστήρ*, the stomach).—*See* STOMACH, Diseases of.

**GASTRIC ULCER** (*γαστήρ*, the stomach).—*See* STOMACH, Diseases of.

**GASTRITIS** (*γαστήρ*, the stomach).—Inflammation of the stomach. *See* STOMACH, Diseases of.

**GASTRODYNIA** (*γαστήρ*, the stomach; and *δύνη*, pain).—A painful affection of the stomach, generally considered as of a neuralgic nature. *See* GASTRALGIA.

**GASTRO-ENTERIC** (*γαστήρ*, the stomach; and *ἔντερον*, the intestine).—This term is applied to those morbid states in which the stomach and intestine are simultaneously affected. The continuity and similarity in structure of these viscera render them peculiarly liable to coincident disease; and this is more particularly noticed in affections of an inflammatory type (gastro-enteritis), or of a degenerative nature. At the same time, as gastritis may occur and run its course without giving any indication that the intestine is involved, so enteritis may be developed with little or no disorder of the stomach. The same causes appear to determine similar diseases in the stomach and in the intestine; but why at one time both should suffer, and at another time one should escape, is not known. Occasionally it would seem that the entire alimentary canal beyond the œsophagus is attacked at once, and the gastro-intestinal catarrh of children is an example of this. Sometimes the disorder

commences in one part of the canal, and spreads until both the stomach and the intestine are involved. *See* INTESTINES, Diseases of; and STOMACH, Diseases of.

W. H. ALLCHIN.

**GASTRORRHŒA** (*γαστήρ*, the stomach; and *ῥέω*, I flow).—An excessive flow of mucus from the lining membrane of the stomach, due to catarrh. *See* STOMACH, Diseases of.

**GELATINIFORM CANCER**.—A synonym for colloid cancer. *See* CANCER.

**GENERAL**.—This word, as employed in relation to medicine, has several applications. Thus we speak of a *general disease*, which is a disease affecting the whole system, as distinguished from a local affection. As further examples may be mentioned *general debility*, *general paralysis*, and *general dropsy*. So, with respect to therapeutics, *general treatment* refers to remedial measures intended to affect the organism as a whole. In connexion with the sciences of therapeutics, pathology, &c., the word *general* is intended to denote the essential principles of these sciences, as distinguished from their special divisions.

**GENERAL PARALYSIS OF THE INSANE**.—*SYNON.*: General Paresis; *Dementia paralytica*; Fr. *Paralysie générale incomplète*; *Périencéphalite chronique diffuse*; Ger. *Allgemeine progressive Gehirnlähmung*; *Paralytischer Blödsinn*.

**DEFINITION**.—A gradual loss of the power of co-ordinated movement, accompanied by gradually increasing mental disturbance and decay.

**ÆTIOLGY**.—The subjects of general paralysis are most frequently of the male sex, and between 30 and 50 years of age. It may be brought on by excessive mental labour, by severe anxiety, by alcoholic or venereal excess, or by any prolonged strain upon the mental organisation. It may also be induced by a blow or other direct injury to the head. Hereditary predisposition is probably not without influence upon its production, but this is not so frequently present as in most other forms of insanity. The disease most frequently attacks persons who have previously been apparently in the enjoyment of vigorous health.

**ANATOMICAL CHARACTERS**.—General paralysis is characterised by atrophic and degenerative changes of the cerebrum chiefly affecting the convolutions of the vertex, of the anterior and middle lobes, and round the olfactory bulbs at the base. These changes involve destruction of the nerve-cells of the outer layers of the cortex, and augmentation of the lymph-connective system. There is also thickening of the membranes, adhesion of the pia to the surface of the convolutions, thickening of the coats of the blood-vessels,

enlargement and sacculation of the perivascular canals, and engorgement with disintegrated material. In advanced cases the degenerative changes affect every part of the nervous system.

**SYMPTOMS.**—*Physical.*—The physical symptoms of general paralysis of the insane are first apparent in the muscles of articulation and expression. There is an occasional thickness of utterance, perhaps observable only in the pronunciation of the more complex sounds, and a peculiar convulsive tremor of the upper lip accompanying the least excitement. A similar fibrillar trembling may also be observed in the tongue, when an attempt is made to hold it out. A loss of freedom of stroke in the handwriting is manifested at an early stage. This loss of muscular control gradually spreads over the whole system, its onward course being, however, not infrequently interrupted by very remarkable though generally brief remissions. Before the patient is laid completely prostrate, the affection both of articulation and of gait strongly resembles the failure of co-ordinate movement produced by drunkenness. In the last stage the patient lies quite helpless; the power of articulation and every kind of voluntary movement are lost; there is also a strong tendency to the formation of bed-sores; and spots of ecchymosis are apt to appear on the application of even gentle pressure. Inequality of the pupils is usual from the commencement of the illness; but they do not often remain long in one condition, sometimes one pupil and sometimes the other being dilated or contracted. A characteristic feature of the disease is the occurrence of congestive or epileptiform attacks, but they are very variable, both in their frequency and in the stage at which they are first observed.

*Mental.*—The mental symptoms generally precede the physical, though some cases occur in which the condition of the mind is not such as to attract attention till some time after the motor symptoms have become obvious. Sleeplessness and general restlessness are usually the first indications of the derangement, and with these may be associated transient states of depression and hypochondriacal fancies. Enfeeblement, shown by forgetfulness and incapacity for continuous thought, is generally an early symptom; and in some cases a gradual increase of this enfeeblement, till absolute fatuity is reached, constitutes the prominent mental symptom. A common condition from the commencement is an inordinate disposition to embark in any, even the most impracticable, undertaking that may be suggested. There is always observed a peculiar facility of disposition, generally good-humoured, but liable to be interrupted by fits of passionate excitement. Frequently there is a very remarkable extravagance, both in thought and act. In most cases acute maniacal attacks

take place, in which the excitement presents a specially extravagant character. The semblance of probability and coherence found in ordinary acute mania is generally absent. The prevailing ideas are of grandeur, colossal size, infinite number, power, wealth, and rank, all heaped together in wild confusion. Every such maniacal attack marks an appreciable step in the progress of mental decay; and the tendency is always more or less steadily to complete fatuity. In some comparatively rare cases, especially where there is a tendency to phthisis, the mental condition is mainly one of depression. Remarkable remissions of all the symptoms are sometimes met with.

**DIAGNOSIS.**—The diseases from which general paralysis of the insane requires to be distinguished are paralysis due to cerebral hæmorrhage, embolism, encephalitis, or tumour of the brain; hysterical and toxic paralysis; syphilitic disease of the brain; locomotor ataxy; alcoholic insanity; senile dementia; and muscular atrophy. The diagnosis is generally easy, if attention be paid to the presence or absence of the convulsive tremors in the muscles of articulation at the commencement; the general and progressive course of the loss of co-ordination; and the peculiar mental facility and extravagance.

**PROGNOSIS.**—Complete recovery seldom, if ever, takes place in general paralysis of the insane. The ordinary duration of the disease is from a few months to three or four years, though cases of ten years' duration occasionally occur.

**TREATMENT.**—In ordinary circumstances the treatment cannot be properly carried out in a private house; removal to an asylum will therefore generally be necessary. Little benefit is to be derived from drugs. The diet ought to be nutritious, but non-stimulating. The food ought to be minced or pulpy, and care should be taken to prevent an accumulation of it in the pharynx, as fatal choking is sometimes produced in that way. If this should be threatened, the tongue should be immediately pulled forward, and the bolus extracted with the finger. On account of the liability to bed-sores, the patient should as long as possible be prevented from lying constantly in bed. JOHN SIBBALD.

**GERMS OF DISEASE.**—This is a phrase in common use, the acceptance of which is various, and often more or less vague. So far as it refers to actual things or objects, they also are probably diverse in nature, though at present our knowledge of many of them is based rather upon conjecture than actual experience.

As a phrase, 'germs of disease' is most commonly used in the following modes:—

(a) A person may be said to inherit the germs of disease when there is reason to believe that the constitutional, general, or

local disease from which he is suffering is of a kind which has been common in the family or stock whence he has descended, and when the disease is one which seems prone to manifest itself in this way. It may, in this sense, be said that a person inherits the germs of gout, scrofula, tuberculosis, cancer, or syphilis; and, in either of such cases (with the possible exception of the two latter), no one would, on reflection, be able to find that he meant anything else than that the patient had inherited a certain general disposition, habit, or bodily tendency, in which, under the influence of slight exciting causes, one of these morbid conditions should be prone to manifest itself.

(b) In a still looser sense, the phrase is sometimes used to signify the mere commencement or initial stage of a certain disease, as when it is said that the germs of a phthisical patient's malady date from a certain catarrhal attack, or that a patient now suffering from a severe brain-affection contracted the germs of his disease in India or elsewhere on the occasion of some slight sunstroke. Here the expression would be used in a purely metaphorical sense.

(c) The phrase is most commonly employed, however, in reference to the real or supposed causes of communicable morbid processes or diseases, either local or general—those which spread either from part to part in the same person, or from person to person in the same community.

In the spread from part to part, as during the 'generalisation' of some malignant new-growth, the agency of 'germs of disease' is, perhaps, not infrequently more imaginary than real. Results are apt to be ascribed to 'infection' where nothing of the kind has been in operation—as when similar perverted tissue-changes may chance to manifest themselves, either simultaneously or consecutively, in different parts of the body, as results of some single or similar underlying cause or tendency.

In the spread from person to person of local or general contagious affections, the same possible source of fallacy has to be borne in mind. We must be upon our guard against ascribing too general an influence to 'germs of disease.' In certain cases these may have been in the first place non-existent, as when such a disease has been 'autogenetic,' and in no sense a derivative of antecedent disease of the same kind. This caution is especially applicable in regard to such an affection as erysipelas—which, although certainly contagious, is also on very good grounds judged to be 'generable,' especially during certain states of lowered health induced by renal disease and some other visceral affections. If it is not so certainly established, it is by many deemed probable, that a similar caution may be necessary in regard to some more general contagious affections, such as

diphtheria, typhoid, typhus, and relapsing fever, and cholera, which, though certainly contagious, may also be autogenetic. On this subject, however, much doubt and uncertainty still prevail.

The consideration of this third use of the phrase 'germs of disease' conducts us naturally to the question as to the nature of the things or objects which may be included under the same name, and this again to the modes in which they operate. But as these are questions which are discussed under CONTAGION, further reference to them here is unnecessary. See also MICRO-ORGANISMS.

H. CHARLTON BASTIAN.

**GERONTOXON** (*γέρων*, an old man; and *τόξον*, a bow).—A synonym for arcus senilis. See ARCUS SENILIS.

**GEYSERS, The, in Sonoma County, California, U.S.A.**—Hot springs, with alum, iron, and sulphur. See MINERAL WATERS.

**GEYSERS, The, in Wyoming Territory, U.S.A.**—Thermal waters. See MINERAL WATERS.

**GIDDINESS** (Sax. *gidig*, turning round).—A synonym for vertigo. See VERTIGO.

**GISSHÜBEL, in Bohemia.**—Acidulous alkaline table water. See MINERAL WATERS.

**GINGIVITIS** (*gingivæ*, the gums). Inflammation of the gums. See MOUTH, Diseases of.

**GLANDERS** (*glans*, an acorn).—SYNON.: *Equinia*; Fr. *Morve*; Ger. *Rotz*. Its associated condition is named farcy; Fr. *Farcin*; Ger. *Wurm*; *Hautwurm*.

DEFINITION.—A contagious febrile disease, associated with the presence of a specific microbe—the *bacillus mallei*; communicated to man from the horse, ass, or mule; characterised by inflammatory lesions of the nasal and respiratory mucous membranes, and of the lymphatic vessels and glands, with general pyrexia, pains in the joints and muscles, and great prostration, and usually accompanied by a pustular cutaneous eruption.

The local manifestations vary in order of appearance, and in comparative severity in different cases. In those in which the nasal and respiratory mucous membranes are earliest or most severely affected, the disease is customarily called 'glanders;' while to those in which the lymphatic system first and especially suffers, the designation 'farcy' is applied. But no sufficient reason exists for considering glanders and farcy as distinct diseases. They are commonly associated; and whichever set of symptoms may appear first, the other, as a rule, sooner or

later follows. Further, it has been proved that the same virus may give rise to either set of symptoms, or to both. And it would seem that the order of appearance is determined in great measure by the mode of communication of the virus, as well as to some extent perhaps by the condition and constitution of the recipient.

Glanders in man is a very rare disease; and it is only within the last sixty or seventy years that its occurrence has been clearly recognised, and its history made out. When it does occur, it proves fatal in a very large proportion of cases. Taking the acute and chronic cases together, 208 out of 245 are recorded as having terminated fatally. Nevertheless, out of 2,026,296 deaths in England and Wales during the four years 1871-1874 no more than 19 were registered as due to glanders, and during the four years 1885-1888 only 18 out of 2,101,755 deaths. The slight decrease in the proportion during the later period is worthy of note as due in measure probably to the more stringent precautions observed. During the year 1889, however, there were recorded 8 fatal cases of glanders out of 518,353 deaths.

**ETIOLOGY.**—There is no evidence to suggest that glanders ever originates in man, whatever may be the case with regard to the lower animals. It is always communicated; and almost always by direct inoculation of virus from a diseased animal. Horses, asses, mules, some carnivorous animals, as lions, tigers, &c., are liable to take the disease; and, among the smaller animals, guinea-pigs, hedgehogs, field-mice, and wood-mice. Oxen seem to enjoy immunity even when experimentally inoculated, as also rats, white mice, &c.; and pigs, dogs, sheep, and rabbits are only slightly susceptible. In some few instances the disease has been communicated from man to man. It is scarcely ever met with except in those who are more or less constantly employed among horses, and who are therefore liable from time to time to come in contact with diseased animals, or the morbid discharges from them. The mode of communication can in most cases be easily traced; and generally the virus is derived from the secretion of the nasal mucous membrane of the diseased animal. The rarity of the disease in man, considered in conjunction with its comparative frequency and the rapidity with which it spreads among horses, would seem to indicate that the virus is more or less fixed, and not readily diffusible through the air, and that man is not very susceptible to its influence.

In most cases the history renders it clear that the virus has been received through some cut or abrasion of the skin or mucous membrane. In some few instances it appears probable that the virus may have been absorbed through the unbroken mucous membrane or skin. The recent sad case

resulting in the death of Dr. von Hofman, of Vienna, who was engaged in experimental investigations into the propagation of glanders, seems to show conclusively that absorption may take place through the mucous membrane of the respiratory tract from inhalation of the virus; and the observations of Dr. Babès, in Roumania (where glanders appears to be more common than in this country), go to prove that without solution of continuity of skin the glanders bacilli may penetrate by following the hair-follicles, in which they multiply and, proceeding thence, may infect the system.

**SYMPTOMS AND COURSE.**—However the virus may have been communicated, a period of incubation varying from three to eight days, and in some rare instances prolonged even to three weeks or more, ensues before the symptoms of constitutional infection become manifest. The longer the period of incubation, the less acute, as a rule, is the course of the disease. The duration of the disease, as well as the order of development of the local affections, varies greatly in different cases. Hence the classification, on the one hand, into cases of *acute*, *subacute*, and *chronic glanders*; and, on the other, into cases of *acute* and *chronic glanders*, and *acute* and *chronic farcy*. But nothing like clear lines of distinction can be drawn between any of these classes.

The earliest constitutional symptoms are a sense of general discomfort, fatigue, prostration, and chilliness, with headache, and obscure pains in the muscles and joints. As the disease advances, these pains become more severe, and simulate those of rheumatism. Pyrexia, at first but slight, rapidly becomes established. The pulse is quickened and sometimes full, the skin hot and dry, the tongue foul, the urine scanty and high-coloured; and the patient suffers much from restlessness, sleeplessness, and loss of appetite, often with obstinate constipation. Sometimes the feverishness is intermittent, but more frequently continued, or intermittent at very irregular intervals. Still later, rigors occur—more severe than such as may have occurred at an earlier period, followed by profuse sour perspirations and clamminess of skin; the pulse becomes very rapid, weak, and compressible; diarrhoea, with very fetid stools, succeeds the constipation; the thirst is excessive; respiration becomes more and more difficult and laboured; low delirium, with tremors, is followed by coma; and death ensues from exhaustion. In the acute form, the disease runs its course in an average period of about sixteen days; some cases have terminated fatally within a week; others have been prolonged for four weeks. In the less acute form the duration may be from six weeks to two months. And in the most chronic form, in which all the symptoms are less severe, the duration may extend over

several months, and in some cases recovery ultimately takes place.

*Local manifestations.*—In association with the constitutional symptoms thus indicated, the following local manifestations present themselves, but, as already stated, somewhat differently in different cases.

The wound or abrasion through which the virus has been introduced, or the spot at which it has been applied (for the wound may have healed), becomes inflamed, tense, painful, and surrounded by spreading erysipelatous redness. The ulcer which appears enlarges, assumes an unhealthy, corroded, chancreoid aspect, and discharges dirty sanious and often offensive matters.

The lymphatic vessels of the part become inflamed, and present a knotted, cord-like, and subsequently irregularly nodulated condition—the *farcy-buds* in the human subject. The glands are infiltrated and enlarged, and the whole part is swollen and œdematous. The lymphatic glands and vessels of other parts subsequently become affected, but not perhaps to the same extent as in the horse. Resolution and absorption to a greater or less extent sometimes take place; but much more frequently suppuration of low type, abscess-formation, and the production of foul, ulcerating cavities, with hard irregular edges, and fistulæ follow.

At a variable period in the course of the disease—from within forty-eight hours to the end of three or four weeks—an eruption, regarded as characteristic, appears on the skin, especially on the face, chest, and abdomen. This first shows itself as irregularly scattered collections of red spots, 'which are very small and resemble fleabites, but soon acquire a papular elevation, subsequently rising above the level of the surface, like small shot, and assuming a yellowish colour. They lie each in a kind of hole in the corium, as if the latter had been punched out' (Virchow). They appear to be due to the deposit of some neoplastic material, which subsequently softens and breaks down. By-and-by they become vesicular (some say from the first they are vesicular), and then rapidly sero-purulent, with inflamed livid bases. The surrounding skin is red, swollen, and erysipelatous-like. When close together, these pustules become confluent, and give rise to irregular ulcerated surfaces, with soft, brownish, sloughy coating. Large collections of similar deposit in the subcutaneous tissue give rise to hard, painful, boil-like formations, which, breaking down, lead to extensive sloughing of the skin and deeper structures, with thick, dirty white or sanguineous offensive discharge.

The mucous membranes—and first and especially that of the nose—are sooner or later affected by specific inflammation and ulceration. Whether the inflammatory process as a secondary affection more often begins in them or in the skin is not clearly

made out. But in some cases in man, as commonly in the horse, the disease is probably communicated by application of virus to the mucous membrane of the nose or other part of the respiratory passages, which thus becomes primarily affected.

When the nose is affected (as is always the case primarily or secondarily in the form of the disease especially called glanders), there is first a discharge of comparatively thin, colourless, 'catarrhal' mucus. This soon becomes thicker and coloured; and there is considerable pain, heat, redness, and œdematous swelling about the nose itself and the adjacent parts of the face. Ultimately the discharge becomes thick, sticky, tenacious, and semi-purulent, of a dirty yellowish or brownish colour, and often stained with blood. In all cases probably there is ulceration of the mucous membrane, following tubercle-like deposits in it; and the ulceration sometimes extends so deeply as to lead to perforation of the septum, or partial destruction of the turbinated or palate bones. The ulceration often occurs only in the upper part of the nose; and the mucous membrane of the frontal and other sinuses is liable to be similarly affected.

When the lymphatic and cutaneous systems suffer first and especially, the nasal mucous membrane is not affected until towards the termination of the case; and in some instances death has occurred before this affection of the nose has taken place.

Bronchial catarrh, with rhonchi heard all over the chest, accompanied by severe cough with profuse expectoration, indicates the implication of other parts of the respiratory mucous membrane. The conjunctiva and the mucous membrane of the mouth, gums, fauces, and especially the tonsils, are often affected to a serious extent; so also is the larynx; hoarseness, pain, and difficulty in speaking resulting therefrom.

**ANATOMICAL CHARACTERS.**—The pathological lesions found on *post-mortem* examination are such as might be anticipated from the signs and symptoms manifested during life. To these, however, may be added fluidity of the blood; softness and rottenness of the muscles, with hæmorrhagic abscesses in them (considered by Billroth as characteristic); patches of grey hepatitis in the lungs, or lobular pneumonia; abscesses in the parotid, submaxillary, and cervical glands; and nodules hard or softening down in the liver, spleen, and membranes of the brain.

The characteristically associated bacilli may be found in the nodules (especially in such as are comparatively recent), in the mucous membranes, skin, liver, spleen, &c. They have also been found in the blood and various morbid secretions; but in these they are often very difficult to discover. Flüge describes them as 'thin rods, similar to the tubercle-bacilli, but more uniform in size,

and somewhat broader; like these, they are often slightly curved.<sup>1</sup> This description of Flügge's has been adversely criticised by Fränkel and others, who have worked with the bacilli of glanders. They are much more readily stained than are tubercle-bacilli; are shorter and much thicker; and they often present a single unstained spot midway between the ends, quite unlike tubercle-bacilli. The specimens are best prepared by staining with alkaline methylene blue, and subsequent careful treatment by very dilute acetic acid. When inoculated upon sterilised potato from a farcy bud, they present a characteristic tuberculated chocolate-coloured growth in forty-eight to seventy-two hours, if kept at the temperature of the blood.

**DIAGNOSIS.**—The diagnosis of glanders in man may be difficult in the early stage, particularly if the history be defective and no external wound appear. But when the disease is fully developed, the signs and symptoms, especially if taken in conjunction with the occupation and history of the sufferer, are sufficiently characteristic. In some rare instances, however, in which the constitutional symptoms have been slight, and the local manifestations have very slowly developed themselves, great difficulty has arisen, and the true nature of the disease has not even been recognised until after death. Rheumatism, typhoid fever, and pyæmia, and, in regard to the more chronic cases, syphilis and tuberculosis, are the diseases with which it is said that glanders may possibly be confounded. The presence of the characteristic bacilli, if discovered, may be regarded as pathognomonic and conclusive; and in doubtful cases, at any rate, they should always be sought for in the morbid secretions and discharges.<sup>1</sup>

**PROGNOSIS.**—The prognosis in glanders must be extremely unfavourable. Two or three cases only are on record in which recovery has taken place from the more acute form of the disease.<sup>2</sup> In the more chronic

forms, however, the deaths have been only about fifty per cent. The more slowly and less severely the symptoms develop themselves, and the longer the patient survives, the better is the chance of ultimate recovery.

**TREATMENT.**—The constitutional treatment should be supporting, stimulating, and soothing, and varied from time to time according to the indications afforded. Quinine in large doses, and perchloride of iron with or without perchloride of mercury, may be useful. But at present, although very many drugs have been tried, none has been found having any marked specific effect on the course of the disease, unless indeed exception be made in favour of mercury. Gold reports a case successfully treated by prolonged mercurial inunction. Locally any suspicious wound should be freely cauterised or excised as soon as attention is directed to it. All abscesses and collections of morbid material should, as far as possible, be freely incised, and their contents thoroughly evacuated. The resulting cavities and fistulæ should be frequently and thoroughly washed out with germicide and disinfectant solutions—perchloride of mercury, chloride of zinc, sulphate of copper, or carbolic acid in safe dilution; and antiseptic dressings, covered or not according to circumstances by poultices, should be applied. The operator and those who dress the wounds should wear indiarubber gloves. Inhalations of iodine, or carbolic acid vapours or sprays, should be frequently used, and the nasal passages and cavities should be thoroughly syringed from time to time with disinfectant solutions, as dilute carbolic acid, iodised water, or Condy's solution.

ARTHUR E. DURHAM.

**GLANDULAR DISEASES.**—A general denomination for diseases of glands of all kinds. See BRONCHIAL GLANDS, Diseases of; LYMPHATIC SYSTEM, Diseases of; and MESPENTERIC GLANDS, Diseases of; also the several special glands.

**GLAUCOMA** (γλαυκός, sea-green).—In its modern acceptation, this word is used to include all the conditions, whether acute or chronic, primary or secondary, which are produced by heightened tension or increased fluid-pressure within the eyeball. The word was originally applied only to those cases of heightened tension in which there is a greenish opaque appearance behind the pupil. See EYE, AND ITS APPENDAGES, Diseases of.

**GLEET.**—SYNON.: Fr. *Goutte militaire*; Ger. *Nachtripper*.

**DEFINITION.**—An urethral discharge, milky, viscid, scant in quantity, appearing as a drop at the meatus urinarius or as shreds floating in the urine, composed of leucocytes, epithelium cells of various forms, and crystals of phosphates and urates.

<sup>1</sup> In Eastern Prussia, where glanders is very frequent, an extract (*mallein*) of the *bacillus mallei* (obtained with glycerine at 95° F., from pure growths of the bacillus upon potato) has been employed in the diagnosis of doubtful cases of glanders or farcy in the horse. The extract is filtered free of bacilli, and concentrated; about four minims of this are injected under the skin of the suspected horse. If latent glanders be present, rise of temperature of more than 3° is said to follow within the next eight hours. If the animal be free from glanders or farcy, the temperature is said to rise, if at all, less than 2°. Favourable reports are already given of this method of diagnosis both in Germany and at the Institut Pasteur in Paris. Unfortunately the preparation of the extract is not devoid of danger. The manipulations have proved fatal to two investigators: one of them Dr. Kalning of Dorpat, the original discoverer of *mallein*.

<sup>2</sup> Professor Karl Fränkel, of Königsberg, accidentally inoculated himself in the spring of 1891, and recovered, though after several months' illness.

**ÆTIOLOGY AND SYMPTOMS.**—In most cases of gleet, though some chronic urethral discharges arise from masturbation and excessive venery, a clap has preceded the discharge by some months or years. In such cases the discharge may escape notice for a time without really disappearing altogether, and again become plentiful without obvious cause. Pain is either absent, or a slight tickling or smarting at the parts inflamed is felt during micturition or at other times. But these symptoms vary so greatly in different patients, that much reliance cannot be placed on them for indicating the seat of the inflammation. Nevertheless, when pain is described to be felt in the loins, thighs, or anus, the prostatic portion is generally affected. If the urine is voided in three portions, and the first portion is stocked with floating shreds, while the second and third portions are clear, the discharge is probably formed in front of the prostate. If there be a clear second portion, and a few shreds appear in the third portion, that being also fairly clear, some of the discharge is prostatic. If the third portion is turbid the uvula of the bladder is inflamed.

**Physical signs.**—When seeking for the cause and locality of a gleet, it is well to divide the examination of the urethra into two parts; the pendulous and bulbous portions being examined first, the membranous and prostatic portions secondly. The precise situations of the lesions in the penile portion are ascertained by passing along the urethra a bullet-sound or bougie (No. 25 or 26 of the French scale), or Otis's urethrometer, if the meatus be too small to admit a bullet large enough to expand the passage to its full extent. As the bullet reaches an inflamed spot a slight resistance is noted, and the patient feels pain when the instrument passes over the inflamed or rigid area. Both resistance and pain cease when the area is left behind. Should the inflammation have existed long enough to produce fibrous bands, the onward passage of the bullet-sound is impeded, or arrested if the bands be too short to permit the bullet to slip past. A smaller bullet must be taken, or the urethrometer's bulb must be contracted until the obstruction can be passed. By this means the amount to which the normal capacity of the urethra has been lessened is learned. The bullet-bougie is not usually passed farther than the bulb, though it may be pushed to the bladder by a little manœuvring. A tolerably accurate diagnosis can thus be made, though it is always advantageous, and sometimes indispensable, to complete the examination by ocular inspection. This is done by the endoscope; and, by its means, the condition of the obstructing patches may be more precisely ascertained, and, as well, the absence or presence of inflammation of the glands of Littré and lacunæ Morgagni, or of the

numerous ducts which open into the membranous portions of the canal. See **ENDOSCOPY**.

The conditions causing gleet are: (1) Chronic inflammation of areas of the mucous membrane; (2) Granular patches; (3) Warts; (4) Ulcers; (5) Inflammation of glands and sinuses; (6) Chronic catarrh of the mucous surface and ducts of the prostate; (7) Chronic prostatitis.

**1. Chronic inflammation of the mucous membrane.**—In this, the most common form of gleet, the interior of the urethra, naturally rosy-pink when not inflamed, is dull red or purplish-red in patches or streaks. It must not be forgotten that the mucous membrane of the urethra, especially of the penile portion, after several attacks of inflammation, may assume a greyish or slaty red hue, or a brownish stain from absorbed pigment, when the colouring matter of the blood has escaped into the tissues. Here and there viscid grey flocculi of muco-pus adhere to the inflamed surfaces. The red areas are less elastic than the unaffected parts, and in course of time may develop into bands of stricture-tissue, which more or less diminish the expansile power of the urethra. Common positions for these inflamed areas are the pars cavernosa at two or three inches from the meatus, or at four or six inches from it—*i.e.* in the bulbous portion of the urethra. But the membranous and prostatic portions are also the seats of chronic inflammation in gleet. In long-standing cases white unyielding areas are disposed among the patches of inflammation. They are fibrous toughenings of the tissues. These may remain or disappear. When they are left, some obstruction is caused to the expansion of the urethra—that is, more or less stricture. Usually several months are needed for the patches of inflammation to develop into fibrous bands.

**2. Granular patches.**—These are excessively common in any part of the penile and bulbous portions of the urethra, while they are not rare in the membranous and prostatic portions. They develop on the areas of chronic inflammation, especially if these patches have been excoriated or ulcerated; and it is rare not to find them in a case of six months' duration on the third and fourth inches from the meatus. The bullet-bougie reveals their presence by a slight resistance to the passage of the sound, and by a smarting pain felt while the bullet passes over them. When the sound is withdrawn, it is often marked with a little bloody muco-pus. If the endoscope is then inserted, the granular patches appear as bright carmine-red areas of uneven or velvety aspect, readily bleeding if the instrument be roughly pushed over them. Here and there they are flecked with little dots of greyish mucus, which is very adherent, and which protects the granulation

from the action of astringents. In course of time the granulations may heal into white fibrous scars, or may subside and leave the membrane pale and smooth, but supple as before. Their duration is very long and uncertain, and their cure is tedious.

3. **Warts.**—Warts, which are most commonly situate just within the meatus, may stud the whole length of the urethra. Near the meatus they are arboriform; lower down, sessile, or only slightly pedunculated. Identical in structure with the warts on the glans or in the furrow of the penis, they are ordinary papillomata. They are also frequent in the membranous and prostatic portions. Sometimes a single sessile wart in this position will be the sole cause of a gleet which is troublesome to the patient through its obstinacy, otherwise most harmless.

4. **Ulcers.**—Ulcers are not uncommon in the early stages of chronic urethritis. They appear at any part of the canal, and are indicated by smarting felt at a particular point of the passage during micturition, or when the bullet-sound passes over them. But they can be diagnosed only with certainty by the endoscope. When seen, they appear as minute, round, shallow excoriations, with sharply defined edges and yellow floor. If left untreated, these shallow erosions may heal and contract into thin and somewhat prominent layers of scar-tissue, disposed sometimes longitudinally, more often obliquely or transversely across the urethra. When fully matured they constitute the 'bridle stricture.'

In persons who have tough, well-formed scars of stricture-tissue, ulceration of some parts of this scar-tissue is not uncommon. These ulcers form again and again, and are best treated by thorough dilatation of the strictures, and washing with mild astringent injections.

5. **Follicular sinuses.**—These sinuses, inflamed during acute gonorrhœa, often secrete discharges long after the gonorrhœa has ended. In the anterior urethra generally only a few such crypts are chronically inflamed, but in the prostatic portion several crypts secrete a thin turbid fluid. This form of discharge may be considered to be caused by chronic prostatitis, and it will be described farther on. In cases of hypospadias, at the extreme end of the urethra, close to the margin of the meatus in the glans penis, there is in many persons on each side of the orifice a crypt three-quarters of an inch long. These crypts often continue to discharge pus long after the gonorrhœa has subsided elsewhere. Other sinuses, but shorter ones, open into the interior of the normal urethra. These follicular sinuses do not form indurated patches in the substance of the urethra; hence they never cause stricture. Temporary narrowing may, however, result from the distension of an inflamed and suppurating sub-mucous

follicle, which before the evacuation of its contents may project on the wall of the urethra. The discharge from such a cavity is much more copious than from a simple follicle, and is very slow to dry up, though this end is usually reached if there be no stricture in front of the suppurating follicle to impede the easy flow of the urine past the follicle. Usually the bulbous and membranous parts of the urethra are the slowest to recover.

6. **Chronic catarrh.**—In chronic catarrh of the prostatic urethra, with a history of preceding gonorrhœa, there may be a scanty milky-white discharge seen at the meatus when several hours have elapsed after micturition, or the orifice may be sealed by the dry discharge, or there may be only shreds or flocculi in the urine. In addition to the slight discharge, the patient may complain of occasional great increase of it after hard riding, or dancing, or free indulgence in wine, but this does not always happen. The shreds consist mainly of pus-cells held together by mucus; there are also present epithelial cells of pavement and transitional forms, granular cells, crystals of urates, &c. They have no characteristic value, as similarly formed shreds are washed from granular patches placed in the anterior urethra. Fine filamentous shreds are formed in the folds of the mucous membrane while the urethra is at rest. They are also formed as casts of the prostatic ducts when inflammation has extended into the body of the prostate. These casts are generally built up more of epithelial and granular cells, and less of pus-cells, than those formed in the urethra. The prostatic origin of these shreds is shown by the fact that urine drawn through a catheter from the bladder is bright, clear, and free of shreds. When the mucous membrane of the prostate is only, or mainly, affected, the symptoms of disease are very indefinite, and are often limited to the scanty viscid discharge. Hence no diagnosis can be positive without an examination of the urethra throughout its length.

To examine the urethra with a sound, a No. 25 or 26 of the French scale should be used. If a sound of this calibre will not enter the meatus, it is better to cocaine the mucous membrane within the meatus and nick the orifice at once to a sufficient size; for while the meatus is contracted, the cure of prostatic gleet is almost hopeless. The sound is then passed slowly and gently along the urethra. No pain is felt until the prostate is reached, when a hot or scalding pain is felt, which ceases directly the instrument is withdrawn. The pain is the same whether a curved sound or flexible bougie be used. When the prostate is swollen there is more resistance, but the pain is less scalding to the patient.

If the urethra between the triangular ligament and the orifice into the bladder be

inspected, several changes from healthy conditions may be found. The mucous membrane in health is pale pink, or bluish pink in colour, faintly marked with longitudinal carmine lines, which of course seem to radiate from the centre of the lumen, owing to the funnel-shaped contraction of that part of the urethra which is exposed to view by the endoscope.

When there is chronic inflammation, and the surface has not become indurated, it is of deep carmine hue, closely beset with small eminences which give it a strawberry-like appearance, a resemblance often enhanced by the minute flocculi of pus which adhere to the surface among the prominent granulations, and simulate the yellowish seeds of the ripe strawberry. These vascular excrescences are at first general over the mucous membrane; but, as time goes on, they subside, and are represented by a few papillary tufts or excoriations, which become permanent if not treated. The congested surface bleeds readily, so it must be well mopped, using tincture of hamamelis, if the bleeding is obstinate, before inspecting it or applying other astringents to the surface. Most commonly, in chronic catarrh of the urethra there are excoriations, sometimes granulations, and, rarely, pedunculated warts. If the inflammation has lasted several months, the verumontanum is often shrunken and white, and the orifices of the ejaculatory ducts are invisible or show as dark greyish dots.

7. **Chronic prostatitis.**—If there be general congestion of the organ, and not merely superficial catarrh, the symptoms are more pronounced. In such cases there is usually some pain, which is variously described. Sometimes it consists of a sensation of heat extending along the whole urethra, often radiating to the buttocks, but felt most after micturition. In other persons there is often dull pain in the perinæum, a sense of weight or fulness of the rectum, rather worse by night than by day; aching above the pubes is often complained of. Micturition is called for twice or more by night. By day it is not increased in frequency. When micturition is attempted, the urine is often slow to come, and usually a few drops dribble off after the stream ceases. Walking fatigues easily, and brings on the sense of fulness in the rectum. The finger in the rectum generally finds some swelling and slight tenderness of the prostate. A sound traverses the urethra without causing pain till the bulbo-membranous portion is reached: the instrument is then grasped for a few seconds, to pass on again to the neck of the bladder; here again pain is felt, resistance is made, and the resistance ceases suddenly as the sound enters the bladder, though the pain, of a pressing or squeezing character, still remains. As the instrument is withdrawn, it is expelled rapidly until it is beyond the bulb, where it lies quietly enough and all

pain ceases. When, however, the prostatic mucous membrane is also much congested, the pain attending the passage of a sound is, as already mentioned, hot or burning.

**TREATMENT.**—1. *Inflamed areas.*—When the pain caused by the passage of the exploring sound is acute, the resistance small, and the discharge white and thick, the condition is mainly one of congestion, which is benefited by instillations at the places where the pain is felt. A suitable solution is one of ten to twenty grains of nitrate of silver to the ounce of water, injected by means of a Guyon's bullet-catheter and syringe. This may be repeated every three or four days, while an astringent injection of alum and sulphate of zinc is used in the intervals. If there be cling or hitch as the bullet passes along the urethra, the use of a No. 25 or No. 26 (millimetric scale) steel sound, or even a larger one, twice weekly, is requisite. When no cling exists, the last remains of the discharge can sometimes be dried up by using soluble bougies at night containing chloride or sulphate of zinc in suspension (*porte-remède Reynal*) for ten or fourteen nights. Much may be done with injections by the patient himself when the inflammation is limited to that part of the urethra which extends in front of the triangular ligament. When the disease has extended behind that limit, very few patients can apply an injection to that portion, and the treatment must be carried out wholly by the surgeon. The injections ordinarily used for gonorrhœa are seldom sufficiently astringent to penetrate into the mucous membrane, and solutions having a strength of four or five grains of alum, as much of sulphate of zinc, and of one-fourth or one-half grain of sulphate of copper to the ounce of water, are needful in this chronic inflammation of small areas.

When congestion has produced induration, or even contraction which does not yield to gradual dilatation, the fibrous band should be divided by an urethrotome of suitable shape, until the urethrameter, expanded to the largest size in which it moves freely along the unaffected parts, travels without hitch or cling past the contracted patches. Valuable aid can be obtained by means of the endoscope, through which the inflamed patches can be attacked with mops, used under the control of the eyesight, soaked in aqueous solutions of the nitrate of silver containing 10 to 20 per cent. of the salt. Indeed, many otherwise intractable affections of the urethra are easily managed if treated endoscopically.

2. *Granular patches.*—The treatment of granular patches is tedious and uncertain without the endoscope. Something may be done by instillations of solutions of nitrate of silver of fifteen or twenty grains to the ounce of water, while the patient employs a strong injection between the sittings. But the precise

applications of stronger caustics through the endoscope is far more sure and satisfactory. Brushing the granulations with a 20 per cent. solution of nitrate of silver, or with a 1 per cent. solution of bichloride of mercury in alcohol, after scraping their surfaces with a curette, causes these granulations to disappear after four or five applications, and to leave no trace of their presence. The pain of this method is unimportant, and need not interfere with the patient's ordinary avocations. Careful dilatation of the contractions is also most necessary during the application of the caustics. In treating granular patches, the writers have abandoned the use of soluble bougies and medicated bougies of all kinds, on account of their feeble and uncertain action.

3. *Warts* yield only to topical treatment. When very small, they may be destroyed by pressing a pencil of cupric sulphate against them. They may be nipped off with scissors through the endoscopic tube. If arboriform and pedunculated, the galvanic cauterium is the most effective treatment. It is well to cocaine the urethra by injecting into it ten minims of a 10 per cent. solution of hydrochlorate of cocaine, and allowing five minutes to elapse for absorption, before passing the endoscopic tube through which the topical application is made.

4. *Ulcers* are usually cured by light touches of the cupric sulphate pencil, and by dilating any stricture which may be present.

5. *Follicular sinuses*.—These often continue to secrete pus, which, escaping into the urethra, forms shreds, which appear in the urine. The best treatment is mopping with an astringent solution (10 per cent. solution of silver nitrate), or pressing into the follicle a fine point of cupric sulphate. After this application, the patient should use a strong injection to wash the sinus freely for some days. In time the sinuses cease to secrete discharge even if not treated. But before this cessation takes place, the sinus may become distended and break again, commonly into the urethra as before; rarely the collection of matter may penetrate into the erectile tissue and through the fibrous tissue to the skin, and so reach the surface of the body. When this threatens, the abscess should be promptly incised; it usually closes after evacuation without further trouble.

6. *Chronic catarrh* of the prostatic urethra is little benefited by general treatment or by drugs. Abstinence from stimulants, violent exercise, and sexual excitement are requisite; and when the secretion of pus is abundant, the specifics for catarrh, such as copaiba, cubebs, and buchu, are beneficial. But when there is little more than a shreddy discharge in the urine the proper treatment is topical. The remedies are local instillations, by a Ulzmann's curved catheter syringe, of solutions of ten, fifteen, or twenty grains

of nitrate of silver to the ounce of distilled water directly on to the inflamed surfaces, made at intervals of five or seven days, the length of time being decided by the amount of reaction caused by the instillation. All soreness and increase of discharge which follows the caustic application should subside before another is applied. Usually several such instillations, with solutions increasing in strength, must be made to obtain a cure.

The use of the endoscope renders the treatment more certain and expeditious, as in treating similar patches in the anterior portion of the urethra. By this means the inflamed areas can be distinctly seen, and the remedy applied directly to them. Useful astringents in which to dip the mops are tincture of iodine, solution of perchloride of iron of the British Pharmacopœia, of mercuric chloride (1 per cent. in spirit), or of silver nitrate of 10 or 20 per cent. in water. These applications must be repeated, like the instillations, at intervals of a week or more, according to the amount of irritation they set up. Often, under most favourable conditions, the cure is slow and sometimes very tedious, but it may be always gained with patience.

7. For *chronic prostatitis*, when that is present as well as catarrh of the prostatic urethra, the treatment required is that needful for prostatitis. The general treatment is here of great value, including moderate exercise without fatigue, careful restriction of the diet to unstimulating food, tonics if required, removal of constipation, regular bathing in tepid or cold baths, occupation for the mind without lascivious or erotic excitement, and, when all congestion has been thus removed, cold injections into the rectum. Also, at this stage the passage of full-sized steel sounds, and instillations of solutions of silver nitrate into the previously cocaind prostatic urethra, are among the most effective remedies for this troublesome ailment. Before caustic injections are used it is well to empty the bladder of urine, and to inject eight or ten ounces of some bland fluid, such as cold boiled water, or cold saturated aqueous solution of boric acid, with which the patient may flush out the urethra after the caustic solution has taken effect. This precaution prevents much of the soreness which invariably follows the use of strong astringents in this region. The patient should also keep his bed, or at least his room, for a couple of days after each cauterisation, to lessen the probability of an acute inflammation of the prostate or of the uvula vesicæ following the injection, of which there is always some risk.

BERKELEY HILL. CHARLES STONHAM.

**GLEICHENBERG**, in Austria.—Muriated alkaline waters. See MINERAL WATERS.

**GLOBUS HYSTERICUS** (*globus*, a ball; *hystericus*, connected with hysteria). **SYNON.**: Fr. *Globe hystérique*; Ger. *Hysterische Kugel*.—A subjective sensation experienced by hysterical patients, as of choking, or of a ball rising in the throat. See Hysteria.

**GLOSSALGIA** (γλῶσσα, the tongue; and ἄλγος, pain).—Pain in the tongue. See Tongue, Diseases of.

**GLOSSITIS** (γλῶσσα, the tongue).—Inflammation of the tongue. See Tongue, Diseases of.

**GLOSSO-PHARYNGEAL NERVE, Diseases of.**—The glosso-pharyngeal nerve is distributed to the back of the tongue and the soft palate, the upper portion of the pharynx, the Eustachian tube, and the tympanum; and also to the stylo-pharyngeus, the middle constrictor of the pharynx, the levator palati, and the azygos uvulæ muscles. But the nerve is connected with the fifth, facial, and pneumogastric nerves, and with the pharyngeal plexus; it is certain that many of its terminal branches merely distribute fibres from these other nerves; and the early ideas of the function of the glosso-pharyngeal, founded on its terminal anatomy, are widely erroneous. Although its twigs go to the structures that subserve taste on the back of the tongue, it is certain that these, with those for common sensation in the same part and in the fauces, come from the fifth nerve; and that the fibres that innervate the palatine muscles are derived from the spinal accessory.

**Paralysis.**—Very little is known of the precise effects of paralysis limited to the glosso-pharyngeal nerve, since it is rarely paralysed alone. The middle constrictor of the pharynx is, however, paralysed, and the mucous membrane of the pharynx is rendered insensitive. From their position, the fibres of origin are commonly damaged in conjunction with fibres of the hypoglossal, spinal accessory, and pneumogastric. The common causes of disease in this situation are meningitis, syphilitic and other growths, and bone-diseases. The nerve may also be paralysed from disease of its nucleus of origin in the medulla oblongata, and then commonly suffers in association with the nerves to the larynx, the tongue, and often the lips (see LABIO-GLOSSO-LARYNGEAL PARALYSIS). The sensory part of the nerve may then escape, and only the motor part be paralysed, the chief effect of which is paralysis of the pharynx. The causes of paralysis from disease of the nucleus are slow degeneration, softening, hæmorrhage, and the effects of diphtheria.

**Spasm.**—Nothing is known of separate spasm in the muscles supplied by the glosso-pharyngeal nerve. In conjunction with the other motor and sensory nerves to the pharynx, it takes part in the production of

the spasm of hydrophobia, and in some hysterical phenomena.

**TREATMENT.**—The treatment of disorders of the glosso-pharyngeal nerve is never special, but always that of the cause, and is sufficiently described in the several articles which deal with the above-mentioned ætiological conditions. W. R. GOWERS.

**GLOSSO-PHARYNGEAL PARALYSIS.**—A synonym for labio-glosso-laryngeal paralysis. See LABIO-GLOSSO-LARYNGEAL PARALYSIS.

**GLOSSY SKIN.**—Glossy skin is the name given to a peculiar atrophic condition of the skin, seen principally in the hands, in consequence of irritative lesions of the nerves of the forearm. The skin of the back of the hand, fingers, and even of the palm, is thin, smooth, shining, without furrows, and redder than natural—either uniformly or in patches. The skin seems as if it were stretched over subjacent parts, which are firmer than natural. It may be more or less fissured, or even denuded of epidermis in patches. Secretion of sweat may be increased or altered in quality. The parts may or may not be the seat of neuralgic pains. It was thought by Sir James Paget (who described this condition in the *Medical Times and Gazette* for May, 1864) to be a very rare affection, but it was observed by Weir-Mitchell and his colleagues, in the late American war, no less than nineteen times in fifty cases of partial division of the nerves of the forearm. It may occur also as a sequence of dislocations of the wrist, where the median nerve is compressed or otherwise damaged.

**GLOTTIS, Diseases of.**—See LARYNX, Diseases of.

**GLYCOSURIA** (γλυκός, sweet; and οὔρον, urine).—A condition of urine in which sugar is present; generally used as a synonym for diabetes. See DIABETES MELLITUS.

**GLYCURONIC ACID.**—Glycuronic acid is a substance allied to the carbohydrates, and represented by the formula  $C_6H_{10}O_7$ . It occurs as an abnormal constituent of urine, as the result of ingestion of certain chemical substances, chief among which are camphor, chloral hydrate, butyl chloral hydrate, morphine salts, nitro-benzol, and some of the quinine derivatives. After chloroform (but not ether) narcosis its presence can be detected in the urine of the patient.

The presence of glycuronic acid in urine is of interest as it is liable to be mistaken for grape-sugar (dextrose), like which it is capable of reducing alkaline copper solutions (Fehling's test), as also Böttger's bismuth test (see DIABETES MELLITUS). The possible presence of glycuronic acid ought to be kept

before the mind in the examination of urine for sugar. Dextrose can be readily distinguished from glycuronic acid by its behaviour with yeast, glycuronic acid yielding a negative result when so treated. When pure it is not crystalline. It is readily soluble in water, much less so in absolute alcohol, very sparingly in ether. Its pure solutions are dextro-rotatory. The only reliable test at present known for the positive recognition of glycuronic acid is its isolation from the urine by a process long and tedious.

JOHN HAROLD.

**GOÏTRE.**—*SYNON.*: Bronchocele; Derbyshire Neck; Fr. *Goître*; Ger. *Kropf*; *Struma*.

*DEFINITION.*—Simple hypertrophy, or cystic, fibroid, or fibro-cystic enlargement of the thyroid gland.

*ÆTIOLOGY.*—*a. Locality.*—Goitre is prevalent in magnesian-limestone districts, for example, Derbyshire—hence the name ‘Derbyshire neck.’ Amongst the continental countries it prevails in are France, mostly in Savoy; Germany, mostly in the Black Forest; Austria, mostly in Styria; Northern Italy, mostly about the Alps; Switzerland, mostly in the Valais; and Russia, mostly about the Altai mountains in Siberia.

Goitre is not only indigenous to mountainous districts, as has been supposed, but it is present in some plains—for instance, the plains of Lombardy.

*b. Water-supply.*—Goitre is due in a large number of cases to an impurity in potable water. Snow-water was in former days considered to be the cause, but this opinion is met by the statement that goitre does not exist in Greenland or Lapland, and that it is prevalent in Sumatra, where there is never any snow. The impurity has been stated by many to be due to an excess of lime and magnesia, which theory seems borne out by a geological examination of the localities in which goitre is indigenous in England. In parts of the Indian Punjab, where goitre affects 60 per cent. of the population, 59 grains of lime have been found in a gallon of water, ‘10 grains being an undesirable proportion’ (Professor Frankland). On the other hand, in many limestone localities bronchocele is entirely absent. Scotland, Ireland, Norway, and Sweden are comparatively exempt from the disease, although mountain limestone is found largely present in their geological formation. Several observers have attributed goitre to the presence of iron pyrites in the water-bearing strata. Dr. Thursfield, of Shrewsbury, who has personally investigated the disease in various parts of England, believes that impure water, although not always or necessarily a factor in the production of goitre, is frequently a strongly predisposing cause; and that when this is the case, the impurity in the water which causes goitre is undoubtedly protoxide of iron in carbonic

solution. Thus is explained the fact that goitre is so often found in magnesian-limestone districts; for iron pyrites, when brought into contact with water containing carbonate of lime and magnesia in carbonic solution, passes by a process of oxidation into the soluble carbonate of iron.

*c. Other causes.*—Epidemics of goitre have been notified as chiefly occurring amongst soldiers in France, probably the result of forced marches through goitrous districts, combined with scarcity of food. In such districts the practice of carrying loads on the head seems largely to increase the ratio of the disease. Dr. Thursfield has proved by investigation, spread over many years, that goitre has largely decreased in those districts where the practice of carrying weights on the head has decreased, although there has been no other change in the habits of the natives, nor in the condition of the water of the district. He has found that much walking up and down hills, diminished barometric pressure, and child-bearing are factors in goitrous districts. He believes goitre, if endemic, to be essentially a disease of childhood, disappearing at adolescence unless the above-mentioned causes are brought into play. He has also noted that ‘the influence of heredity, direct, collateral, and atavistic, is most strongly marked’; and ‘that intermarriage in comparatively isolated communities, acting through successive generations, is a potent predisposing cause in the local prevalence of goitre.’

Women in this country are much more liable to suffer from bronchocele than men, perhaps on account of their being more frequently water-drinkers, for in India it has been noticed that both sexes suffer alike. Besides, there is an association between the enlarged gland and the uterine functions. During the time of puberty the disease commonly first attracts attention, although it may occur from any time of life up to forty years of age, children having even been born with a thyroid enlargement.

*ANATOMICAL CHARACTERS.*—The whole of the thyroid gland, or both lobes, or one lobe only, may be the seat of the goitrous enlargement. The isthmus is rarely affected by itself. In exceptional cases an accessory lobe is present, and becomes enlarged. The enlargement may be simple soft hypertrophy of the gland, cystic, or fibroid; or cysts may be found interspersed in the substance of a fibroid enlargement. Cysts are formed from the normal follicles of the thyroid, by their distension with colloid material, the epithelial lining degenerating as the cysts increase in size. The cyst-wall is formed by the interlobular septa and capsule of the gland. In pure cysts the serous fluid which is secreted from the walls replaces the colloid contents. In the fibroid form the connective stroma increases at the expense of the follicles, and

tough bands of nucleated fibrous tissue traverse the organ in all directions, the change generally commencing centrally and extending peripherally. The follicles mostly atrophy, but some may persist in the form of small cysts.

**SYMPTOMS AND COMPLICATIONS.**—The appearance of the swelling, and a sense of fullness in the neck, are often the only symptoms of goitre. Added to the sense of fullness, there may be a feeling of dragging or constriction about the throat. More serious symptoms are sometimes present—namely, dysphagia and dyspnoea. The former is rarely present except when the tumour has reached an immense size, or when it presses on the oesophagus, as happens when the lateral lobes meet behind the gullet. Dyspnoea, a more common symptom, may be due to pressure from the goitre on the front and sides of the trachea (found more commonly in young people, before the tracheal rings have gained much power of resistance), or on both recurrent laryngeal nerves, causing partial paralysis of the abductors of the vocal cords (crico-arytenoidei-postici muscles); or to a portion of the gland passing behind the trachea, or becoming enlarged beneath the sternum. There is often a temporary enlargement of a goitre during the catamenial period and pregnancy; and flooding in childbirth is not uncommon. A tendency to the hæmorrhagic diathesis has been noted. Exophthalmos, with palpitation of the heart, may accompany goitre. See EXOPHTHALMIC GOÏTRE.

Goîtres in some countries are allowed to reach such dimensions that the inhabitants have to support them in bags. A cystic goitre projects externally far more often than the fibroid variety.

Cretinism is met with, but rarely, in the goitrous districts of this country. More than half the number of cretins are born of goitrous parents; but a goitrous enlargement need not necessarily be present in a cretin, for instances of cretinism have been reported in which the gland has been entirely absent; still they have the same ætiological connexion. When the thyroid gland is absent and cretinism prevails, the late Dr. Hilton Fagge believed that fatty tumours will be found, almost without exception, in the posterior triangles of the neck. See CRETINISM; and MYXŒDEMA.

**DIAGNOSIS.**—A soft hypertrophy of the thyroid gland can be diagnosed by its general diffusiveness, and the want of consistent hardness.

The presence of a cystic goitre can be ascertained by the fluctuation of fluid within its walls. Illumination of the sac for the purpose of diagnosis is of no use. Any doubt can be cleared up by passing a very fine trochar into its centre. Owing to the fact that some cystic goîtres contain a large amount of soft trabecular structure, the fluid

withdrawn is likely to be mixed with blood. When the fluid has been entirely withdrawn from a pure cyst, it often happens that blood will be freely discharged from the mucous walls.

A fibroid enlargement of the thyroid gland can be diagnosed by the consistency and hardness of its substance. In some cases, fibroid nodules lie scattered in a general hypertrophy of the gland, and may be of stony hardness. Some difficulty may be experienced in distinguishing between a globular fibroid and a small cystic goitre that has thick walls, both of which lie deeply in the neck; but if the exploratory examination be made, as recommended above, the difference can at once be recognised.

The diseases which simulate goitre are cancer of the thyroid gland; calculus embedded in its substance; lymphadenoma; aneurysm; and fatty and other tumours of the neck. A leading point in making a diagnosis is to ascertain, by directing the patient to swallow, whether the swelling is attached to the trachea. In the cases of cancer and lymphadenoma, the history and general condition of the patient will give a leading clue. In connexion with cancer, it must be borne in mind that a fibroid enlargement of the gland may become the seat of carcinomatous change. When a fibrous goitre closely overlies the carotid artery, it simulates an aneurysm, owing to the forcible pulsation conveyed through its substance.

**PROGNOSIS.**—The prognosis in goitre is favourable. The occurrence of death from suffocation, due solely to the effects of a goitrous enlargement, is rare. It occurs in those countries where little or no treatment is tried, the tumours being allowed to attain an immense size—their weight sometimes reaching several pounds. The disease is more likely to endanger life when the gland passes behind the trachea, and completely embraces it. All the varieties of goitre are amenable to treatment, but especially the cystic. Goîtres have been known to disappear without any treatment. This result may be due to the removal of the affected person from endemic influences; or it may happen when the enlargement has arisen during pregnancy.

**TREATMENT.**—The general rules requiring attention are removal of the patient from a goitrous district, or complete abstinence from the drinking-water, unless it has been ascertained to be perfectly innocuous, also from carrying weights and ascending hills. General medicinal remedies are only of service when the gland is enlarged by fibroid change, and then iodide of potassium (two to three grains twice a day) may be tried. When the hypertrophy is of the simple soft form, blistering, or painting the surface with the tincture of iodine, or the use of iodine ointment, will be sufficient. If the enlargement is fibroid and of

some consistence, then injections of the tincture of iodine into, or setons passed through, the substance of the gland, are most useful. Fluoric acid has been given internally, with or without the injections, in some cases with success. The injection of ℥xv to ʒj of tincture of iodine should be used twice a week, then weekly, and afterwards fortnightly. The greater number of cases answer well to this treatment, whilst on others no impression can be made. The time the treatment takes depends on the size of the goitre; a month is the minimum required for the cure of a very small goitre. The process to be followed when injecting is as follows: Having frozen the skin over the portion decided upon for injection, by means of the ether spray, and care being taken to avoid transfixing any vein, or the trachea, the needle of the syringe is pushed into the substance of the goitre, and the fluid injected slowly. The best form of syringe for the purpose is one similar to that used for hypodermic injection, made with a screw-piston. Pain in the course of neighbouring nerves is sometimes felt during the injection, for instance in the ear, or at the root of a tooth. When the operation is finished, the needle should be rapidly withdrawn, and the skin where the puncture has been made rolled between the finger and thumb, to prevent any escape of the iodine. If the needle be passed well into the substance of the goitre, no fear need be entertained of the formation of an abscess. In favourable cases there will be a gradual enlargement of the tumour, with a slight degree of pain, for some eight or twelve hours; after that time the bronchocele will very slowly decrease in size. As a rule, in two or three weeks' time it will be evident whether the injection is to prove effective. By this form of treatment all scars are avoided. The length of time required for its adoption is often an objection, even when improvement is noted; a case occurred in the writer's experience in which the time needed for the completion of the cure was over a year, the neck being reduced from 17½ to 13½ inches. Setons made of from two to eight lengths of silk form a very serviceable plan for the treatment of fibrous goitres. In employing either injections or setons, there may be a slight rise of temperature, but it is rarely necessary for the patient to discontinue his usual employment.

Binioidide of mercury smeared over the enlargement, the patient being then made to sit with his neck exposed to the rays of the sun for many hours, has proved an effectual remedy in India. A Captain Cunningham, the originator of this plan, treated gratuitously about 60,000 natives in two years. He is said to have never produced salivation, and to have rarely failed in effecting a cure. Ligature of the thyroid arteries has only proved of temporary benefit. Division of the

isthmus has been advised when dyspnœa is present. This operation may cause the rest of the gland to diminish. A goitre may be removed when it endangers life or keeps the patient from work. The operation, though a serious one, is of comparative safety under the antiseptic system, and when each vessel is divided between ligatures. Entire removal of the gland is, unhappily, often followed by myxœdema. See MYXŒDEMA.

The treatment of cystic goitre is of one kind, unless removal of cyst is practised, and is always successful. The plan consists in emptying, or partially emptying, the cyst with a trochar and cannula, and then injecting a solution of iron (two drachms of the solution or tincture of the perchloride to an ounce of water) sufficient to expand the cavity again. The patient should be kept in bed on account of the rise of temperature, after a successful injection, with the cannula plugged, until the third or fourth day, when a suppurative discharge should have been set up. Should the injection not be successful, it can be repeated at intervals of a few days, until a discharge is established. The metal cannula should then be replaced by an indiarubber one (made with a middle layer of webbing), the cannula being cut shorter as the cyst becomes smaller, and poultices applied until the goitre has disappeared. A cure will be effected in one month to four or five months.

During the injection of the iron solution into cystic goitres, it may happen that air passes into a vein which has been wounded by the trochar. As this accident has been followed by instantaneous death, the writer, to prevent it, uses conjointly the two following plans, taking care to avoid superficial veins: (1) A tape is passed round the neck under the base of the goitre, and held tightly by an assistant standing behind the patient, whilst the injection is going on, and for two or three minutes after. (2) The nozzle is fixed at right angles to the body of the syringe, to enable the injection to be made without holding the syringe below the level of the opening into the cyst, thus preventing the injection of air. See THYROID GLAND, Diseases of. PUGIN THORNTON.

**GONAGRA** (γόυν, the knee; and ἄγρα, a seizure).—An attack of gout in the knee. See GOUT.

**GONARTHRTIS** (γόυν, the knee; and ἄρθρον, a joint).—Inflammation of the knee-joint. See JOINTS, Diseases of.

**GONORRHŒA** (γονή, seed; and ῥέω, I flow).—SYNON.: Clap; Blenorrhagia; Fr. *Chaudefuisse*; Ger. *Tripper*.

DEFINITION.—A contagious purulent inflammation, affecting primarily, in men, the urethra, and in women the vaginal mucous membrane. Occasionally the conjunctival

and rectal mucous membranes, to which the nasal has been added on doubtful evidence, are attacked by gonorrhœa. Certain rheumatoid affections and other complications, to be subsequently mentioned, may also attend the disease.

**ÆTIOLOGY.**—True gonorrhœal inflammation is due to inoculation of discharge from a surface already the seat of gonorrhœa. Membranes covered by pavement epithelium, and possessing a rich lymphatic supply, are the most readily affected; those covered by columnar epithelium, *e.g.* the rectum, are not so liable to become diseased; while the mucous membrane of the mouth, stomach, and air passages appears to be exempt. Healthy mucous membranes are as liable to contract the disease as are those the seat of some morbid change. Although it is highly probable that gonorrhœa arises from gonorrhœa alone, there are cases met with of acute purulent inflammation of the mucous membranes of the vagina, uterus, bladder, conjunctiva, &c., where the disease appears to have arisen independently of contagion from specific gonorrhœal pus. It is therefore better, in the present state of our knowledge, to admit the possibility that contagious inflammations of these mucous membranes may originate from contact with pus secreted by acutely inflamed surfaces which have not been primarily affected by gonorrhœal poison. There are, moreover, great difficulties attending the isolation and cultivation of the micro-organism found in acute gonorrhœa—the gonococcus of Neisser. Previous to the discovery of this organism, various authors suggested and described certain forms of vegetable life which they maintained constantly appeared in gonorrhœal pus, and to which they attributed causative properties when inoculated on suitable mucous membranes.

In 1879 Neisser first described a diplococcus found in gonorrhœal pus, which he believes, and states that he has proved by experimental cultivation, to be the proper organism exciting gonorrhœa. This diplococcus, arranged in pairs or fours separated by a clear interval, is found on the surface and in the interior of the pus or pavement-epithelium cells, and also freely floating in the liquor puris; the organism is never found in the nuclei of the cells. The diplococcus is most numerous in the discharge from a recently infected case, and least so when the disease is of long standing. It is not at present proved incontrovertibly that the diplococcus in question is really the specific poison of gonorrhœa. A very similar organism is found in the buccal mucus of some persons; and even in gonorrhœal pus it is often accompanied by other organisms which are at home in discharges not connected with gonorrhœa. The diplococcus gonorrhœa is cultivated with great difficulty. Neisser has cultivated it in

human blood-serum, but many other observers, repeating his experiments, have failed to do so. Neisser declares that he has produced a contagious urethritis by inoculating the cultivated microbes in the human urethra. Bockhart claims to have met with similar success. Other observers state that they have found the gonococcus in the fluid from acutely inflamed joints, and even in the blood itself in cases of rheumatism originating in urethral gonorrhœa. It is theoretically assumed that the organism penetrates and multiplies in the epithelium cells, and advances into the underlying lymphatic spaces. Here it rapidly proliferates, exciting inflammation and exudation, the migrated leucocytes containing the microbes. The process advancing, the diplococci pass into the substance of the corpus spongiosum, to the glands and lacunæ of the urethra, to the erectile tissue of the corpora cavernosa, &c., and light up inflammatory mischief in the parts affected. The facility with which the gonococci pass along the lymphatics explains why certain portions of the urogenital canal, *e.g.* the navicular fossa, bulb, neck of the bladder, and epididymis, are the parts most commonly affected. Moreover, the microbes, aggregated into masses, may be carried away in the lymphatic and blood currents as infective emboli; this would explain the occurrence of gonorrhœal rheumatism, endocarditis, &c.

Adopting the theory of phagocytosis, it may be supposed that the white cells, and others acting as phagocytes, are hostile to the development of the gonococcus, and this may serve, in some measure, perhaps, to explain the fact that different persons and organs are not equally affected by the gonorrhœal poison; in certain persons or organs their innate strength of resistance to infective poisons may be sufficient to destroy the microbes or arrest their development. When the proliferation of the poison takes place mainly in the pus-cells, the microbe has but little power, and consequently the course of the disease is mild; but if the organisms develop freely in the liquor puris the course is usually severe, and specially marked by involvement of lymphatic structures, giving rise to lymphangitis and bubo.

Assuming the gonococcus to be the active exciting cause of the disease, there are yet certain predisposing causes which may exert an influence on the development of the poison, and so augment or modify the severity of the disease.

First, there is no doubt that the urethral mucous membrane differs greatly in its susceptibility to inflame in different individuals. It is, moreover, asserted that both sexes grow accustomed to the contagious secretions of the same individual, so that they neither infect nor are infected by their usual companions, while they readily receive from or impart disease to strangers.

In women the arrangement of the genital mucous membrane favours the retention of secretions coming in contact with it. Among other causes favouring the development of gonorrhœa may be mentioned the presence of gleet, alcoholic excess, and previous attacks. Men with slight hypospadias are specially liable to contract gonorrhœa.

**INCUBATION PERIOD.**—The symptoms of gonorrhœa are not usually manifested until four or five days after infection; presumably the contagion needs this time for multiplication and development before it is capable of exciting inflammation. According to circumstances, the incubative stage may be shortened or prolonged, ranging from two to eight or ten days. As a general rule, the shorter the incubation stage the more severe is the disease. The period of incubation in the case of gonorrhœal conjunctivitis is shorter than that of urethritis, varying from six to seventy-two hours, the average time being about twenty-four hours.

Gonorrhœa differs so considerably in the two sexes, owing to the difference in the seat of the affection, that it will be advisable to describe it in the male and female separately.

(a) **Gonorrhœa in the Male.**—**ANATOMICAL CHARACTERS.**—The fossa navicularis, rich in follicles and their ducts, is the primary seat of gonorrhœa in the male. The poison passing into the ducts remains in security during the period of its development and multiplication, and penetrates into the deeper tissues and lymphatic network. As the disease progresses it gradually passes backwards by direct extension, and penetrates into the deeper tissues and lymphatic network. As the disease progresses it gradually passes backwards by direct extension, but often pauses at the bulb or membranous part of the urethra. In more severe cases the disease may extend to the submucous tissue, the prostatic urethra and prostate, neck of the bladder, epididymis, bladder, and kidneys. Pyelitis and nephritis are less often the result of direct extension of the inflammatory process; more commonly they are the result of sympathetic irritation, especially in cases where the neck of the bladder is involved.

In the acute stage of gonorrhœa, the mucous membrane, where inflamed, will be deeply congested and of a bright red colour; it is swollen, and the epithelium on the surface proliferates and desquamates. A thick, creamy, purulent discharge soon makes its appearance. The dorsal lymphatics are often found enlarged and tender, and the inguinal glands are not infrequently involved and may suppurate. As the disease subsides, the inflammation dies away first in those parts first attacked; the redness will then be less marked, and is often punctiform and patchy. When the disease subsides, the products of inflammation are absorbed by the lymphatics.

In more severe cases the surface epithelium may be destroyed, and small ulcers and erosions formed; these may subsequently

form granular patches (*see* GLEET), and may ultimately give rise to stricture. This necrosis of tissue very rarely extends beyond the submucous tissue. The ulcers may heal without producing any ill effects. If, however, they become chronic, fibrous tissue develops in their bases and gradually tends to contract; in other cases granulations spring up, and may develop into warty growths, similar to those seen round the corona.

From the varieties of intensity of the inflammation, various appearances are produced which have given rise to a distinctive nomenclature being applied to them; thus: (1) In some cases several layers of epithelium-cells are raised into small eminences by serous effusion; these burst and leave small erosions—*phlyctenular gonorrhœa*. (2) The inflammatory lymph, entangling leucocytes in its meshes, may form a continuous, more or less tough, membrane on the surface, which can be peeled off in flakes—*diphtheritic gonorrhœa*. (3) If the discharge is copious, and mixed with much serum and mucus, the case is sometimes spoken of as *catarrhal*.

**SYMPTOMS.**—Clinically, gonorrhœa may be conveniently divided into three stages corresponding with the development, period of vital activity, and destruction of the poison.

During the *first or incubative stage* there is some tickling and itching at the meatus and tip of the penis, and slight scalding on micturition. A feeling of heat, tension, and weight in the testicles and perineum is often complained of. Nocturnal erections and emissions are not uncommon, and the sexual appetite is increased. Micturition is more frequent than usual. These symptoms may be so slight as to be unnoticed by the patient, and only come on during the latter half of the incubative stage.

In the *second or acute inflammatory stage* the signs are pronounced. The lips of the meatus are swollen and red, the urethra is tender to the touch, and the corpus spongiosum is swollen and engorged. The discharge soon makes its appearance; at first it is seropurulent and scanty, but in a very short time it becomes greenish-yellow, thick, creamy, and copious. Micturition is frequent and painful; in consequence of the swelling of the urethral walls, the stream of urine is diminished in size, or it may escape in drops. The pain and scalding are often severe, especially if raw patches are present in the passage, or if the urine is concentrated and highly acid. Swelling and tenderness of the dorsal lymphatics are common, with enlargement of the inguinal glands (sometimes but not usually going on to suppuration). In more severe cases the whole penis becomes tender and swollen, and the subcutaneous veins engorged. As the disease spreads to the erectile tissue of the corpora cavernosa, it becomes infiltrated with inflamma-

tory exudation, and the affected parts are no longer able to be distended with blood. Consequently, erections, which are very troublesome at night, are only partial, and the straining and tightening of the inflamed parts, by the distension of the uninflamed, produces severe pain, and alteration of the shape of the erect penis—chordee.

In some cases copious hæmorrhage occurs from the distended parts. Small quantities of blood, giving a red tinge to the discharge, may also come from ulcerated and eroded areas of the inflamed mucous surface.

Peri-urethral swellings, giving the urethra a beaded feeling, are by no means uncommon, especially at the fossa navicularis and bulb. They are due to follicular inflammation, and to exudation into the spongy tissue round the urethra. If this be very acute, peri-urethral abscess may result.

When the disease spreads to the membranoprosthetic urethra and neck of the bladder, there is almost constant desire to micturate, with great pain and spasm following the act. Towards the end of micturition a few drops of pus and blood may escape. The discharge is less copious, and the urine often turbid from admixture with muco-pus, which forms a plentiful deposit. From inflammatory congestion of the passage micturition may be difficult, the urine dribbling away. In other cases the irritation in the passage excites spasm of the compressor urethrae muscle, resulting in complete retention. When the disease has extended so far back there is distinct heat and pain in the perineum; if the patient have an emission of semen, which is likely on account of the inflammatory irritation of the verumontanum and ejaculatory ducts, the act will be painful, as if something had been torn in the perineum. The semen, instead of escaping in jets, gradually trickles away, and is often mixed with and followed by a few drops of blood. In cases of gonorrhœa, fever may or may not be present; it is usually slight and transient, unless the disease be complicated. The duration of this second stage varies according to the severity of the disease, the treatment adopted, and the care of the patient himself. Usually the acute stage passes off gradually in from three to six weeks. The symptoms are, *cæteris paribus*, more severe in a first attack, but subsequent ones are more likely to become chronic.

After a varying time the acute inflammatory stage of gonorrhœa passes into the *third*. The symptoms may gradually abate, the pain and discharge disappearing, and the disease terminate. In other cases the discharge becomes less in quantity, more watery, and of a sero-purulent nature; the other symptoms disappearing. Lastly, all the signs may subside with the exception of a thin whitish discharge, especially noticeable in the morning. In such cases the disease per-

sists in a patchy form, and constitutes gleet. *See GLEET.*

During the third stage the acute symptoms may at any time be lighted up again if the treatment be abandoned, or in consequence of injudicious conduct on the part of the patient.

DIAGNOSIS.—Acute urethritis is not invariably gonorrhœal; and it may be of great importance, especially in cases likely to lead to legal proceedings, to determine whether the urethral mischief be gonorrhœal or not. Much credence must not be placed on the patient's statements. Certain complications are said only to follow gonorrhœa, and no other form of urethritis; but this is not so clearly established as to be of certain diagnostic value. In cases where a definite opinion as to the venereal nature of the case is necessary, the pus should be examined for the gonococcus by staining with fuchsin or methyl violet. But, as already stated in discussing the ætiology of gonorrhœa, there is still some doubt as to the specific properties of this organism.

Urethritis may be easily distinguished from urethral chancre; but it must be remembered that both conditions may be present. Urethral chancre is nearly always situate just within the meatus, never more than one inch from it; the ulcerated surface can be seen if the lips of the urethra be separated, and a short aural speculum introduced.

The discharge from a chancre is not creamy, but shreddy, and the pain during micturition is stinging and limited to the raw surface. Occasionally a slight muco-purulent discharge, without pain or much swelling, is present during the initial stage of syphilis, but it subsides spontaneously in one or two weeks, and is accompanied by the indurated sore, and succeeded by secondary symptoms.

In balanoposthitis (which may or may not be venereal in origin) there need be no urethral discharge. This can be ascertained by thoroughly syringing away all the pus from under the prepuce, and then squeezing the penis to see if pus escapes at the meatus.

Prostatic and perineal abscesses may discharge into the urethra. In such conditions the discharge, profuse on one occasion, at once subsides to a scanty gleet. They may be diagnosed by the history of the case, and a careful examination of the patient, but it must be borne in mind that they occasionally complicate gonorrhœa.

PROGNOSIS.—The prognosis of gonorrhœa is usually favourable if proper precautions be taken early. But, in spite of attention and care, the disease is sometimes very severe, especially in young lads of lymphatic temperament, or in men of nervous, irritable constitution. Gonorrhœa is said also to be liable to run a severe course in persons who suffer from acne. It is more likely to become

chronic after repeated attacks, and in those who suffer from stricture or have a narrow meatus.

Various complications, to be afterwards referred to, may arise in the course of the disease, and certain sequela follow it. In rare cases gonorrhœa terminates fatally by general pyæmia, or more frequently may lead to joint-affectations of more or less severity.

**TREATMENT.**—The so-called *abortive* treatment of gonorrhœa has, on account of its non-success and attendant risks, been practically abandoned, and calls for very short notice here. It consists in the internal administration of large doses of copaiba or cubebs, or of injections of caustics or strong irritants and astringents. The former are open to the objection that they not infrequently produce constitutional disturbance, and moreover are unsuccessful in attaining the object in view. Strong injections too often aggravate, instead of relieving, the local mischief, and may thereby make the course of the disease more severe and chronic. In the early stages of acute gonorrhœa, rest, if the patient can take it, is important. The bowels should be opened and kept acting by the use of saline aperients, and the diet should be low and of a non-irritating description. All alcohol, especially malt liquors, highly seasoned dishes, great exercise, and sexual excitement should be rigorously avoided. Warm baths and strict cleanliness of the parts, with support of the penis and testes, are useful. The salts of potassium, with tincture of hyoscyamus, are also useful, to diminish the acidity of the urine, and allay the smarting and pain during micturition; the same may, if excessive, be relieved by immersing the penis in iced water during micturition. In extreme cases, cocaine may be injected before micturition. As long as there is much smarting and signs of acute inflammatory congestion, strong astringent injections should be avoided, but relief is obtained by washing out the urethra with tepid water after micturition. One-eighth gr. of permanganate of zinc to one fluid ounce of distilled water is an injection which may generally be employed in the earliest stage. When the acute stage has passed, the continuance of the congestion is shortened by astringent and antiseptic injections, and the internal administration of copaiba, sandal oil, or cubebs. Injections should not be strong at first—2 to 3 grs. of sulphate of zinc to the ounce being sufficient. The patient should be directed to pass his water, and then wash out the passage with a little tepid water; after that the injection should be used, and kept in the urethra for two minutes. Many substances are used as injections, and it is difficult to say in a given case which will suit best. Sulphate of zinc grs. 2 to ʒj, or mixed with acetate of lead;

chloride of zinc gr.  $\frac{1}{4}$ -gr. 1 to ʒj; sulpho-carbolate of zinc grs. 2 to ʒj; permanganate of zinc gr.  $\frac{1}{8}$  to ʒj, are all useful. Alum, acetate of lead, hydrastis, and the extract of belladonna may also be used. Extract of belladonna should not be used with permanganates, as they form an explosive mixture. The mercuric chloride in solutions of various weakness,  $\frac{3\text{ss}}$  to  $\frac{2\text{ss}}$ , is used on theoretical grounds by some; but in the writers' experience they are most uncertain, and more often excite irritation than allay it in the urethra.

Whatever injection is chosen, it must be used judiciously, and care taken that it does not aggravate the condition it is intended to benefit. As a rule, the injection should be made at least three times a day by the patient; if the membranous-prostatic urethra be involved it may be necessary for the surgeon to apply astringents himself (*see GLEET*). Iodoform and eucalyptus bougies are highly thought of by some; they should be introduced at night. The writers have not seen any great benefit follow their use. If the injections increase the discharge, or heighten the soreness, they should be abandoned, and soothing internal remedies alone given. The chief internal specifics are copaiba, sandal oil, and cubebs; infusion of uva ursi, Canada balsam, tolu, turpentine, and benzoic acid. They are often beneficial, but are apt to cause dyspepsia; and, in the case of copaiba, sandal wood, and Canada balsam, congestion of the kidneys, especially in the later stages of the disease. These remedies should be given thrice daily for several weeks. In the later stages the preparations of iron in combination with quinine are often useful.

Chordee is frequently a very troublesome symptom, for, in addition to the pain it causes, the patient's rest is broken, and consequently his health weakened. Perhaps the most useful remedy is a suppository at night of opium or belladonna in combination with camphor (1 gr. and 3 grs.); bromides, chloral hydrate, cannabis indica, and lupulin are also useful; while the erection can be immediately stopped by the application of cold water. In some cases a hot bath just before going to bed, with the use of the suppository mentioned, will prevent chordee. When gonorrhœa becomes chronic, accompanied by a gleet discharge, further treatment may be necessary. *See GLEET*.

**COMPLICATIONS AND SEQUELÆ.**—Various complications may arise during the progress of gonorrhœal urethritis; many of these are referred to in special articles, to which the reader is referred for information.

1. *Balanitis* and *Balanoposthitis*.—*See* BALANITIS.

2. *Phimosis* and *Paraphimosis*.—*See* PENIS, Diseases of.

3. *Retention of urine* may occur in consequence of swelling of the mucous mem-

brane, and muscular spasm closing the passage at the membrano-prostatic portion; this is specially likely to occur if there is inflammation of the neck of the bladder. Under the influence of warm baths, saline aperients, rest, and morphine suppositories, the congestion is lessened, and the temporary retention is usually relieved. It causes great pain and anxiety when present. Gonorrhœa affecting a patient who is the subject of stricture may, by causing acute congestion of the narrowed portion of the urethra, temporarily occlude the passage and cause retention. The treatment is the same as given above; in both cases it may be necessary to pass a fine soft instrument in order to afford immediate relief. In the case of stricture further treatment will be necessary when the gonorrhœa is cured. See MICTURITION, Disorders of.

4. *Lymphangitis and Bubo*.—See BUBO; and LYMPHATIC SYSTEM, Diseases of.

5. *Hæmorrhage from the urethra*, if profuse and continuous, is probably due to rupture of the corpus spongiosum during erection. A few drops of blood may come away with the discharge or after micturition; this may be due to rupture of the congested vessels, or to the presence of a granular patch, and needs no special treatment. If hæmorrhage is continuous, ice will be serviceable in arresting it, and hamamelis may be injected. In some cases a catheter may be passed, and the penis compressed by a bandage. See HÆMATURIA.

6. *Peri-urethral abscess* may occur close to the glans penis, between the layers of the perineal fasciæ, or in the glands of Cowper. Peri-urethral abscess may cause acute symptoms, but is often chronic in its progress. The pus may escape into the urethra, externally through the skin, or both ways; in the latter case a urinary fistula is formed, which will, however, usually heal in time. When peri-urethral abscess is diagnosed, it should be freely opened; if the abscess has originated in a distended follicle, and has burst into the urethra, it may be treated successfully by laying it open, through the endoscopic tube, with a canaliculus knife.

7. *Inflammation of the neck of the bladder*, often erroneously spoken of as cystitis, is due to extension of the disease to the membrano-prostatic urethra. Cystitis itself is a rare complication. This complication does not usually come on until the second or third week; it causes much pain and spasm, and has a very depressing effect on the patient's general health.

Inflammation of the neck of the bladder causes great frequency of micturition, with pain during and scalding at the end of the act, caused by muscular spasm. A few drops of blood usually escape after the urine, with a little muco-pus. The usual gonorrhœal discharge ceases almost entirely during the

attack, but usually returns when the inflammation of the neck of the bladder subsides. Relapses are common.

Low diet, warm baths, rest, and anodynes will prove of great value in this complication.

When the bladder itself is affected, the disease usually attacks the base, and may spread to the kidneys. The inflammation may be acute or chronic, and may attack the mucous membrane only, or the whole thickness of the wall (parenchymatous cystitis). See BLADDER, Diseases of.

8. *Prostatitis and prostatic abscess*.—Prostatitis is a severe complication of gonorrhœa. It is due to the spread of the disease to the uvula vesicæ, and along the prostatic ducts to the body of the gland. It may be excited by injudicious treatment, wet and cold, or venereal excitement during gonorrhœa. The disease may run on to suppuration, or may become chronic.

In the acute form the gland is swollen and tender, causing painful and difficult micturition, and sometimes retention. There is a sense of weight, heat, and fulness in the perineum and above the pubes, with rectal irritation and pain on defecation.

Should suppuration occur, the local and constitutional symptoms deepen. Eventually the abscess bursts, commonly into the urethra, more rarely into the rectum or bladder or on to the perineum. The treatment is the same as that described for inflammation of the neck of the bladder, combined with the passage of a catheter if retention should occur, and the evacuation of pus as soon as it shall be diagnosed. Such abscesses are best opened through the perineum; should they burst into the urethra they will usually heal spontaneously; if not, a perineal opening must be made. See PROSTATE, Diseases of.

9. *Epididymitis and orchitis*.—Inflammation of the epididymis is the most frequent complication of gonorrhœa. The disease is usually unilateral, but in a small proportion (3·5 per cent.) of cases both sides are affected. The spread of gonorrhœa to the epididymis is much more commonly seen among hospital out-patients than among private patients, doubtless because the latter take more care of themselves. The epididymis is not affected until the urethritis has spread backwards to the membrano-prostatic portion—i.e. towards the end of the third week. See TESTES, Diseases of.

10. *Inflammation of the rectal mucous membrane* occasionally occurs, more especially in women. It is due to direct inoculation of the discharge. There is smarting and burning pain, much increased on defæcation. The mucous surface is bright red, and may be granular and ulcerated. A yellowish discharge escapes *per anum*. Thorough cleanliness, with the use of astringent lotions, will effect a cure.

11. *Gonorrhœal conjunctivitis* is a very grave complication, especially if not seen early, as it may destroy the whole eye. The patient should be warned that no discharge from the urethra must be allowed to get near the eye. See EYE, AND ITS APPENDAGES, Diseases of.

12. *Scleritis* and *iritis* occasionally occur in the course of gonorrhœa, but are very rare. See EYE, AND ITS APPENDAGES, Diseases of.

13. *Rheumatism* of the joints, fasciæ, and great nerves, &c. See RHEUMATISM, GONORRHOËAL.

14. *Stricture* may occur as a sequela. It usually follows granular urethritis, resulting from gonorrhœa. Some months or even years elapse before the stricture is established. See URETHRA, Diseases of; and GLEET.

15. *Warts* may occur on the glans penis or in the urethra. See GLEET; PENIS, Diseases of; VERUCCA.

(b) *Gonorrhœa in the Female—Vaginitis.*—MORBID ANATOMY.—In the female, gonorrhœa first attacks the lower part of the vagina, and may spread to the vulva and urethra, or upwards to the upper part of the vagina and the uterus; and, by continuous extension, to the Fallopian tubes, peritoneum, and ovaries in one direction, and to the bladder, ureters, and pelves of the kidneys in another direction, besides attacking the joints, fasciæ, and eyes with rheumatic inflammation as in the male subject. The disease may become chronic, and be accompanied by complications to be afterwards mentioned.

In the first stage the mucous membrane is congested, swollen, and tender; the parts are drier than natural, in consequence of a diminution of the natural secretions; but in a few hours there is a thin transparent mucous discharge, which quickly becomes sero-purulent, purulent, creamy, and copious. The discharge when fully established has often a greenish tinge, and is offensive and highly irritating. The affected mucous membrane is often studded with little eminences due to enlargement of the papillæ (vaginitis granulosa). In more severe cases patches of erosion may be seen, especially if the disease spreads to the os uteri. Owing to the irritating nature of the vaginal discharge the vulva becomes inflamed, swollen, and excoriated. The glands of Bartholin not infrequently become involved, and suppurate. The inflammation becomes chronic in from six to ten or twelve days, the pain, swelling, and congestion subsiding, and the discharge, now muco-purulent, remaining plentiful. In the chronic form, the upper part of the vagina, the *cul de sac*, and the neck of the uterus are the parts specially involved. They are of a brighter colour than the rest of the mucous membrane, and secrete the discharge. Not infrequently, when the inflammation has ceased in the vagina, pus can still be squeezed

from the meatus urimarius or some of the crypts opening round that orifice, if the finger be drawn forwards along the under-surface of the urethra.

SYMPTOMS.—In the acute stage there is heat, itching, and swelling of the parts, smarting on making water, aching pains in the back and loins, with great tenderness on examination. The bright red colour of the congested mucous membrane, together with the offensive greenish purulent discharge from the surface, is sufficient evidence of vaginitis, but is not sufficient to determine its specific origin. As the disease becomes more chronic the acute symptoms pass away, but there is still a plentiful discharge.

DIAGNOSIS.—The diagnosis of vaginitis and vulvitis is easy, but it is sometimes impossible to speak with certainty as to its gonorrhœal origin. The actual source of the discharge should be ascertained, since copious yellow irritating discharge may come from the cervix uteri, independently of any pre-existing vaginitis. If a positive opinion as to the gonorrhœal nature of the case is required, the gonococcus must be sought for in the discharge. If there is purulent urethritis, the contagious nature of the case is satisfactorily determined, as irritating uterine discharges do not affect the urethra.

PROGNOSIS.—If assiduously treated, so that extension to the vaginal *cul de sac* and cervix uteri is prevented, the disease has a duration of about three weeks. If neglected, it may become chronic and prove very intractable, lasting sometimes for many months; this is especially likely to be so if the disease has spread to the cervix uteri and endometrium, or to the urethra. Although many complications may arise during the progress of the disease, but few are commonly of immediate danger. The length of time that the discharge remains contagious is most uncertain. Probably any discharge, however scanty and serous it may have become, may cause disease if increased by accidental irritation.

TREATMENT.—In the acute stages of vaginitis the treatment consists in allaying irritation by rest in bed; warm baths; frequent injections of warm water, solution of boric acid, or weak astringents—such as weak solutions of acetate of lead, of alum, &c.; and the administration of saline aperients. If the pain be great, a little laudanum may be added to the warm water injections. The diet should be low, all sources of irritation should be removed, and the habits and health of the patient regulated. When the acute stage has subsided, astringent injections should be used somewhat stronger than those employed at first. The vulva should be protected from contact with the discharge by vaseline, and the labia should be separated by a piece of lint wetted with lead lotion.

In the chronic forms of the disease the

strength of the astringent injections must be increased. The most useful are the sulphate, chloride, and sulpho-carbolate of zinc, tannic acid, and liquor carbonis detergens. In the intervals between the injections, tampons saturated with the astringent lotions may be introduced into the vagina, and alum or tannin may be applied to the deeper parts of the canal by means of the speculum. The injections must be efficiently done, after washing away the discharge with warm water, so that the whole mucous surface of the vagina, especially that of the *cul de sac*, is thoroughly laved. Astringent vaginal suppositories are also useful. If in chronic cases there are patches of erosion, similar to those met with in cases of gleet in the male, they should be mopped with a 20 per cent. solution of silver nitrate.

Internal remedies are useless unless the urethra is affected, when they have a certain value.

**COMPLICATIONS.**—*Vulvitis* is one of the earliest and commonest complications of gonorrhœal vaginitis. The labia, nymphæ, and clitoris are red and swollen, and secrete a copious yellow fetid discharge. The nymphæ often project between the swollen labia, and the parts may be excoriated in consequence of destruction of the surface epithelium, and sometimes from ulceration of the mucous follicles. There is considerable pruritus. Usually, if the parts are kept clean and free from irritation, the inflammation subsides in a few days. Warm baths and sedative lotions are useful to allay the irritation, and the labia should be separated by a piece of lint kept moist with lead and opium lotion.

*Urethritis* is the most constant accompaniment of gonorrhœa; rarely so acute as to cause much irritation, it may produce severe suffering. It begins with itching and smarting at the meatus, which is red and swollen. A purulent or muco-purulent discharge oozes from the passage unless the patient has just micturated; even then a little discharge can be found in the mouths of the follicles which open close to the meatus. This discharge is very persistent, and is probably a source of contagion long after the disease is cured elsewhere. The treatment of urethritis consists of frequent warm baths, astringent injections, and copaiba internally. Obstinate chronic discharges may be arrested by caustic solutions, carefully applied.

*Abscess of the glands of Bartholin* is not uncommon in gonorrhœa. There is a painful swelling, which may attain the size and somewhat the shape of a bantam's egg, lying in the labium majus, in which fluctuation can be detected early. The abscess may not empty itself through the duct, but into the cellular tissue of the labium, where a large collection may form before it opens superficially. The treatment consists in free incision as soon as fluctuation is felt. Often a trouble-

some fistula is left, which, alternately closing and opening, causes fresh accumulation and fresh discharge of pus from time to time. This is best treated by free incision and drainage.

*Bubo* is rare in the female, but may occur when the urethra is involved.

*Acute inflammation of the cervix and os uteri* is a frequent consequence of gonorrhœa. The neck of the uterus is swollen, red, and often excoriated about the os, whence a copious discharge issues, at first clear and viscid, then purulent. This subsides to a thin muco-pus, and either soon ceases or more commonly passes into chronic catarrhal discharge from the glands of the cervix, which lasts an indefinite time and long retains its contagious quality. Chronic cervical and corporeal endometritis, associated with a thick viscid discharge in the former and a thinner discharge in the latter, may result from gonorrhœal infection. In the acute forms, complete rest, warm baths, with astringent applications and saline aperients, may arrest the disease. In the chronic stage the treatment is that of uterine catarrh. See **WOMB**, Diseases of.

Among the rarer and more dangerous complications of gonorrhœa in the female may be mentioned metritis, perimetritis, salpingitis, pyosalpinx, hydrosalpinx, ovaritis, and peritonitis. Pyæmic infection may occur through the vaginal veins.

Certain complications, more general in their nature, may also occur as in the male sex. Gonorrhœal rheumatism is less common in women than in men.

BERKELEY HILL. CHARLES STONHAM.

### GONORRHOËAL RHEUMATISM.

An affection of the joints associated with gonorrhœa. See **RHEUMATISM**, **GONORRHOËAL**.

**GOOSE-SKIN.**—A condition of the skin in which this structure is rough and wrinkled, like that of the plucked goose. It is of a transient character, being due to contraction of the muscular fibres of the skin, producing wrinkling of the integuments, and prominence of the hair-follicles—horripilation; and is observed as the result of the direct application of cold, or of a shock, and in the early stages of fevers.

**GOUT** (*gutta*, a drop).—**SYNON.**: Podagra, Chiragra, Gonagra (when the disease affects the foot, hand, or knee respectively); Fr. *Goutte*; Ger. *Gicht*. The name *gout* is supposed to have originated in the idea of the dropping of a morbid fluid into the joints, and is of very ancient date.

**DEFINITION.**—Gout is usually regarded as a general or constitutional disease, associated with the presence in the system of excess of uric acid, the complaint being commonly looked upon as a manifestation of the so-called *lithic* or *uric acid diathesis*, and as

due to *lithæmia*. It may be hereditary or acquired; and is characterised ordinarily by a peculiar inflammation of the joints—*articular* or *regular gout*, attended with the deposit of urates, consisting almost entirely of urate of sodium, in their structures, affecting usually and especially the smaller joints, and at first more particularly the metatarsophalangeal articulation of the great toe, but afterwards extending to other joints. Similar deposits of urate may occur in other tissues—*tophaceous gout*; and certain organs of the body are liable to become the seat of functional disorders, or of pathological changes, during the progress of the disease—*non-articular* or *irregular gout*; while it is also usually attended with general symptoms. Gout in the early part of its course is in the large majority of cases an *acute* affection, occurring in periodic attacks or 'fits'; but subsequently it tends to become more or less *chronic* and permanent, though even then generally presenting exacerbations from time to time. The gouty diathesis may, however, be present without giving rise to any joint-affection or other evident organic mischief.

**ÆTIOLOGY.**—There are certain definite points which require consideration under this heading.

1. It will be expedient at the outset to discuss the circumstances under which the gouty diathesis is developed, and to point out the more obvious *causes* with which this condition is associated. Ætiologically, cases of gout may be practically arranged into three main groups, according as the disease arises from—(a) *Hereditary transmission*. (b) Certain errors in regard to *food and drink*; often associated with *deficient exercise*. (c) Impregnation of the system with *lead*. In not a few instances, however, it must be remembered that these causes are more or less combined.

(a) *Hereditary transmission*. Gout is one of the most striking examples of an hereditary disease, and, once established, it may be transmitted through several generations, even when every endeavour is made to eradicate it; but as the malady is, as a rule, more or less intensified by pernicious habits, it becomes in most cases a permanent legacy. Sir Alfred Garrod found that in more than half his cases hereditary taint could be distinctly traced; and the proportion is much greater among the upper classes. It sometimes happens that when gout becomes developed *de novo* in an individual, children born previously are free from the complaint, while those born subsequently are affected. Gout appears to be more readily transmitted by the female than the male line. According to Hutchinson, the diathesis becomes stronger in the parent with advancing years, and consequently the disease shows itself with greater frequency, and in more marked

form, in the younger than in the older members of a gouty family. Hereditary influence may be so powerful that the complaint arises without any other obvious cause; but most commonly it is aided by more or less indulgence in certain injurious habits to be presently mentioned. Gout sometimes exemplifies the so-called 'law of atavism,' which is usually due to the fact that in the generation free from the complaint every precaution is taken to avoid causes which tend to originate a gouty paroxysm. It has been affirmed, however, that this 'missing a generation' only applies to the transmission from a grandfather through his daughter to the grandson. The hereditary nature of gout is shown not infrequently in the age at which the disease reveals itself. Should the predisposition be powerful, the complaint may appear in very early life; and the younger the subject who is attacked, the more likely is there to be an hereditary taint. Hutchinson is of opinion that many obscure joint-affections in young persons are due to hereditary gout.

(b) *Errors relating to food, drink, and exercise*. In a considerable number of cases gout is originated *de novo*, in consequence of certain errors affecting the diet and habits; or an inherited tendency to the disease is thus considerably aggravated. In general terms these errors may be summed up as excessive eating, especially of particular articles of food; undue indulgence in alcoholic drinks; and indolent habits, with deficient exercise. They are frequently associated in the production of gout in individual instances.

The gouty diathesis is chiefly promoted by foods which are rich in nitrogen, and especially meat. Beef is believed by many to be particularly baneful. Undue consumption of sugar and starchy foods has also been regarded as aiding materially in the development of gout. Many articles of diet, either from their inherent nature or from the manner in which they are cooked, may help in the production of the complaint, by giving rise to digestive disorders.

The relation of intemperance in the use of alcoholic drinks to the gouty diathesis is highly important, and is abundantly proved by everyday experience, notwithstanding the attempts made by certain self-constituted 'authorities' on gout to cast a doubt upon this fact. The more potent wines have the greatest influence, and port wine has proverbially been regarded as the most injurious of all. Champagne, burgundy, madeira, sherry, and marsala are also undoubtedly capable of developing or aggravating gout. Hock, sauterne, moselle, and light claret seem to be least injurious; but even these, if indulged in to excess, may in course of time set up, or, at any rate, intensify the gouty diathesis. Strong malt liquors stand

next to wines as originators of gout. Spirits are comparatively feeble in their power of producing the disease, and this is the explanation usually given of its infrequency in those countries or districts where this class of drinks are chiefly used, such as Scotland and Newcastle-on-Tyne, where whisky is the common beverage. Rum is said to form an exception to this statement. Cider and perry may unquestionably set up gout, if taken to excess, especially when sweet and not properly fermented. Excessive indulgence in a mixture of alcoholic drinks is probably more deleterious than if one is adhered to. The quality of wines has much to do with their tendency to induce gout. Factitious wines and others of inferior quality, as well as those which are very sweet, or which contain much tannin, are most liable to produce this complaint. Drinks which cause a marked diuretic action are less injurious than those which have but little action of this kind.

Deficient exercise undoubtedly promotes the development of gout in many cases. Persons who follow sedentary occupations, or who live indolent and lazy lives, are most liable to the disease; and not a few become gouty because improvement in their circumstances enables them to 'keep a carriage,' and they are thus deprived of the exercise which they were previously accustomed to take.

(c) Another group of cases of gout are those which occur in connexion with *impregnation of the system with lead*. Sir Alfred Garrod found among his hospital patients that 33 per cent. of those suffering from gout had been subjected to the influence of lead in their various occupations; and the association is now generally acknowledged. This metal does not appear, however, to originate the gouty diathesis, unless aided by more or less indulgence in alcoholic drinks, though the amount of the latter consumed is usually far less than would alone account for the condition. Gouty persons are remarkably susceptible to the influence of lead; and when this metal is given to such subjects for medicinal purposes, it is very liable to bring on a severe attack of acute gout.

It is important to note here that, according to Dr. Thomas Oliver, of Newcastle-on-Tyne (*Goulstonian Lectures*, 1891), this intimate relation between gout and saturnine poisoning is not seen in the north. 'Workmen from the south develop it in the north of England. The natives of the north, though equally exposed, seldom become gouty even when the kidneys are affected.' This authority does not regard the difference in the drinking habits of the people as the explanation of the difference, but thinks it is probably the result of external conditions, though he cannot explain it.

2. *Predisposing causes*.—Beginning with age, distinct gouty attacks in a large majority

of cases make their first appearance in persons between thirty and thirty-five or forty years old. Those which occur under thirty are, with few exceptions, more or less hereditary. Well-marked gout is exceedingly rare under twenty, but it may occur in younger subjects, probably even in children. Dr. Lorimer, of Buxton, found that saturnine gout appears at an earlier age than non-saturnine. The complaint is usually declared before fifty, and becomes progressively less frequent in its manifestation for the first time after this period of life. It is quite exceptional for gout to commence after sixty-five, but it may begin even in extreme old age. With regard to *sex*, males are far more commonly the subjects of well-marked gout than females. This is mainly accounted for by the difference in the habits of the two sexes. It has also been partly attributed to the occurrence of menstruation in females, which is supposed to act as a safeguard, and in these subjects gout generally appears after the cessation of this function. When strongly hereditary, gout may, however, develop even in young females, of which the writer has seen some well-marked examples.

*Bodily conformation and temperament* have been credited with a predisposing influence in relation to gout, persons of a sanguine temperament, and of corpulent, plethoric habit of body, being supposed to be most subject to this disease, and to have it in its most acute form. These conditions are often produced by the very habits which originate gout, and certainly persons who are gouty by inheritance often do not present any of these characteristics, while those exhibiting marked contrasts in appearance and temperament seem to be equally the subjects of the complaint. It is not uncommon in individuals of a nervous temperament, thin and wiry in frame, and they are said to be more subject to the irregular and asthenic forms of the disease. *Social position and occupation* materially influence the occurrence of gout. Formerly the complaint was met with almost entirely among the higher classes, and it was looked upon as an aristocratic disease. Now, however, it is common enough among the middle classes, chiefly those who are in affluent circumstances; while it is very prevalent amongst those following certain occupations, such as butlers, coachmen, butchers, publicans and barmen, coal-heavers, porters, hair-cutters, and painters or others who have to do with lead. Conditions which involve excessive mental labour and fatigue, prolonged worry or anxiety, and other effects which exhaust and depress the nervous system, seem to predispose to gout. Sir Dyce Duckworth is of opinion that *sexual indulgence* in early life, by its generally enervating influence, is potent, not only in causing early and severe attacks of gout, but also in the premature induction of gouty cachexia. As regards *climate*, those

climates which are cold or temperate, and especially at the same time damp and changeable, present by far the greatest number of cases of gout, and in most tropical countries this complaint is unknown. In the south of Europe it is far less frequent than here. Gout is much more prevalent in England than in Scotland or Ireland; though it is uncommon in the north of England.

3. *Causes of a gouty fit.*—An acute paroxysm of gout may come on without any evident exciting cause whatever, especially if the disease is strongly hereditary, or has been long established. Under these circumstances outbreaks of the complaint seem to become habitual at certain seasons, or they arise from very slight causes, which are less and less obvious as the case progresses. Often, however, some distinct exciting cause can be made out, affecting the digestive organs, the vascular or nervous systems, or the functions of the skin or kidneys, or disturbing the system in other ways. The most important are eating or drinking too much, either on some particular occasion, or habitually for a longer or shorter period, until at last a fit of gout terminates the indulgence; indigestible articles of food; neglect of the act of defecation, or prolonged constipation; undue physical work or exertion; exposure to cold or wet, or sudden suppression of perspiration; excessive mental work, worry, or anxiety; emotional causes, sudden, powerful, or depressing, such as joy, a fit of rage, fright, or deep grief; sexual indulgence or excess; hæmorrhage, acute illness, or other like debilitating causes; and injury. The indiscriminate use of certain medicines, such as preparations of lead or iron, may excite a gouty paroxysm. The implication of a particular joint may be due to a strain or injury, which may be very slight, such as the pressure of a tight boot, or the toe being trodden upon. Injury to the knee has caused that articulation to be first affected. As *predisposing causes* of acute gouty attacks, climate and season are highly important. Undoubtedly gouty paroxysms may often be averted by residence in a warm climate, either permanently or during the colder seasons of the year. Early attacks are most frequent in late winter or spring, and the first paroxysm occurs especially towards the end of January or beginning of February. Afterwards a fit of gout takes place also in the autumn; and subsequently the intervals become shorter and more irregular. One seizure predisposes to another, and the peculiar tendency to recurrence in gout is increased with each succeeding paroxysm. The occupation of an individual may predispose to the occurrence of gout in particular joints; thus butlers have it in the feet, coachmen and washerwomen in the hands.

**PATHOLOGY.**—The pathology of gout is still in a very unsettled state. With some authori-

ties it is a comparatively simple matter, while others regard the complaint as a highly complex condition, having wide relationships in diverse directions within the system. It will be impossible within the limits of this article to do more than bring out the main facts bearing upon the subject, and to indicate the chief theories founded thereon.

1. Gout is by the large majority of writers referred to the general or constitutional group of diseases, and it is customary to speak of the *gouty diathesis* or *dyscrasia*, of which the local changes are but a manifestation. Further, it is believed by most authorities that a person may be truly gouty, and yet be free from any of the special lesions usually associated with the condition. According to Hutchinson, Charcot, Duckworth, and others, there exists a 'basic arthritic diathesis,' upon which may be built up, under the influence of special causes, a tendency to gout, rheumatism, or their various modifications and combinations.

At the present day it is almost universally admitted that gout has an intimate relation with the presence of *lithic* or *uric acid* in the system, which is generally regarded as the morbid agent or 'peccant matter.' As Duckworth expresses it—'No uric acid, no gout.' To Sir Alfred Garrod we are indebted for having clearly and positively demonstrated this relationship. In its pronounced form gout is characterised by the deposit of urate of sodium in connexion with the joints and other structures, which distinguishes it from all other complaints. Dr. Haig has recently revived the view that there is no real distinction between the rheumatic, gouty, and rheumatoid forms of arthritis, but that they are all due to uric acid. This question need not, however, be discussed here.

Assuming the essential relationship between gout and lithic acid to be established, beyond this point there is much diversity of opinion. Many adopt a simple *humoral* theory, attributing the disorder to excess of this acid in the blood and tissues. Some, however, do not so much regard the quantity of acid, as the conditions under which it exists, and its insolubility. Gout is often spoken of as a mere manifestation of the *lithic* or *uric acid diathesis*, and the condition of the blood containing excess of the acid was termed *lithæmia* by the late Dr. Murchison. Sir William Roberts<sup>1</sup> has proposed the term *uratosis* as distinguished from lithæmia, to designate the 'precipitation of the crystal urates in the tissues or fluids of the body.' He draws attention to the fact that 'lithæmia is only an exaggeration of a normal condition, for traces of uric acid always exist in healthy blood; but uratosis, even in the minutest degree, is pathological.' He is of opinion that there are different kinds of uratosis, and would

<sup>1</sup> *Med. Soc. Trans.* vol. xiv. p. 84.

thus speak of *gouty uratosis* and *saturnine uratosis*, believing that the gouty diathesis and plumbism are radically distinct, differing in all respects except in their tendency to uratic deposits. He further considers it quite possible that precipitation of the crystalline urates may occur not only in the form of coarse masses, but also in the form of minute stars or detached crystals, which can only be detected with the microscope; and that this 'microscopic uratosis,' either in the substance of organs or in the blood itself, may account for the visceral neuroses, thrombosis and embolism, and the occasional sudden death, which are encountered in gout, and may explain some very obscure incidents in its history.

Uric acid exists in the blood and in the fluid that diffuses therefrom mainly as a salt of sodium, but whether the combination be a quadriurate or a biurate, or a mixture of the two, or sometimes one and sometimes the other, is not known. The quadriurates are highly soluble in blood-serum, and cannot be thrown down therein in the crystalline state, while the biurates are very sparingly soluble in that medium, and are readily thrown down in it in a crystalline form (W. Roberts). The acid is being constantly formed in the system in connexion with the processes of nutrition and metabolism, but under ordinary circumstances it is either destroyed or is eliminated by the kidneys, so that it is kept within normal limits. According to Garrod, the final stage is the production of urate of ammonium in the kidneys.

We may now proceed to consider the main views held to explain more immediately the lithæmic condition, or the accumulation of uric acid in the tissues. They may be summarised as follows:—(a) *Excessive formation*. It certainly appears reasonable to suppose that certain of the recognised causes of gout must lead to an absolute increase in the amount of uric acid formed in the system. Ebstein is of opinion that in the gouty this acid forms in perverse localities, as in muscles and the marrow of bones. (b) *Undue retention*. Imperfect excretion or elimination of uric acid, leading to its retention in the body, can never be overlooked as an important factor in relation to the gouty condition. According to Garrod, such retention is due to defective action of the kidneys, either temporary or permanent, and when these organs are in a state of advanced organic disease, their excretory functions must obviously be interfered with considerably. Sir William Roberts thinks it probable that the defective power of the kidneys to eliminate uric acid is due to diminished alkalinescence of the blood, which renders it less soluble. He has found that if sodic quadriurate lingers too long in the blood it becomes converted into a biurate, and when the accumulation of this salt has reached a certain point of saturation, it is sud-

denly precipitated as a crystalline deposit in the joints or elsewhere. Dr. Ralfe is of opinion that the first step in the production of gout is diminished alkalinity of the blood, by reason of the accumulation in it of acid and acid-salts; and that retention in the system is due to a fault in the tissues, leading to incomplete elimination. Haig, as the outcome of his extensive researches relating to uric acid, strongly supports the view that excess of this acid in the blood and system is generally due to retention, and that this is the result of deficient alkalinity of the blood, which leads to gradual accumulation of the acid in certain parts; he would thus explain all the phenomena of the gouty state. According to Garrod, with whom Haig agrees, it appears that uric acid is attracted in different degrees by different structures, and tends to be held back by, and accumulate in the liver and spleen, and in fibrous tissues, especially those of the joints, probably because these structures are acid, or less alkaline than the rest of the tissues and fluids of the body. (c) *Imperfect reduction*. According to this view, it is supposed that the normal process, by which uric acid is changed in the blood and certain organs into other substances more soluble and less noxious, fails, or is imperfectly carried on, and thus it accumulates in the system. It used to be believed that uric acid was converted into urea, but more recent investigations are opposed to this idea. (d) *Special views*. It will be convenient under this heading to mention certain individual opinions with regard to the pathological relations of uric acid and gout. The late Dr. Murchison regarded gout as a mere result or variety of lithæmia, and attributed this condition to imperfect digestion, and more especially to 'functional derangement of the liver.' He brought forward facts to show that the healthy liver plays an important, and perhaps the principal, part in carrying on those chemical changes by which albuminous substances are disintegrated in the body, which normally result in the production of urea. If this process is persistently deranged, lithic acid, a less oxidised body, is formed. Professor Latham also advocates the hepatic origin of gout, but considers that 'imperfect metabolism of glycocine is the primary and essential defect.' Dr. Ord regards gout as arising from a tendency to a special form of degeneration, or want of tissue organisation in some of the fibroid tissues, with excessive formation of urate of sodium, which is discharged into the blood, and deposited in parts least freely supplied with vascular and lymphatic structures. According to Ebstein, the primary factor in gout is nutritive tissue-disturbance, a necrotic change taking place in the affected structures, which is followed by deposit of urates.

Such are the principal facts and theories bearing upon the relations of uric acid to

gout. It must be mentioned that some eminent writers have disputed this close relationship, but it would not serve any practical purpose in this article to discuss the various hypotheses which have been advanced in the endeavour to explain the nature and origin of gout.

One important point remains to be noticed, and that is the relation of the *nervous system* to gout. Other writers on the subject have maintained that such a connexion exists, but Sir Dyce Duckworth<sup>1</sup> has developed this idea more fully, and has brought forward much evidence in favour of the view that gout is a *neuro-humoral* disease. He recognises a *gouty neurosis*, which may be primary or central, or secondary or induced; and maintains that there is a functional disorder of a definite tract of the nervous system, which he is inclined to localise in some part of the medulla oblongata, but does not insist on this point. He further believes 'that this diathetic neurosis imposes its type upon the affected individual in definite nutritional modes, affecting the assimilating and excreting powers, exhibiting marked peculiarities in nervous impressibility, and determining, in a more or less degree, a physiognomy of the gouty.' This authority recognises the dependence of many gouty phenomena upon 'perverted relations of uric acid and sodium salts in the economy' which result from the morbid peculiarities just mentioned. He considers that in primary or inherited gout, the toxæmia is dependent on the gouty neurosis, of which it is a secondary manifestation; while in secondary or acquired gout, if, together with toxæmia induced by certain habits, distinctly depressing and exhausting agencies, affecting the nervous system, come into operation, the special neurotic manifestations of the gouty diathesis will occur, and be impressed more or less deeply upon the individual and his offspring.

Space will not permit the discussion of the modes in which the several causes which produce the gouty condition or its local manifestations individually act. In general terms, it may be stated that they produce their effects chiefly by increasing the amount of uric acid formed, or interfering with its elimination; disordering digestion and the hepatic functions; increasing the acidity of the blood and tissues; affecting tissue-change and metabolism; influencing the circulation; or producing some change in the nervous system.

2. We have thus far been concerned mainly with the pathology of the gouty diathesis, and it remains to offer a few remarks with regard to its *local manifestations*, and more especially to the occurrence of acute attacks of gout, and the formation of uratic deposits. According to the lithæmic theory, any cause which tends suddenly or rapidly to increase

the amount of uric acid in the blood in a gouty individual may lead to acute articular gout, or some other obvious manifestation of the disease. Dr. Haig would attribute the joint-affection to conditions increasing the acidity of the blood, and thus driving the uric acid into the joints, the structures of which are less alkaline than other tissues. Others call in the aid of the nervous system to explain the phenomena. As regards the gouty inflammation, this is often looked upon as the result of the irritant action of the urate, and Garrod affirms that even after the first attack a deposit of this substance is found. Another idea is that the acute paroxysm is the consequence of an attempt on the part of the articular structures to destroy and eliminate the urates accumulated in connexion with them. The tendency to impaction of joints and allied structures seems unquestionably to depend to a great extent upon their slight vascularity, and this especially applies to the smaller joints, which are chiefly affected in gout, and which are also distant from the centre of the circulation, so that the blood passes through them in a feeble and languid stream. They are, further, much exposed to the direct influence of cold, and are very liable to slight injury or strain, whereby their nutrition is affected, which predisposes them to the gouty change. The metatarso-phalangeal joint of the great toe is subjected to much pressure, and also to sudden shocks (Garrod).

It is an undoubted and well-known fact that marked gouty changes, with uratic deposits in the joints and elsewhere, may develop without any definite acute attacks; and as gout advances in a particular case, the deposit often becomes very abundant, while the paroxysms progressively diminish in intensity. Some writers think that true gouty lesions may occur, and yet without any formation of urates.

The opinion is held by many authorities that uratic deposits only take place in structures the nutrition of which is impaired, and which have already begun to degenerate. As already stated, Ebstein maintains that a nutritive disturbance of the affected tissue is the primary change, leading ultimately to its necrosis, and that uratic crystallisation is secondary, not occurring before complete death of the damaged texture. He looks upon sodium urate as a directly poisonous irritant wherever deposited, the injurious effects varying according to the quantity and concentration of the deposit, and the vulnerability of the special tissue involved, firm textures resisting better than those of looser character. He regards the incrustation as analogous to calcification; and believes that lime-salts may be subsequently deposited in gouty tissues.

ANATOMICAL CHARACTERS.—In its most typical manifestations gout is characterised

<sup>1</sup> *A Treatise on Gout*, 1889.

anatomically by the occurrence of a peculiar form of inflammation affecting certain joints, attended with the deposit of urate in connexion with their structures. Taking an individual articulation, this is at first usually the seat of an acute inflammatory process, indicated by the usual signs of increased vascularity and redness, tumefaction, and serous effusion into the interior of the joint, as well as into the surrounding tissues. The results of *post-mortem* examinations show that even in the very earliest period a deposit of urate takes place; and as the attacks become repeated again and again, the signs of inflammation become less and less prominent, while the deposit increases, until at last it may form considerable masses, and infiltrate extensively all the structures entering into the formation of the articulation. The joint then becomes permanently enlarged and distorted, while the ligaments are thickened and more or less stiff or even quite rigid, until ultimately complete ankylosis may be produced. The deposit seems to commence in the substance of the cartilage covering the ends of the bones, starting near its superficial or free surface, and gradually extending more deeply, though for a time a thin layer of cartilage lies between it and the cavity of the joint. This deposit at first forms a whitish opacity, but as it becomes more abundant it encrusts the cartilages, and also the inner surface of the ligaments, and the surfaces of fibro-cartilages where these exist. More or less extensive spots or patches become in time distinctly visible, and even the entire surfaces of the bones forming a joint may be covered with a chalky-looking substance. 'The appearance of articular cartilage in which simple deposit of urates has taken place exactly resembles that which would result from smearing or splashing the surface with fresh white paint' (Duckworth). The synovial membrane may also present white points, but the synovial fringes at their margins seem to escape, on account of their vascularity. In the larger articulations the synovial fluid may be thickened, and may even contain separate crystals or tufts of the urate. Subsequently the ligaments and adjoining structures are infiltrated, and it is to this cause that the stiffness or rigidity of gouty joints is mainly due. Distinct masses of deposit may, however, form, which also interfere with movement. They are known as *tophi* or *chalk-stones*. In course of time the tissues covering a gouty joint may be destroyed, including the skin, the chalky-looking substance being thus exposed, and unhealthy suppuration and ulceration set up, or even gangrene. Very rarely suppuration takes place in a gouty joint itself, or hæmorrhage may occur.

The opaque white substance characteristic of articular gout is found on microscopic examination to consist of fine crystals, in the

form of needles or prisms. They are chiefly arranged in minute clusters, radiating from a centre; and in the cartilages they form a more or less compact network. Chemical examination shows that they are usually composed almost entirely of urate of sodium.

The metatarso-phalangeal articulation of the great toe is the one primarily attacked in the large majority of cases of gout; and this joint, on one or both sides, may alone present any change. As usually seen, however, the disease has progressively involved many joints. In the feet it may implicate all the articulations, but it is a curious fact that the tarso-metatarsal and the phalangeal joints of the great toe generally escape, or are but little affected. Similarly, all the joints of the hands and fingers are often involved, but the metacarpophalangeal joint of the forefinger is one of the most commonly attacked. The gouty change not uncommonly extends to the larger joints, more especially those of the leg, but the shoulder and hip-joints are very rarely implicated. In exceptional cases other articulations are found involved, such as the temporo-maxillary, those of the spinal column, of the pelvis, or even of the larynx in rare instances.

Coming now to other structures, deposits of urate may occur in various parts of the body, sometimes abundant and widely spread, in connexion with bursæ, tendons and aponeuroses, sheaths or the substance of muscles, the sclerotic coat of the eye, the cartilages of the external ear, eyelids, nose, or larynx, under the skin, or rarely in the cerebral and spinal meninges. Urates have also been found in concretions on the aortic and mitral valves, and in plates from the arch of the aorta. More or less serous effusion may be present in bursæ which are the seat of uratic deposits. The exact composition of these tophi varies, and they may contain calcium urate, oxalate, or phosphate, as well as sodium chloride and animal matter, in addition to sodium urate.

As regards bone, the periosteum is often affected, and some writers have described a deposit of urate in bone itself; but Garrod has not found evidence of its having originated in this tissue. He considers that the periosteal formations sometimes acquire sufficient size to press on the osseous tissue, and to cause its absorption.

The condition of the *kidneys* associated with gout is of great importance, and these organs probably begin to be affected at a very early period in the history of a case, for they may be found distinctly diseased when there have been little or no external manifestations of the complaint. In the first instance a deposit of sodium urate takes place, probably within the renal tubuli, which afterwards involves their walls and penetrates to the intertubular tissue. This is seen in the form of white streaks in the course of the

tubuli, and of white points at the extremities of the papillæ. The deposit goes on increasing, and a chronic inflammatory process is set up, ending ultimately in the production of a variety of 'granular contracted kidney,' known as the 'gouty kidney' (see BRIGHT'S DISEASE). Other morbid states in connexion with the urinary organs observed in some cases of gout are the presence of calculi consisting of uric acid, urates, or oxalate of calcium; chronic cystitis; or urethritis.

It must again be noted here that many writers recognise degeneration of the involved structures as an essential part of the gouty change, and appearances characteristic of such degeneration have been described in different tissues; thus the cartilages are said to be generally fibrillated and eroded before the deposit of urates takes place.

In the course of gout other morbid changes often arise, affecting different structures and organs, and either occurring as acute or even fatal events, or being of a chronic nature. Only the more prominent of these can be mentioned here, and they mainly include changes in the pharyngeal structures; congestion, catarrh, or more serious inflammation of some part of the alimentary canal; congestion or catarrh of the air-passages, chronic bronchitis, emphysema, and, some think, pneumonia; fatty disease of the liver, and perhaps cirrhosis; meningitis, neuritis, cerebral hæmorrhage; cardiac changes, including chronic valvulitis and degeneration of the valves, and hypertrophy, followed by degeneration of the cardiac walls; atheromatous changes in the vessels, hypertrophy of the muscular coat of the small arteries, or arterio-capillary fibrosis; various diseases of the skin, such as erythema, urticaria, eczema, psoriasis, as well as changes in the nails; iritis, hæmorrhagic retinitis, and other conditions of the eyes; and affections of the ear. How far some of these conditions can be directly attributed to the gouty diathesis, or are merely the result of the same causes which have induced this diathesis, may be fairly disputed. It is worthy of remark that acute inflammation in connexion with the heart is not met with in cases of gout, and this has been attributed to the great vascularity of the endocardium and other cardiac tissues, for which consequently the urate has no affinity, or it is destroyed in its passage through them.

The condition of the blood in gout may be here noticed. In early cases the chief deviation from the healthy state presented by this fluid is that during the acute paroxysms the serum contains a distinct excess of uric acid, in the form of a salt of sodium; and this can be obtained in appreciable quantity, even in a crystalline form. In the intervals the blood is quite normal. When the gouty condition becomes chronic, the excess of uric acid is constant; and oxalic acid can also be fre-

quently detected. In course of time the serum becomes lowered in its specific gravity, its albumen is deficient, and its reaction is less alkaline, in extreme cases becoming almost neutral, owing to the presence of excess of acids. The red corpuscles often diminish in number, and the blood deteriorates in quality as a whole in many cases of chronic gout.

Uric acid has been obtained from the fluid contained in the blebs produced by blisters; from inflammatory serous effusions; from dropsical accumulations, such as ascites; and from the subarachnoid fluid.

CLINICAL HISTORY AND SYMPTOMS.—The clinical history of gout is a very varied one, and the symptoms observed in different cases which are regarded as of a gouty nature are exceedingly numerous and diverse. Whether they are always directly due to uric acid in the system is, to say the least, a matter of considerable doubt. The habits which generate gout often give rise to symptoms, and even to definite morbid changes, which cannot justly be looked upon as part of this complaint. Again, the custom which some practitioners adopt, of looking upon every acute illness, particularly inflammation of organs, occurring in gouty subjects, as being due to the lithæmic condition, and of a special nature, and applying the term 'gouty' to all such complaints, is certainly going too far, though it may be acknowledged that gout does often modify their clinical history. It is not easy within a limited space to give even a sketch of the various clinical phases of gout, but before considering the symptoms in detail it may be well to indicate their general nature. 1. In its typical form gout is attended with acute symptoms referable to certain joints, and these tend to recur at intervals, constituting 'fits of gout,' the intervening periods becoming shorter and shorter as the case progresses—*acute articular or regular gout*. 2. These attacks culminate in obvious chronic changes in the affected joints—*chronic articular gout*—but even then more or less acute paroxysms are liable to arise from time to time. 3. In connexion with the acute attacks, and sometimes preceding them, *general or constitutional* symptoms are usually observed; and symptoms belonging to this class become permanent in most cases of chronic gout. 4. When gout affects other organs and structures besides the joints, corresponding symptoms are developed, according to the part implicated. In general terms these are variously grouped as cases of *non-articular, irregular, misplaced, latent, masked, or anomalous gout*; and when such symptoms are acute in their character, affecting some internal organ, and coming on during the course of an attack of acute articular gout, the joint-symptoms at the same time subsiding, this constitutes what is termed *retrocedent gout*. The

symptoms which may occur in these cases are conveniently subdivided as follows:— (a) Those indicating more or less functional disorder of certain organs, varying much in intensity, and either being constantly present, or only coming on at intervals. (b) Those due to acute inflammatory affections of organs. (c) Those resulting from the chronic structural changes in tissues and organs induced by gout, and from the deposit of urates in different parts. Having given this outline, we may now discuss the symptoms of gout in some detail, but it will be convenient in doing so to adopt a rather different arrangement from that just sketched.

**1. Premonitory Symptoms.**—There are certain symptoms, of a somewhat indefinite character, and not of any marked intensity, which are often met with in gouty subjects, or even in persons who have never actually suffered from declared gout, but which seem to be distinctly associated with the complaint. These may be regarded as *premonitory symptoms*, for they frequently give warning that the gouty condition is in process of development, and, if duly recognised, enable the patient so to regulate his mode of living as to ward off the disease. Indeed, it will be found on careful inquiry that gouty phenomena, which may be very marked, are commonly noticed from time to time before the first actual fit of gout occurs, and there may even be suspicious twinges or uncomfortable sensations about the toes or fingers now and then. In a large number of cases, however, no definite premonitory symptoms immediately precede the first gouty paroxysm; but in connexion with subsequent paroxysms prodromata are usually marked, so that confirmed gouty patients can predict when an attack is imminent. As to the nature of these symptoms, they vary in different persons, and this may depend upon individual predisposition. The most obvious are digestive and hepatic disorders, attended with marked flatulence and eructations, heartburn, acidity, and constipation or diarrhoea, with unhealthy stools, the tongue being often large, flabby, and much furred; palpitation or uncomfortable sensations about the heart; catarrh of the throat and respiratory passages, violent fits of sneezing, or asthmatic attacks; derangements of the nervous and muscular systems, indicated by a liability to headache, giddiness, attacks of migraine, noises in the ears, disorders of vision, marked irritability of temper and fretfulness or lowness of spirits, languor, impairment of mental vigour and intellectual hebetude, heaviness or drowsiness, sleep, however, being restless, disturbed, and attended with unpleasant dreams, peculiar grinding of the teeth, sometimes associated with somnambulism, numbness or tingling in the limbs, especially in the fingers or toes, neuralgia or neuritis in various parts, twitch-

ings, startings in the limbs, muscular cramps, especially in the calves of the legs, or possibly even convulsions, lumbago and other forms of muscular rheumatism; profuse perspirations; certain skin-affections; and changes in the urine. This excretion usually tends to become high-coloured and deficient, and to deposit lithates abundantly, or even lithic acid crystals, though at the same time the quantity of this acid eliminated within the twenty-four hours is below the normal. In advanced cases of gout, however, the urine presents very different characters from those just stated, as will be hereafter pointed out, and when habitually depositing urates it may become pale, watery, and clear immediately before an acute attack supervenes. Some patients are warned of the approach of a gouty fit by feeling unusually well, both physically and mentally.

**2. Acute Articular Gout.**—The first acute fit of gout comes on as a rule during the night, usually between 2 and 5 A.M. The patient is disturbed out of his sleep by uneasiness or pain, generally referred to the ball of the great toe on one side, and the joint is found to be inflamed, the inflammation increasing in intensity, until it usually becomes extremely severe. In some instances the corresponding joints on both sides are attacked simultaneously, in rapid succession, or alternately. Although, however, in the majority of cases gout first attacks the metatarso-phalangeal articulation of the great toe, it must not be forgotten that it may start in any of the smaller joints of the foot or hand, or even in the middle-sized joints, especially the knee or ankle. It has been affirmed that, next to the great toe, the metacarpo-phalangeal joint of the index-finger is most liable to be first attacked. Exceptional cases have come under the writer's notice, which there was every reason to believe were of a gouty nature, and in which the disease implicated several joints at a very early period of its course, the feet, however, being free.

Proceeding now to notice the clinical characters of the joint-affection, severity of the pain is certainly a striking feature in the majority of cases of acute gout, especially in early attacks. When the foot is affected, any attempt to stand causes much suffering from the first, and the pain speedily increases, until it becomes very intense, or almost unbearable. It is described in different cases as burning, throbbing, aching, tearing, plunging, boring, piercing, &c. The pain prevents sleep during the night, but towards morning it tends to diminish, and during the day there is usually comparative ease, an exacerbation again taking place as evening approaches, which goes on increasing towards night. Tenderness is very marked, and is often so exquisite that the patient dreads to be touched, and cannot bear the least move-

ment or jarring of the affected part, or sometimes even the weight of the bedclothes, or the slightest shaking of the room. The objective signs of inflammation in connexion with a gouty joint also soon become very prominent as a rule. These are marked redness, which may be very deep and sometimes tends to lividity, while the veins are often enlarged and turgid; considerable local heat, as revealed to the touch and the clinical thermometer; and much swelling, the skin covering the part assuming a tense and shining appearance. When several joints in the foot or hand are affected, diffused redness and swelling are noticed. The tumefaction is due to effusion not only into the interior of the articulation, but also into the surrounding tissues, subcutaneous œdema being a marked feature in connexion with gouty inflammation. This can be better appreciated when the acute symptoms subside, so that pressure can be borne, which shows the pitting characteristic of œdema, and this may hold on for some time. However intense the objective signs of inflammation may be, it is an extremely rare event for acute gout to end in suppuration. As they subside, marked desquamation of the skin usually takes place. As the swelling increases, the subjective sensations generally diminish in severity; but during the progress towards recovery, intense itching is apt to supervene.

An acute attack of gout is almost always attended with more or less *general* symptoms; but it is an important fact that their severity depends upon the extent and intensity of the local manifestations of the disease, and upon the accompanying symptoms. Chills or even actual rigors may be felt at the outset, followed by febrile phenomena, sometimes slight, in other cases marked, the pyrexia being as a rule strictly secondary or symptomatic. The skin feels hot, and usually perspires, but not profusely; the temperature is moderately raised, presenting no definite variations, though marked remissions are generally observed towards morning; the pulse is increased in frequency; and the digestive organs are much disordered, as evidenced by anorexia, thirst, thickly furred tongue, offensive breath, and constipation. The urine is generally deficient in quantity, and may be very scanty, high-coloured, and concentrated; its acidity is increased; and on standing, an abundant deposit of amorphous lithates often forms, varying in colour according to circumstances, being pale-buff, yellowish-red, dark or brick-red, or intense pink if the fever is high. The relative quantity of uric acid in a particular specimen of urine is often increased, but the absolute amount discharged within the twenty-four hours is much diminished. The patient is usually exceedingly restless, and cannot lie with comfort in any position; sleep is much

disturbed or altogether prevented; and cramps of the calves of the legs or of other muscles may still further aggravate the suffering. All these symptoms tend to increase the constitutional disorder. The temper is generally very irritable, or may even be violent.

The duration of the first fit of gout varies according to its severity and other circumstances. It usually ranges from four or five days to a week or ten days, but may last two or three weeks or more, there being then commonly intermissions or remissions, and several joints being involved in succession. The termination of the gouty paroxysm may be attended with critical phenomena, such as free perspiration, diarrhœa, or a very abundant discharge of urates. After the attack the patient may not recover his former state of health for some time; but not uncommonly he feels better than before, and as if the system had got rid of some deleterious element. As a rule, the affected joints are apparently quite restored after early attacks of gout; but more or less stiffness or even deformity may remain. œdema may also persist for a considerable time, especially if the inflammatory condition has been prolonged.

One of the characteristic features of gout is the tendency which it exhibits to recurrence in its acute form. This may not happen if the patient is sufficiently careful, but such an event is rare. The rule is for the attacks to be repeated, and to recur with increasing frequency. In not a few instances the second fit does not occur until an interval of two or three years or more has elapsed, but in most this is not prolonged beyond a year. The same interval may be noticed between the next few paroxysms, but as the disease progresses they return twice a year, then more frequently, and at last become more or less constant. At the same time the mischief extends as regards the joints. It may be limited to the great toe for some time, but in successive fits spreads to the other articulations of the foot, to the hands, the ankles and knees, the wrists and elbows, very rarely to the hips and shoulders. In short, gout tends in time to involve nearly all the joints indiscriminately, and several may be implicated during a fit. Moreover, those articulations which are repeatedly attacked become more and more disabled and deformed. The symptoms in connexion with a particular joint tend to diminish in intensity the more often it is affected. As additional articulations become involved, however, the general symptoms often increase in severity, and the patient does not recover in the intervals. The duration of the attacks becomes longer as their frequency increases. The rapidity of the progress of gout is very different in different cases; and the time taken to produce permanent mischief in the joints varies considerably.

It must be borne in mind that considerable variations in the intensity and exact characters of the symptoms of acute gout are observed in individual cases. In feeble persons the subjective and objective symptoms may be comparatively slight, the inflammation assuming an asthenic character, but then the ultimate effects upon the joints are often much worse. In some instances gout develops very insidiously, without any evident acute attacks. The pain in connexion with a particular joint depends considerably on its structure, being usually much more marked if its ligaments, or the parts around, are rigid and unyielding. Previous injury or disease affecting a joint may likewise modify the symptoms. Some individuals suffer much more than others, being more susceptible to painful impressions.

**3. Chronic Articular Gout.**—In course of time more and more of the joints become permanently and obviously changed, and prevented from fulfilling their functions properly, so that a condition of chronic articular gout is established, exacerbations still occurring, however, from time to time. The permanent changes are indicated by the articulations becoming enlarged, deformed, and irregular in shape, often presenting nodulations or bulgings, which may attain a large size. They are also stiff and crippled in their movements, at last becoming quite immovable and rigid; and being either permanently flexed, extended, or sometimes even bent backwards. The interference with movement and the deformity do not bear any necessary proportion to each other, the one or the other predominating according to the mode in which the uratic deposit has taken place. The more this infiltrates the ligaments and surrounding tendons, the greater becomes the impediment to movement. The fingers are particularly liable to be much altered in chronic gout. A common condition is said to be stiff flexion of the metacarpophalangeal and second phalangeal joints of a finger, with over-extension of the first phalangeal joint, so that its knuckle presents a deep hollow. Deflexion of the fingers to the ulnar side of the fore-arm is observed in some gouty cases. The toes are also sometimes distorted towards the outer side of the foot.

Gouty concretions in connexion with joints feel hard, and by their mere mechanical and irritative effects they are liable to cause damage to the adjoining structures. They may be seen stretching or shining through the skin, and causing it to assume a bloodless appearance, or, on the other hand, rendering it congested and bluish, the veins also being enlarged. Ultimately a gouty abscess may form around a concretion, which opens externally; or the skin may merely give way from the continued pressure. Thus the *chalk-stones* are exposed, and may come

away either in a liquid form or as solid particles or masses, or occasionally there is a free discharge of pus as well. Ulcers are left, of an unhealthy and atonic character, and usually presenting no disposition to heal. There may be a number of these ulcers in the same individual, on the hands and feet.

When bursæ are involved, much deformity is produced. They are easily felt, usually presenting a combination of hardness and fluctuation, due to the presence of both concretions and fluid in the bursal cavity. These signs are chiefly noticed in the bursa over the olecranon. Abscesses may also form in connexion with these deposits, and the latter may thus be completely got rid of, the abscess subsequently healing rapidly.

In cases of chronic articular gout the general system tends to become permanently affected. The patients are generally more or less feeble and wanting in tone; they may be thin and pale or sallow-looking; or plethoric, but with flabby tissues, and presenting signs of languid circulation, with enlarged capillaries about the face. They often suffer from disorders of digestion and other symptoms already described; but not uncommonly, as gout assumes a more chronic form, patients feel better, becoming habituated to their condition. It is remarkable that those suffering from numerous gouty abscesses often exhibit but little constitutional disturbance.

The urine in chronic gout generally becomes abundant, very watery and pale, of low specific gravity, deficient in solid ingredients, especially in uric acid, which at times may be almost completely wanting, or it may be thrown out in an intermittent manner. Deposits of urates in urine are not often observed in cases of advanced chronic gout, except perhaps before an acute exacerbation. Glycosuria is not uncommon in gouty persons, and there may be distinct saccharine diabetes. Oxaluria is also frequent.

**4. Irregular Gout.**—The clinical phenomena which are recognised as irregular manifestations of gout may assume either an acute or chronic form. They may be observed in persons who are distinctly subject to articular gout; or in those in whom the disease is not so obviously revealed. Moreover, their intensity is often in an inverse ratio to that of the joint-affection, and the two classes of symptoms may exhibit a remarkable tendency to alternation, when the articular symptoms are prominent those connected with other parts being slight or absent, and *vice versâ*. Acute symptoms are particularly liable to arise when, from any cause, during the progress of a gouty fit the joint-inflammation is checked suddenly or rapidly—*retrocedent gout*. At other times internal symptoms seem to be due to a want of development of the external phenomena—

*suppressed gout*; and when the latter appear, the former subsides.

It must suffice to indicate here the general nature of the symptoms of irregular gout. The *acute* symptoms are mainly associated either with the digestive apparatus, the vascular system, the respiratory organs, or the nervous system. In connexion with the *digestive apparatus*, acute dysphagia may occur, attended with spasm of the pharynx and œsophagus. The most important symptoms belonging to this group are, however, those due to gastric disturbance. This may be of the nature of severe cramp or gastralgia, characterised by a sudden, intense, spasmodic pain in the epigastrium, relieved by pressure, and accompanied with a sense of great weight and oppression; the patient presenting an aspect of much suffering, distress, and anxiety; or being even more or less collapsed and prostrated. In other cases the symptoms are those of acute gastritis, bilious vomiting being prominent. Intestinal colic, or even muco-enteritis, may also occur in connexion with gout. Hepatic disorder is common. The *vascular system* is not uncommonly implicated. The heart is liable to be disturbed in its action during the attacks of gastralgia, but this disturbance may also be observed independently. There may be severe palpitation, the action of the heart being very rapid, irregular, or even intermittent, accompanied with most unpleasant sensations over the cardiac region, precordial anxiety, often a feeling of oppression or constriction, dyspœna or a sense of suffocation, and much distress, anxiety, and dread of death; the pulse tends to be weak and small, or may be irregular; and sometimes the attacks are attended with signs of collapse. In other instances the cardiac disorder is evidenced by very feeble or slow action, with a tendency to syncope. Again, there may be all the phenomena of a severe anginal attack (see ANGINA PECTORIS). Irregular gout affecting the *respiratory system* is mainly indicated by asthmatic paroxysms. In some cases there is a marked liability to acute catarrh of the air-passages, and crystals of uric acid have been found in the expectoration. Bronchitis is more obstinate in gouty subjects. Pulmonary congestion is also supposed to be a manifestation of retrocedent gout in some cases; and gouty pneumonia has been described. In connexion with the *nervous system* gout may give rise to attacks of severe headache or vertigo; delirium or even acute mania; epileptiform or convulsive fits; cerebral or spinal meningitis; acute neuralgia, either external or internal, and especially sciatica, probably due to neuritis; or severe muscular cramps. Apoplexy from cerebral hæmorrhage has been attributed to suppressed or retrocedent gout, but this can only occur if the vessels of the brain are diseased. Cerebral

congestion might give rise to a temporary apoplectic attack. Among the acute manifestations of irregular gout may be further mentioned certain skin-affections, as eczema, erythema, or urticaria; affections of mucous membranes, such as the conjunctivæ and lacrymal passages; functional renal disorder, with albuminuria, or irritability of the bladder; and external local signs of inflammation, associated with uratic deposit.

Many of the more *chronic* symptoms associated with gout have already been pointed out, when speaking of its premonitory symptoms, and only certain special phenomena need be alluded to here. Chronic skin-diseases are of frequent occurrence, namely, psoriasis, chronic eczema, prurigo—either local or general, and acne. These may alternate very distinctly with articular gout. The nails are sometimes peculiar, being coarse and fibrous, striated and fluted, or lined vertically. Their substance may grow thick and brittle. In some gouty subjects daily paroxysms of heat and redness of the nose, attended with severe itching and irritation, cause considerable annoyance or distress. Violent sneezing fits are common. Many of these individuals are also liable to chronic catarrh, affecting the throat and the air-passages; and special appearances of the throat, tongue, and teeth have been described. In course of time gouty patients often become permanently asthmatic, the lungs being emphysematous, and dry bronchial catarrh being established. Gravel or urinary calculus may give rise to symptoms; or those indicative of chronic urethritis or cystitis may be present, especially in persons advanced in years. Permanent disorders of sensation, or slight local paralysis, may result from chronic changes involving particular nerves. Gouty persons are usually very sensitive to pain. Certain affections of the eyes have been attributed to gout, and amongst them a form of gouty iritis, described by Hutchinson as coming on insidiously, and almost painlessly, and ending in destruction of the eye. Changes in the external auditory meatus may also be associated with this complaint.

With regard to tophi, these can be seen or felt, provided they are superficial. Those connected with the helix of the external ear are most common; but they may also be looked for in the sclerotic or eyelids, in the nose, and under the skin, in the region of tendinous aponeuroses, especially in the leg or thigh. Tophi are originally liquid, and if one is punctured at an early period, an opalescent or milky fluid escapes, which on microscopic examination is found to contain an abundance of delicate, acicular crystals; subsequently they become more consistent, and ultimately quite solid and hard, being then made up entirely of these crystals, which are closely aggregated together and interlaced. Taking the ear as an illustration,

at first a small elevation appears under the skin of the helix, like a vesicle, having a soft feel. This gradually hardens, until finally a little bead-like or pearl-like body is formed, presenting a whitish colour as seen through the skin. In course of time the cutaneous covering may be destroyed, leaving the little concretion exposed; or this may even become detached and removed, so that only a small depression is left.

**5. Symptoms due to Chronic Organic Diseases.**—In addition to what has been stated under the preceding heading, it is only necessary to refer here to the renal and cardio-vascular changes which are associated with gout. Disease of the kidneys is indicated mainly by the changes in the urine, which may be slightly albuminous, or even contain a few casts. Other symptoms of chronic Bright's disease may be present, but they are often very obscure. The cardiac lesions are revealed by their respective physical signs; and there may be symptoms, first of excessive cardiac action from hypertrophy, and subsequently of cardiac failure, as well as those of particular valvular diseases. The vascular changes are evidenced by examination of the arteries; and by their effects upon the circulation. It is impossible to describe within the limits of this article the numerous symptoms resulting from the various chronic organic diseases which may develop during the progress of gout, and which in many cases ultimately produce a very complicated clinical picture.

**DIAGNOSIS.**—The degree of difficulty experienced in arriving at a diagnosis with respect to gout is very variable in different cases. A distinct gouty diathesis or tendency may be present, which it is important to recognise, while the joints are quite free from any apparent mischief. In most cases, however, the diagnosis has to deal with the nature of an articular affection, and to determine whether this is gouty or not. The chief diseases from which gout has thus to be distinguished are acute or chronic rheumatism, rheumatoid arthritis, and possibly gonorrhœal synovitis. The data upon which a conclusion has usually to be formed with regard to a first attack, are the presence or absence of an hereditary tendency to gout, as well as its intensity; the age and sex of the patient; the social position, occupation, and previous habits; the presence or absence of any obvious cause for the attack, or of premonitory symptoms; the localisation and characters of the joint-affection; the general symptoms; the characters of the urine; the duration of the illness; and the absence of any cardiac complication.

The subsequent progress of gout is important in diagnosis, with its tendency to periodic recurrence; and if the metatarso-phalangeal joint of the great toe is alone inflamed several times in succession, or even if only

the smaller joints of the feet and hands are implicated, the diagnosis of gout is tolerably certain. The permanent articular changes induced by gout also become evident in time; as well as, perhaps, tophi in other parts, which should be carefully searched for in any doubtful case. Moreover, the urine presents peculiar changes as the disease progresses, and may give evidence of renal mischief. In very doubtful cases it might be desirable to raise a blister, or even to take a little blood from the patient, and endeavour to obtain crystals of uric acid from the serum.

It will here be expedient to offer a few remarks as to the diagnosis from gout of the diseases with which it is most likely to be confounded.

With regard to acute rheumatism, the absence of any hereditary tendency to gout in any doubtful case may be in its favour, and possibly there may be a family predisposition to this complaint. It occurs most frequently for the first time in early life, from sixteen to twenty years of age, and is not uncommon even in young children. Rheumatism, though more common among males, often attacks females. It is not favoured by the habits which generate or promote gout, and affects all classes of persons, but especially those who from their occupation are liable to be exposed to cold and wet. Such exposure, or some other definite cause originating 'a cold,' usually accounts for an attack of acute rheumatism; and it is not preceded by any particular premonitory symptoms. The joints involved are the middle-sized or the larger ones, several of which are generally implicated in succession during the illness, the rheumatic inflammation exhibiting an erratic character; the local symptoms tend to be less severe than in gout; there is less marked œdema about the joints, and no enlargement of the veins, or subsequent desquamation. It must not be overlooked, however, that gout may attack the middle-sized joints. Pyrexia is high as a rule in acute rheumatism, and is often quite out of proportion to the extent of the articular affection; while profuse acid perspiration is almost always a prominent phenomenon. The urine is simply febrile. The attack lasts a considerable time, perhaps several weeks, if it is at all severe; while during its course some acute cardiac inflammation is very liable to supervene. In addition to other differences, chronic rheumatism is at once distinguished from gout by the entire absence of uratic deposits.

Rheumatoid arthritis is usually met with in females between twenty and forty years old. There is neither hereditary taint, nor a history of such habits as generate gout, but, on the contrary, the patients are generally poor, hard worked, and badly fed; all joints seem to be equally liable to be affected, both large and small, and the local symptoms are

not of a very acute character, though the pain may be very severe, but they tend to continue for a long period; the general symptoms are mainly those of debility and anæmia, and Dr. Kent Spender has drawn attention to certain prominent and early symptoms, especially cold hands and feet, constant dampness of the hands, pigmentation of the skin, and a rapid and compressible pulse. Rheumatoid arthritis is a disease which tends to progress, involving joint after joint, but it presents no periodicity in its attacks, and often advances without any intermission, as a subacute or chronic disease. Ultimately it often causes much deformity and crippling of the articulations, which creak and grate on movement, but this results from a very different pathological change from that which takes place in gout, for there is not the slightest deposit of urates, either in the joints or elsewhere; nor in the most extreme cases can any uric acid be obtained from the serum. The urine presents no special characters; and the kidneys are not diseased.

Gonorrhœal synovitis is distinguished from gout by the age of the patient; the distribution of the joint-affection; the absence of any tendency to recurrence, except in association with a fresh urethral discharge; and the different nature of the morbid changes, tophi never being formed.

As exceptional points bearing upon the diagnosis of articular gout, the following may be mentioned. It has happened that pyæmia beginning in the great toe has been mistaken for gout, but the progress of the case would soon clear up any doubt under such circumstances. Again, articular inflammation from injury might resemble gout; and, moreover, it must be borne in mind that such an injury may really set up gouty inflammation for the first time, so that the joint may not recover properly. In some individuals the ends of the phalanges of the fingers are enlarged, especially the terminal ones, and cause nodulations—*digitorum nodi*—which resemble those of gout, and are by some regarded as being of a gouty nature.

The importance of recognising the signs of the gouty diathesis, apart from the joint-affection, has already been alluded to. Equally important is it to be prepared for the acute symptoms in connexion with internal organs which occur in this diathesis, whether along with or independent of articular disease. Lastly, in any gouty case the detection of the organic diseases liable to be set up in its course is of great moment in diagnosis, especially renal and cardio-vascular changes; and also the association with their proper cause of catarrhal affections, skin-diseases, and other complaints, when these are really of a gouty nature.

PROGNOSIS.—The first point relating to the prognosis of gout which calls for notice refers to the immediate dangers in any particular

case. A simple acute attack of articular gout rarely, if ever, kills the patient. When, however, internal organs are implicated, the matter becomes much more serious, and a fatal result may occur, so that the prognosis must be a guarded one under such circumstances. The danger then becomes much greater if the complaint has been long-established, and if the kidneys or other important organs have become structurally diseased. Indeed, these diseases of organs are often themselves immediately dangerous. Again, any acute inflammation occurring in a confirmed gouty subject is the more serious on this account; and the same remark applies to injuries and shocks of all kinds.

In the next place, the future of a gouty patient has to be considered, as regards the prevention of subsequent attacks, or the cure and eradication of the disease. It must always be recognised that gout is a recurrent affection, and complete immunity can never be guaranteed, once the complaint has declared itself. At the same time undoubtedly not a few cases have occurred in which there has been but one attack, but this can only be expected under certain conditions. In giving an opinion on this point, the prognosis in any individual case will depend upon:—  
1. The degree of hereditary tendency to gout.  
2. The age of the patient; for the earlier the period at which the disease begins, the less hopeful is the prospect of a cure.  
3. The time the complaint has lasted from its commencement; and the frequency and duration of the gouty fits. If gout has become established, and especially if distinct tophi have formed, it is quite impossible to eradicate it.  
4. The habits, mode of living, and occupation of the patient. It is only when the patient is prepared to adhere strictly to proper rules of living that a cure can be hoped for. Those who in their occupation are tempted to drink much, or who are exposed to cold or wet, are much less likely to be cured. It may be remarked here that gouty subjects are less able than others to resist exposure.

Another point bearing upon prognosis refers to the duration of life in gouty persons. If the disease comes on late in life, and the paroxysms only occur at long intervals, while the organs are free from any organic mischief, gout may not appreciably shorten life, and the patients may even enjoy good health up to an extreme old age, provided they are sufficiently careful in their mode of living, and no accidental complications arise. Chronic gout does, however, unquestionably tend to shorten the duration of life, to a greater or less degree in proportion to its severity, and more especially to the indications present that the kidneys, heart, arteries, or other important organs are organically diseased. This tendency is now recognised by most life-insurance companies.

It has been affirmed that gout is a pro-

tection against certain other diseases, such as phthisis and diabetes, and therefore its presence has been in some instances regarded as a benefit, but how far there is any real foundation for this belief is a matter of very considerable doubt.

**TREATMENT.**—It is important at the outset to lay stress upon the fact that, although there are certain well-defined principles applicable to the treatment of gout in its various phases, it is a great mistake to follow any regular routine method under all circumstances, and every case must be considered on its own merits. It will be convenient to discuss this part of the subject under certain general headings, premising that the administration of medicines is often the least important part of the treatment, and that the habits of life of the patient always need thorough supervision in all their details.

**1. Preventive and Curative Treatment.**—In a number of cases the primary object which should be aimed at is to prevent the development of gout; or to eradicate the tendency to subsequent attacks, if it has once declared itself. These objects have especially to be kept in view in dealing with individuals who have a marked hereditary predisposition to the complaint; in cases where it has appeared at a comparatively early period of life, or where it is in an early stage; and in persons who, from their occupation, known habits, or the symptoms they present, are likely to become gouty. Moreover, even when confirmed gout has been established, preventive treatment may diminish the number of acute attacks, or possibly even avert them altogether; as well as obviate the implication of organs essential to the well-being of the economy. In order to carry out these purposes in any particular case, the patient must intelligently recognise the fact that success in treatment mainly depends upon himself, and upon his willingness constantly to regulate his mode of living according to principles suitable to his condition, which need to be more or less strict in different instances. The general nature of the rules to be adopted will be evident from a consideration of what has been stated in discussing the ætiology and pathology of gout, but they require to be briefly noticed here.

(a) *Diet.*—Moderation in the quantity of food is the first point to be attended to in the treatment of the gouty diathesis. It is not necessary or desirable to restrict persons who are gouty to a very low diet, especially if they are in any way weak, but an amount sufficient for proper nourishment in each individual case must be consumed, and at no meal should the stomach be uncomfortably filled. The meals must be taken at regular times, and not hurriedly, so as to avoid bolting of the food. Very late dinners, as well as suppers, should be prohibited, but it suits many persons better to dine at six or half-

past six o'clock than at midday. The nature of the food is highly important. It is quite unnecessary, and probably would be in most instances injurious, to restrict the patient absolutely to a vegetable diet, but a due proportion of animal and vegetable aliments should be allowed. At the same time, in persons who have any tendency to gout, an essential part of the treatment often consists in diminishing the amount of meat which they consume, and they should not take it more than once a day. Indeed, the aim must be to reduce all kinds of nitrogenised food, whether animal or vegetable, to such an amount as the system can satisfactorily dispose of, with due regard to the proper nutrition and strength of the body. As regards the kinds of animal food which are suitable for gouty subjects, white fish, chicken or fowl, game, tripe, sweetbread, and mutton are the best forms. Tender and underdone beef may be taken in moderation from time to time, if it is readily digested. Pork or veal, dried and salted meats, and rich dishes of all kinds, should be avoided. Lightly boiled or poached eggs may be permitted. Vegetables which contain much woody fibre, or which create flatulence, must be eschewed; and certain articles, such as rhubarb, tomatoes, especially when cooked, asparagus, and sorrel, are often injurious to the gouty, as well as others which contain much acid. On the other hand, some special beneficial influence has been attributed to celery. Those who are subject to gout should either abstain altogether from, or only take a very limited quantity of, sugar and saccharine articles of diet. Hence, although digestible fruits may often be taken with advantage in moderation, those which are very sweet must be used with particular caution. Stewed and baked fruits often agree well, but fruit-tarts, and, indeed, pastry of all kinds, should be interdicted. The juice of oranges or lemons is considered beneficial for gouty persons. Starchy foods are permissible in moderation. It may be laid down as a rule to be invariably followed, that gouty patients should limit themselves to simple meals, and not indulge in a number of courses; and that they should avoid everything which experience tells them is, in their case, indigestible.

(b) *Drink.*—The question of drink demands the most careful consideration and attention in every case in which gout is either threatened or has become established. No stringent rules can be laid down, applicable to all cases, but there are certain broad principles which have to be borne in mind. An abundance of good and pure drinking-water is to be commended, but it should be taken mainly apart from meals. It is a good plan for the subjects of lithiasis to take a tumblerful of, either cold or hot, water before retiring to rest at night. Effervescing potass- or lithia-water may be

substituted for ordinary water with advantage, but not soda-water. Tea and coffee may be taken in moderation, provided they do not disagree. With reference to alcoholic drinks, in a considerable number of instances one of the first objects in the treatment of the gouty diathesis should be the regulation of the use of this class of beverages. This indication is obvious enough when the condition is evidently due mainly to intemperance; but even when the patient is temperate, it may be that in his case the amount consumed needs to be reduced or total abstinence enforced, especially if there should be a strong hereditary predisposition to gout, or if the complaint appears in early life. Some patients are undoubtedly better if they take no stimulants whatever; others, however, can take those of a proper kind, and of good quality, in strict moderation, with advantage. It may be laid down as a general rule that malt liquors and all stronger wines are injurious, and should be interdicted. Those which are most suitable are sound light claret, hock, moselle, chablis, or saunterne. A small quantity of good dry sherry suits some gouty patients very well. A little brandy, well diluted, often agrees better than any other kind of alcoholic liquor; in other cases whisky or gin answers best. Whatever stimulant is selected, it should only be taken at meal-times, and the habit of drinking between meals is strongly to be deprecated. Persons who are distinctly gouty should avoid any excess on every occasion; and even if they do not absolutely abstain, they may find it beneficial to do so from time to time, especially if there is a tendency to the development of gouty symptoms.

(c) *General hygiene.*—Inadequate exercise is a hygienic error which has frequently to be rectified in the treatment of the gouty state. Sedentary habits must be combated, whether due to occupation or indolence; and it must be insisted upon that a due amount of outdoor exercise is taken daily, but prolonged exertion, tending to cause fatigue and exhaustion, must be avoided. Walking and horse-exercise are highly beneficial, especially in the case of those who live rather too freely. Even carriage-exercise is useful, so that the patient may have the benefit of the fresh air. For those who cannot have much regular exercise, lawn-tennis, fencing or single-stick, golf, and the like may be serviceable. As bearing upon the prevention of an acute fit of gout, it is an undoubted fact that some individuals who are subject to the disease seem to be able to ward it off by walking, riding, or adopting some other form of prolonged and violent exercise on the first intimation of a threatened attack. Gouty persons should retire to rest and get up early. They should as much as possible avoid excessive mental labour, or any great strain upon the mental faculties, but especi-

ally worry and anxiety of all kinds. Attention must be paid to the cutaneous functions, the surface of the body being also protected from the injurious effects of cold. Warm clothing should be worn, in keeping with the weather, and those who can bear it should wear flannel next the skin. With regard to baths, many gouty persons are decidedly the better for using a cold or tepid bath every morning, followed by energetic friction; in other cases the employment of the warm bath at intervals, or even of the Turkish bath, answers best. A course of treatment in a hydropathic establishment from time to time, under due medical supervision, is sometimes decidedly beneficial. Climate demands attention, whenever the circumstances of the patient allow a choice to be made. It may be advisable for gouty subjects to reside permanently in some warm and equable climate, or at any rate during the winter and early spring. In this way attacks may often be warded off, and the disease thus prevented from making progress. Those who are obliged to remain in this climate during the inclement seasons should avoid exposure to wet and cold, as well as sudden changes of temperature, and night air. The bedroom should be warm and well-ventilated; and in cold weather it is desirable to keep a fire burning during the night. Unduly heated and badly ventilated rooms, as well as crowded places of public resort, must be eschewed.

(d) *Medicinal treatment.*—There can be no doubt but that the judicious use of certain medicines may assist materially in warding off or mitigating the gouty condition, and in preventing the occurrence of acute paroxysms. Those which are specially called for in cases of established chronic gout will be presently considered. In the meantime, it may be stated that the digestive functions require particular attention, and medicines which help these functions are often of the greatest service, if they should be disordered. A course of alkalis or acids, according to the indications in each case, may prove most serviceable, combined with some simple bitter infusion or tincture. Certain alkalis and alkaline earths are also valuable on account of their power in promoting elimination of lithic acid, by forming soluble salts with this acid. The careful observations of Dr. Haig have shown that alkalis increase the secretion of uric acid, while acids decidedly diminish it. The alkalis which are most useful are salts of potassium and lithium, the urate of lithium being the most soluble of all. The bicarbonate of potassium, carbonate of lithium, or the citrates are the best salts to administer. They should be taken well-diluted, and on an empty stomach. Magnesia or its carbonate may be given with advantage if there is much acidity, and if the bowels are habitually constipated. Saline aperients are often

of great service, and they may be advantageously administered in small doses, freely diluted, and regularly repeated, when they also act on the kidneys. In many cases other aperients may be employed at intervals with advantage, but strong purgatives must be used with particular caution. An occasional dose of blue-pill or calomel may be decidedly beneficial. The administration of other chologogues from time to time may also be of service.

(e) *Mineral waters and baths.*—Certain mineral waters are of great value in the treatment of the gouty diathesis, when employed under proper medical supervision, and they offer the advantage that patients will often use them, when they will not undergo a course of regular medicinal treatment; while the water thus taken internally is itself of service. Space will not permit a long discussion of this subject here, and it must suffice to mention that in different cases the kind of mineral water employed must be varied according to the object desired to be accomplished, and according to the indications presented by the patient, for what suits one may be highly injurious to another. These waters are employed both internally and in the form of baths. Those chiefly used in gouty conditions are the waters of Bath, Buxton, Cheltenham, Clifton, Harrogate, Leamington, Malvern, and Woodhall in this country; Strathpeffer and Moffat in Scotland; Llandrindod in Wales; Lisdoonvarna and Lucan in Ireland; and Carlsbad, Marienbad, Vichy, Wiesbaden, Baden-Baden, Ems, Royat, Aix-la-Chapelle, Aix-les-Bains, Evian-les-Bains, Contrexeville, Dax, La Bourboule, Luchon, Friedrichshall, Hunyadi János, Pullna, Schlangenbad, Teplitz, Seidlitz, Homburg, Kissingen, Rubinat, Wildbad, Ragatz, Gastein, Elster, Tarasp, Apollinaris, and other continental waters. Some of these may be taken regularly or at intervals, for the purposes which they respectively fulfil; or, if circumstances permit, a systematic course of treatment from time to time, at certain of the places mentioned, may be recommended. See MINERAL WATERS.

Before leaving the subject of the preventive treatment of gout, it needs to be insisted upon that those who are particularly liable to this disease, whether from hereditary predisposition, occupation, or any other cause, should pay special attention to preventive measures; and also that those in whom the disease has already manifested itself must take every precaution to avoid the known causes of acute attacks, for each attack tends to make matters worse.

**2. Treatment of Acute Gout.**—When a fit of acute articular gout sets in, it is on no account to be permitted to run its course unmodified by treatment, else serious mischief is liable to arise. At the same time it is requisite to refrain from adopting too active

measures. Our objects should be to shorten the attack; to restore the affected parts as nearly as possible to their normal condition; and to relieve symptoms. In the first place, particular attention must be paid to the diet. The aim should be to make this as low as is compatible with the condition of the patient, especially if the fit is of a very acute and sthenic type. In young and strong patients the diet should at first consist of milk, farinaceous articles, and abundance of water, barley-water, or toast-and-water. Those who are advanced in years, weak, or broken-down in health, or who have long suffered from gout, require a more nutritious diet, but it should be easily digestible, consisting of beef-tea and good soups, milk, eggs beaten up, and such articles, the quantities being regulated by the requirements of each case. As the symptoms subside, the food should be gradually improved, white fish, fowl, and meat being allowed in succession, but in strict moderation; and due care must be exercised subsequently. If possible, all kinds of alcoholic stimulants should be interdicted, but it may not be judicious to cut them off entirely in some cases, either on account of the previous habits or present condition of the patient, and then it is best to give a definite quantity of brandy or whisky, well diluted, with the food. For those who cannot take spirits, a little good hock or sauterne answers well.

As regards medicinal agents, colchicum has long held the most prominent place in the treatment of acute gout, and is commonly regarded as a specific. There can be no doubt as to the influence of this drug in relieving the symptoms and shortening the paroxysm of gout, although it is by no means settled how it acts. Its effects must be watched, however, for it does not agree in every case. It has been alleged that colchicum renders the patient more liable to subsequent attacks of gout, but for this notion there does not seem to be any real foundation. The tincture or wine of colchicum may be given in doses of ten to twenty or even twenty-five minims every four or six hours, and either of these preparations may be combined with the citrate or bicarbonate of potassium or with a salt of lithium.

Salicylates, especially salicylate of sodium, have been much recommended in the treatment of acute gout, and Dr. Haig is strongly in favour of them, having found that they materially increase the discharge of lithic acid. They certainly have proved useful in some cases in the writer's experience. Another class of drugs employed are antipyrine, antifebrin, and allied agents. A remedy named piperazin has been introduced for the treatment of gout, which is said to be the most powerful solvent of uric acid known, but the writer has no experience of its practical use.

It is necessary to keep the bowels acting freely by means of suitable aperients, and saline purgatives are of considerable value for this purpose. Other aperients, such as compound rhubarb pill, colocynth, podophyllin, calomel or blue pill, may be employed in appropriate cases. Diluents may be given freely, in order to promote the action of the kidneys; and if the cutaneous functions appear to be defective, some mild diaphoretic may be administered, or it may even be desirable to employ the hot-air or vapour bath. Medicines may be needed for the relief of symptoms, especially pain and sleeplessness, for which Dover's powder or other preparations of opium, belladonna, chloral hydrate, sulphonal, or bromides may be indicated, but they must be used with discretion. In very severe cases hypodermic injection of morphine is of much service.

Venesection ought never to be practised in the treatment of acute articular gout; for although immediate improvement may perhaps be thus produced, the ultimate results are highly unsatisfactory. Even the local removal of blood, by means of leeches applied near an affected joint, is dangerous, and had better be avoided, on account of the permanent local mischief which such a measure is liable to induce.

*Local treatment.*—The affected parts in acute gout should be kept entirely at rest, and placed in a comfortable position, supported by pillows, and either horizontal or elevated, according to the feelings of the patient. In ordinary cases it is sufficient to wrap up the joints in flannel, or to surround them with cotton-wool completely covered with oil-silk or gutta-percha sheeting, according to Garrod's method, by which means a kind of local vapour-bath is kept up. If the pain is considerable, other local applications might be used, of which the most efficient are warm fomentations, to which tincture of opium or belladonna may be added, poppy fomentations, lead and opium lotion, localised steaming, belladonna liniment, tincture of aconite, oleate of morphine, or a solution containing morphine and atropine. Some practitioners prefer evaporating lotions containing alcohol, ether, or chloroform; others warm water compresses. A blister in the neighbourhood of a gouty joint may be of service, if the attack be asthenic, and also if effusion or much stiffness persist. During recovery, benefit may be derived from careful friction with some stimulating liniment, application of iodine, shampooing, gentle passive movements, douching with salt and water, or the application of a light bandage or elastic support, should there be a tendency to permanent thickening and stiffness, or to œdema and enlargement of the veins.

The acute forms of *irregular gout* must be treated according to their nature, and here it must suffice to offer a few general remarks

on the subject. If serious internal symptoms arise, which are distinctly of a gouty nature, and especially if they occur as retrocedent phenomena, it is important to try to excite inflammation in the joints, by means of local heat, friction, and sinapisms. Colchicum may be of service in the non-articular, as well as in the articular form of gout. In painful affections opium or other anodynes are called for; and frequently the administration of alcoholic and other stimulants is indicated, with antispasmodics, such as ammonia, ethers, camphor, musk, or belladonna, especially when the stomach or heart is affected. In conditions attended with signs of much depression or collapse, external heat may be applied over the body, or sinapisms to the limbs and over the cardiac region. In the treatment of inflammatory diseases associated with gout much care is required, especially in resorting to depletory measures. The existence of the diathesis must always be borne in mind.

### 3. Treatment of Chronic Gout.—

When gout becomes an established chronic disease, the same general rules of treatment are to be observed as in the prevention or attempted cure of the complaint, but they often need modification in particular cases, according to the conditions present. Similar medicinal remedies are also indicated, lithium salts being particularly valuable, and they may even aid in removing gouty deposits; but others may be added to the list, which are suitable in different cases. Colchicum is often of much service, taken habitually or from time to time, in the form of one of the extracts at night, or a few minims of tincture or wine two or three times a day, combined with other medicines. Among the many therapeutic agents recommended in the treatment of chronic gout under different circumstances may be mentioned benzoic acid or benzoate of ammonium, phosphate of ammonium or of sodium, iodide of potassium, bromide of potassium, carbonate of aluminium, lime-juice, resin of guaiacum, ammoniacum, and tonics, especially quinine, tincture or infusion of cinchona, or mild ferruginous preparations. Undoubtedly most of these are of use in appropriate cases of chronic gout, to serve their special purposes, but they must be used judiciously, especially iron. Symptoms connected with various organs frequently call for attention in this disease, and they must be treated by appropriate remedies. It may be remarked that, should diarrhoea set in in gouty cases, it must not be hastily arrested, as this may be a mode of relief to the system. With regard to the local conditions in chronic gout, it is affirmed that the prolonged use of some of the mineral waters previously mentioned, both internally and in the form of baths, such as those of Aix-la-Chapelle, Aix-les-Bains, and Baden-Baden, may succeed in removing to some extent deposits of

urates. and in diminishing stiffness and thickening of joints. For these purposes local measures may also be of service in some instances, provided the morbid changes are not too far advanced, namely, occasional blistering or application of iodine; the prolonged use of wet bandages; friction with liniments; shampooing and passive movements; or systematic compression by means of some non-irritating plaster. Solutions of alkalis or alkaline carbonates, and especially of carbonate of lithium, have been kept applied to gouty joints and other parts for a long time, under the belief that deposits of urates may be thus dissolved. In the writer's experience no such effect has ever been thus produced, although the constant application of warm moisture may be useful. Superficial uratic deposits should not be interfered with unless they become troublesome, when it may be desirable to puncture the skin, and let the contents out. The propriety of removing large masses by operation may come up for consideration, but this should only be attempted if there is every probability that they can be entirely removed without any serious difficulty, and if the patient is in a fit state for the operation. When abscesses or ulcers form, they come under the treatment of the surgeon; but it may be observed that simple dressings usually answer best in these cases, and they may sometimes be advantageously dressed with solution of carbonate of potassium or lithium. It must not be attempted to heal them up too rapidly, as the discharge may be a relief to the system, and it may even be necessary to enlarge the opening of a gouty abscess. Under any treatment it is by no means an easy matter to induce lesions of this kind to heal in cases of chronic gout.

The treatment of the various chronic organic diseases which are liable to arise in the course of gout must always be kept in mind, but the reader is referred to other appropriate articles for a consideration of this part of the subject.

FREDERICK T. ROBERTS.

**GRAHAM'S TOWN, in Cape Colony.**—See AFRICA, SOUTH.

**GRAND MAL (Fr.)**—A term applied to epilepsy when it assumes the form of a severe convulsive attack. See EPILEPSY.

**GRANULAR KIDNEY.**—A morbid condition of the kidney, in which this organ is the seat of fibroid change, and as a result becomes contracted, hard, and granular. See BRIGHT'S DISEASE.

**GRANULAR LIVER.**—A synonym for cirrhosis of the liver, in which the organ presents a granular appearance, on its surface and on section. See LIVER, Cirrhosis of.

**GRANULATION** (*granulum*, a little grain).—In medical pathology, granulation is synonymous with tubercle in its isolated form, the individual tubercles being called 'grey' or 'yellow' granulations, according to their appearance (see TUBERCLE). In surgical pathology, the term 'granulations' is applied to small vascular prominences, consisting of embryonic tissue, growing on the surface of wounds or ulcers, and by which the healing process is carried on—whence the expression 'healing by granulation.' When granulations assume the appearance of an exuberant growth they constitute what is called 'proud flesh.' See CICATRISATION; and ULCER and ULCERATION.

**GRAVEL.**—DEFINITION.—The deposit in and escape from the urinary passages of gritty particles with the urine.

ÆTIOLOGY.—The same causes which produce dyspepsia are frequently productive of lithic-acid gravel, such as indolent habits, excess of food and drink—especially of nitrogenous and saccharine articles, and the too free indulgence in the use of fermented liquors. Endemic causes connected with climate and the nature of the drinking-water, hereditary predisposition, and many slight or serious organic diseases, may explain the appearance of gravel in those, and especially in women, who commit no dietetic excess, or who are total abstainers. See OXALIC-ACID DIATHESIS; PHOSPHATIC DIATHESIS; and URIC-ACID DIATHESIS.

VARIETIES.—Gravel may be composed of (1) lithic acid and its compounds; (2) oxalate of lime; (3) phosphate of lime; or (4) the triple phosphate. By far the most common form of gravel, and that which alone need now be considered, is lithic acid. This, owing chiefly to its great insolubility, is frequently deposited in the kidney and bladder, and is seen in the newly passed urine in the form of the well-known reddish-brown crystals, often described as resembling cayenne-pepper grains. The supernatant urine is generally clear, rather dark in colour, and of a distinctly acid reaction.

SYMPTOMS.—The passage of uric-acid crystals or gravel frequently causes no subjective symptoms, and is consistent with perfect health. Sometimes, however, it gives rise to, or is accompanied by, both general and local disturbance of function. The general symptoms are those of dyspepsia—namely, flatulence and heartburn after meals, eructations, headache, muscular cramp, depression of spirits, and a sense of malaise. Locally, there is dull aching in the lumbar region, not increased by movement; frequent micturition; a sense of heat and irritation at the neck of the bladder and along the urethra, especially during and after voiding water; and sometimes the appearance of a faint cloud of mucus or a slight tinge of blood in the urine.

**TREATMENT.**—From what has been said, it follows that the most important points in the treatment of gravel are strict limitation as to the *quantity* of food; the avoidance of highly seasoned, very rich, or sweet dishes; the preference for vegetable rather than for animal food; abundant exercise in country air; and abstention from or the very sparing use of alcoholic liquors. Medicinally there may be given diuretics to increase the quantity of the urine, and facilitate the escape of gravel; pure water, alkalis, and alkaline waters freely diluted, to act as solvents of uric acid; and saline aperients and saline waters, to promote digestion, and assist in ensuring the free action of the liver and alimentary canal.

W. CADGE.

**GRAVES'S DISEASE.**—A synonym for exophthalmic goitre, to which the late Dr. Graves, of Dublin, called special attention. *See* EXOPHTHALMIC GOÏTRE.

**GREEN-SICKNESS.**—A popular synonym for chlorosis, applied on account of the greenish colour of the skin sometimes present in that disease. *See* CHLOROSIS.

**GRIPPE** (Fr.)—A French synonym for influenza. *See* INFLUENZA.

**GROWTH, Disorders of.**—*See* ATROPHY; HYPERTROPHY; and MALFORMATIONS.

**GRUTUM.**—SYNON.: *Milium*, a millet seed.

This name is applied to small globular pearly-white bodies seen under the cuticle of the face, especially near the eyelids. They are about the size of a pin's head, and consist of distended sebaceous glands, the duct of which has been occluded. They should

be distinguished from comedones, which are formed in over-distended follicles. When pricked with the point of a lancet, and pressed with a large watch-key, a small, perfectly globular cast of the sebaceous gland is squeezed out, which is quite unlike the long cylinder of the comedo. *See* COMEDONES.

ROBERT LIVEING.

**GUINEA-WORM.**—*See* ENTOZOA.

**GUMBOIL.**—*See* MOUTH, Diseases of.

**GUMMA** (Lat., gum).—A growth occurring in syphilis, so named on account of its supposed superficial resemblance to gum. *See* SYPHILIS.

**GUMS, Diseases of.**—*See* MOUTH, Diseases of.

**GURGLING.**—A physical sign heard on auscultation of the chest or abdomen in certain conditions, due to the movement of gas and fluid within a cavity, whether normal or abnormal. A gurgling sensation may also be felt at times in the intestines, as over the cæcum in typhoid fever. *See* PHYSICAL EXAMINATION.

**GUTTA ROSEA** (*gutta*, a drop; *rosea*, rosy).—A synonym for acne rosacea. *See* ACNE ROSACEA.

**GYMNASTICS** (*γυμνός*, naked).—*See* EXERCISE.

**GYNÆCOLOGY** (*γυνή*, a woman; and *λόγος*, a word).—This term in its literal sense means a doctrine or discourse concerning women. In modern medical language, it comprehends the study of the diseases peculiar to women. *See* WOMEN, Diseases of.

## H

**HABIT OF BODY.**—This expression signifies the sum of the physical qualities of an individual, and is sometimes used as synonymous with *constitution*. We speak of a *full* habit, a *spare* habit, and an *apoplectic* habit.

**HABITS.**—*See* DISEASE, Causes of; and PERSONAL HEALTH.

**HÆMACYTOMETER** (*αἷμα*, blood; *κύτος*, a cell; and *μέτρον*, a measure).—An instrument by the aid of which the number of corpuscles in a given volume of blood can be ascertained.

**DESCRIPTION.**—All methods employed for this object consist in making a definite dilu-

tion of a certain quantity of blood, and counting the number of blood-corpuscles in a certain volume of this dilution. Vierordt, who originated the method, drew uniform lines of diluted blood upon a slide, and, after it was dry, counted the corpuscles in a certain length of line. Cramer substituted for these lines what may be termed a capillary cell; and Potain and Malassez employed a capillary tube, and a microscope provided with an eyepiece ruled in squares. Hayem substituted for the tube a cell, the depth of which gave one dimension of the volume of dilution, while the lines upon the eyepiece furnished the others. The writer's instrument is an adaptation of Hayem's, with cer-

tain modifications: the diluting apparatus is similar, but of different capacity, and the lateral dimensions of the volume of dilution examined are obtained, not by a microscope-eyepiece, but by lines engraved upon the glass slide at the bottom of the cell, on which the corpuscles quickly fall. The instrument can thus be used with any microscope, an important convenience in practical use. The alteration in the capacity of the diluting measures facilitates the counting, and provides a much simpler mode of statement of the result. The apparatus, which is made by Hawksley, consists of (1) a pipette, graduated to 995 cubic millimeters, for measuring the diluting solution; (2) a capillary tube for measuring the blood, containing five cubic mm.; (3) a small glass jar and stirrer for making the dilution; and (4) the cell for counting, .2 mm. deep, and ruled at the bottom in squares, each .1 mm. in length and breadth. The slide bearing the cell is fixed on a small metal plate, to which two springs are attached; these keep the cover-glass in position, and secure uniformity in its pressure on the cell, and therefore in the depth of the contained liquid.

Various solutions have been employed for making the dilution. That which the writer has found to answer best, as differentiating most clearly the red and white corpuscles, consists of sulphate of sodium, 104 grains; acetic acid, 1 drachm; distilled water, 6 ounces.

In using the hæmacytometer, a drop of the dilution is placed in the centre of the cell; the cover-glass and springs are applied; and in a few minutes the corpuscles have sunk to the bottom of the cell, and are seen lying within the squares. The dilution of 5 c.mm. of blood in 995 c.mm. of solution, is 1 in 200; each square contains the corpuscles from a volume of dilution .2 mm. in one, and .1 mm. in each of the other dimensions—that is, 2 cubic .1 mm., or the .002 part of a cubic mm. But the dilution being 1 in 200 this volume of dilution contains just .00001 cm. of blood. The number of corpuscles in a square, multiplied by 100,000, is thus the number in a cubic millimeter of blood—the common mode of statement. In order to limit error, the number of corpuscles in ten squares should be counted, and this number multiplied by 10,000 is the number per cubic millimeter. The average number in health is about 5,000,000. Blood of normal richness thus contains about 50 corpuscles per hæmacytometer square. Therefore, the number in two squares of the instrument will always represent the proportion of the corpuscular richness compared with that of normal blood (= 100)—that is, the percentage proportion to normal. It is, therefore, convenient to take the volume of blood represented by the two squares (.00002 cubic millimeter) as the standard volume, or 'hæmic

unit.' For instance, it is found that the blood diluted presents in ten squares 375 corpuscles or 75 in two squares ('hæmic unit')—that is, 75 per cent. compared with the normal. To learn the number per cubic millimeter we have only to multiply 375 by 10,000 = 3,750,000.

In counting the white corpuscles, if they are not in considerable excess, it is most convenient first to ascertain the number of red corpuscles per square, and note how many squares are contained in a field of the microscope. If, then, the focus is raised, so that the corpuscles become indistinct, the white ones, from their higher refracting power, will appear like bright points, and the number in a series of fields can easily be counted. For example, the number of red corpuscles per square has been found to be 40, and the field contains 15 squares, that is, 600 corpuscles per field. Ten fields contain 15 white corpuscles; the proportion of white to red will, therefore, be 1 to  $\frac{600 \times 10}{15} = 1$  to 400.

With this apparatus we may readily ascertain, within a small limit of unavoidable error, the corpuscular richness of the blood, an important element in many morbid states, such as anæmia; and we can thus ascertain the indications for, and observe the effect of, therapeutic agents. It is, however, very desirable in these cases to ascertain also the richness of the corpuscles in hæmoglobin (*see* HÆMOGLOBINOMETER). Both elements being ascertained and stated in the same terms (percentage), the quality of each corpuscle may be expressed in the form of a fraction. If the blood contains 60 per cent. of corpuscles and 40 of hæmoglobin, the average 'value' of each corpuscle =  $\frac{4}{10}$  or  $\frac{2}{5}$  the normal.

The hæmacytometer may also be employed for ascertaining the globular richness of milk or other liquids.

W. R. GOWERS.

**HÆMATEMESIS** (*αἷμα*, blood; and *ἔμειν*, I vomit).—SYNON.: Fr. *Hématémèse*; Ger. *Blutbrechen*.

**DEFINITION.**—Vomiting of blood, dependent on a variety of morbid conditions.

**ÆTIOLOGY AND PATHOLOGY.**—Hæmorrhage into the stomach may arise: 1. From the laying open of an artery. 2. From venous or capillary congestion of the mucous membrane. 3. From causes affecting the blood itself, so that it tends to transude through the vessels under pressure of the circulation.

1. The most frequent cause of hæmatemesis is an ulcer of the stomach. It occurs, according to the late Dr. Brinton, in about one-third of all the cases of gastric ulcer that come under treatment. The bleeding usually takes place shortly after a meal, and the quantity rejected varies greatly. In some cases, it is so small that it may require careful examination to discover it; whilst in

others enormous quantities are vomited, and often also passed through the bowels. The splenic artery is the source whence the bleeding most frequently proceeds, but it may arise from the coronary, the superior pyloric, or, more rarely, from the blood-vessels of some of the neighbouring organs, such as the pancreas, liver, or spleen, to which the stomach has become attached, and which may form the base of the ulcer. Although it is most apt to occur in chronic cases, instances have been recorded in which a large vessel had been laid open by an ulcer so small as to require careful search for its detection. It must be borne in mind that extensive bleedings may take place without any vomiting, and the source of the fatal illness be overlooked. Such cases are not of infrequent occurrence, and warn the practitioner that he should be on the alert whenever signs of hæmorrhage present themselves, and that he should not rely too much on the absence of pain and vomiting. In cancer of the stomach profuse hæmorrhage is less common than in simple ulcer; the larger vessels being probably compressed by the new-growth, which ordinarily commences in the submucous tissue immediately above them. But a constant oozing of blood is, on the contrary, more common than in simple ulceration. This blood, acted on by the gastric juice, constitutes the 'coffee-ground' vomiting of the older authors. Its occurrence used to be looked upon as pathognomonic of malignant disease, but it is now known that its presence only shows that the bleeding has taken place slowly and in small quantities at a time. Occasionally profuse hæmorrhage takes place from the rupture of an aneurysm into the stomach; and in a case which came under the writer's notice at the London Hospital, fatal vomiting of blood resulted from the perforation of the aorta by a fish-bone that had become impacted in the œsophagus.

2. Congestion of the portal system is a very frequent cause of hæmatemesis. The most marked and fatal cases of this kind occur along with plugging of the vena portæ or its large branches with blood-clots or cancerous matter. Such cases are very rare, and vomiting of blood, from venous congestion, ordinarily results from cirrhosis, chronic congestion, and other diseases of the liver, in which the portal circulation is obstructed, and in which relief is obtained by communication with varicose œsophageal veins.<sup>1</sup> More rarely the like occurrence is observed in persons suffering from diseased heart, especially where there is narrowing of the mitral orifice. In such cases, there is generally a co-existence of chronic catarrhal gastritis, and in all probability the bleeding takes place from the hæmorrhagic erosions

so common in that condition. In one form of this disease enormous quantities of mucus are discharged. Sometimes there is considerable bleeding in these cases, but they are distinguishable from ulcer by the absence of pain; by the vomiting being only occasional; and also by the fact that the blood-stained vomit generally follows a profuse evacuation of colourless mucus, and is always of a dark colour. In females thus affected the catamenial discharge is generally profuse; and the attacks of vomiting do not necessarily coincide with the menstrual periods. It has always been held that hæmatemesis may replace the menstrual discharge. Without denying this, the writer has never met with a well-marked case of the kind. Hæmatemesis due to acute congestion is also a constant result of irritant poisoning.

3. Hæmatemesis also arises from causes affecting the blood, and predisposing it to ooze through the walls of the veins or capillaries. It occurs in this way in purpura, yellow fever, and in some cases of typhus. In jaundice, where bleeding from the gums and other mucous membranes is so often observed, life may be suddenly destroyed by hæmatemesis. Occasionally a hæmorrhagic tendency manifests itself suddenly, without apparent cause, as in a case observed by the writer, in which a woman, about fifty years of age, was affected with severe bleeding from the nose, followed by excessive menstrual discharge, on the cessation of which profuse hæmatemesis took place, from which she sank. She had no jaundice nor other apparent cause for her illness, and after death the most careful scrutiny failed to detect disease in any organ. To this class of causes we should probably refer the hæmatemesis occurring in acute atrophy of the liver, and in pyæmia, as, in all probability, the oozing of blood through the vessels arises from changes effected in its chemical or physical composition.

SYMPTOMS.—Generally the patient in hæmatemesis is suddenly attacked with faintness, accompanied by a feeling of weight at the pit of the stomach; the countenance is pale, the pulse feeble and compressible, and in some cases actual syncope occurs. This state terminates in vomiting, and a greater or less quantity of blood is rejected from the stomach. When a large blood-vessel has been laid open, and the bleeding has taken place rapidly, the blood may be florid; but generally the hæmorrhage goes on so slowly that time is given for the action of the gastric juice upon it, and consequently it is of a dark colour. It is not often that the stomach is completely emptied, or perhaps the bleeding persists in small quantities after the vomiting has ceased, so that the stools are generally of a dark or pitchy character, from the admixture of blood that has passed into

<sup>1</sup> See an instructive communication by Dr. Stacey Wilson, *Brit. Med. Journ.*, vol. ii. 1890.—EDITOR.

the intestines. The hæmorrhage may cease soon after the stomach has been emptied, or the vomiting of blood may recur from time to time, or—and this is very apt to occur in gastric ulcer—months or years may elapse before it again takes place.

**DIAGNOSIS.**—In some cases, when the blood has been slowly effused into the stomach, there may be difficulty in determining whether the dark colour arises from bile or blood. The microscope or spectroscope will be enough to settle this point; or the liquid may be boiled with alcohol, and tested for the biliary salts. It is not always easy to ascertain whether the blood has come from the lungs or from the stomach, as the patient is sometimes so much alarmed that he cannot say whether it was brought up by coughing or vomiting. As a general rule, the blood from the lungs is florid, mixed with mucus, alkaline, and frothy; that from the stomach of darker colour, acid, intermixed with particles of food, and in masses. Again, hæmoptysis is generally preceded by symptoms referable to heart or lungs, such as cough, expectoration, and dyspnœa; hæmatemesis by the symptoms indicative of gastric or hepatic disease, such as those described above.

**PROGNOSIS.**—As a general rule this is favourable in hæmatemesis, more especially in first attacks. Dr. Brinton calculated that death resulted from this cause in only 3 to 5 per cent. of the cases of gastric ulcer; and it is still less frequently fatal where it proceeds from hepatic congestion or cirrhosis. Still, the possibility of the bleeding arising from an obstruction of the portal vein, from the opening of a large artery, or from the bursting of an aneurysm, should be kept in view, and the patient carefully watched.

**TREATMENT.**—Where a large quantity of blood has been ejected from the stomach, the treatment must be prompt and decided. The patient should be maintained in a recumbent posture, and kept perfectly quiet. All food must be forbidden, and pieces of ice placed in the mouth to suck. If faintness be present, it is better not to give brandy, which almost always brings on vomiting, but to apply ammonia to the nostrils; or, if necessary, an enema containing brandy may be given.

The best styptics are gallic acid, alum, and acetate of lead. The gallic acid may be given in 10-grain doses, along with 10 or 15 minims of diluted sulphuric acid, and should be repeated frequently. Alum may be prescribed in infusion of roses; and the acetate of lead in 2-grain doses in the shape of a pill, or combined with acetic acid. Oil of turpentine is also used. Where the bleeding is slight, and there is good reason to believe it arises from portal congestion, the best treatment is to give a small dose of calomel, followed by sulphate of magnesium and

diluted sulphuric acid in infusion of roses every three or four hours, until purging is produced.

For some days after severe hæmatemesis strict quiet should be maintained. In the case of ulcer of the stomach, opium may be used. The diet should be most carefully regulated; if necessary, nutritive enemata should be substituted for food by the mouth, and all purgatives avoided. When portal congestion exists, a free action on the intestinal canal should be commenced after the cessation of the hæmorrhage, so as to diminish the amount of blood in the venous system of the alimentary organs.

S. FENWICK.

**HÆMATHORAX.**—See HÆMATOTHORAX.

**HÆMATIDROSIS** (*αἷμα*, blood; and *ἰδρᾶς*, sweat).—Bloody sweat. See SUDORIPAROUS GLANDS, Disorders of.

**HÆMATIN.**—See HÆMOGLOBIN.

**HÆMATINURIA.**—See HÆMOGLOBINURIA.

**HÆMATOBIUM** (*αἷμα*, the blood; and *βίος*, life).—A synonym for hæmatozoon. See HEMATOZOA.

**HÆMATOCELE** (*αἷμα*, blood; and *κῆλη*, a tumour).—SYNON.: Fr. *Hématocèle*; Ger. *Blutgeschwulst*.

**DEFINITION.**—A swelling occasioned by effusion of blood in the sac of the tunica vaginalis, or in a cyst connected with the testicle.

**ÆTIOLOGY AND SYMPTOMS.**—The extravasation of blood in hæmatocele may take place in a healthy state of the parts, or it may succeed or be combined with hydrocele. In both cases it may be occasioned by a blow, or by violent efforts made in straining, especially in old persons, or when the blood-vessels are diseased. It may happen also from the accidental wound of a vessel in tapping a hydrocele; or, without any such wound, a hæmatocele may supervene if the walls of the sac are very vascular, and a vessel, lacking the pressure of the hydrocele fluid, gives way. The blood effused, if small in quantity, mixes with the fluid of the hydrocele, and occasions slight enlargement without disturbance. If it be large in quantity, coagula are formed; inflammation is excited in the tunica vaginalis; and plastic exudation occurs on its inner surface, sometimes forming layers, and rendering the sac extremely dense and firm.

The testicle preserves the same relation to the remainder of the tumour as in hydrocele, being situated at its posterior part. Its position, however, is liable to similar alterations

as occur in hydrocele, which are very difficult of detection, owing to the great thickening of the parts.

**DIAGNOSIS.**—A hæmatocele may be distinguished from a hydrocele by the absence of transparency; the obscure character of the fluctuation; the heavy feel of the tumour when balanced in the hand; and the sudden and accidental mode of its occurrence. In old chronic cases, in which the tunica vaginalis and its envelopes have become much thickened and indurated, the tumour possesses so firm a character, and feels so heavy and solid, that it is very liable to be mistaken for a chronic enlargement of the testicle; and the diagnosis, at all times difficult, in some instances cannot be satisfactorily made out by the most experienced hands. The records of surgery furnish many cases in which castration has been performed owing to a mistaken diagnosis. When doubt exists, it should be removed by the introduction of a trocar or by an incision before any serious operation, such as castration, is undertaken.

**TREATMENT.**—When hæmatocele succeeds a hydrocele, the blood, if small in quantity, mixes with the fluid of the hydrocele without producing irritation. The tinged fluid may be removed by tapping, and the operation can be repeated afterwards at intervals until the fluid is free from discoloration. Even when inflammation arises, if the sac be tapped and tension removed, and the patient be kept at rest, with ice applied to the part, the inflammation may subside. When, however, the blood effused is large in quantity, and when the inflammation is acute and threatens suppuration, the tumour should be punctured at its upper part, a director introduced, and the sac freely laid open by incision. This must be done with care, so as to avoid wounding the testicle. A chronic hæmatocele with a very thickened sac must be cut into in the same way; and lateral portions of the sac may be excised, so as to lessen the wound for healing. If from old age, or other causes, the patient's general condition be unfavourable for excision, castration may be requisite. The practitioner must bear in mind that the testicle is sometimes situated in front, as in cases of inversion, and is then very liable to injury in the operation of incision, and even in tapping.

**Encysted Hæmatocele.**—Encysted hæmatocele implies an effusion of blood in the sac of an encysted hydrocele. The treatment is the same as that required for ordinary hæmatocele.

**Hæmatocele of the Cord.**—Blood may also be effused in the areolar tissue of the spermatic cord, constituting *diffused* hæmatocele of the cord; or in a cyst in the cord, constituting *encysted* hæmatocele of the cord. Such cases are very rare.

T. B. CURLING.

J. MCCARTHY.

**HÆMATOIDIN.**—See HÆMOGLOBIN.

**HÆMATOMA** (*αἱματώω*, I fill with blood).—A peculiar form of bloody tumour, or a collection of extravasated blood that has undergone certain changes. It is observed more especially in connexion with the ear, the vagina, the scalp, and the meninges. See CEPHALHÆMATOMA; HÆMATOMA AURIS; MENINGES, CEREBRAL, HÆMATOMA OF; FUNGUS HÆMATODES; and TUMOURS.

**HÆMATOMA AURIS** (*αἱματώω*, I fill with blood; *auris*, of the ear).—**SYNON.**: The Insane Ear; Fr. *Othématome*; *Hématome de l'Oreille des Aliénés*; Ger. *Othématoma*; *Ohrblutgeschwulst von Geisteskranken*.

**DEFINITION.**—An affection of the auricle, which occurs almost, if not quite exclusively, in the insane, and consists in the effusion of blood or bloody serum between the cartilage and its perichondrium, to such an extent as to form a distinct tumour.

**ÆTIOLOGY.**—In most of the few cases of hæmatoma auris which have been published to show that this disease may occur in the sane, the description given of the patients rather points to their insanity than otherwise. It is most common in cases of general paralysis and mania (acute and chronic), but also occurs in melancholia, dementia, and idiocy. It is about four times as frequent in men as in women; and more often affects the left ear than the right. Sometimes both ears are affected, but seldom at the same time. There would seem to be, in many or all of the insane, a morbid condition of the vessels or other tissues of the auricle, which predisposes to the occurrence of hæmatoma. If this condition be present to a sufficient degree, the disease may arise spontaneously; in other cases a very slight injury may be sufficient to cause it; whilst in others very considerable violence is necessary for its production.

**SYMPTOMS AND COURSE.**—The disease first makes itself evident by the appearance of a swelling of about the size of a horse-bean; this is almost always upon the anterior surface of the pinna, and usually in the neighbourhood of the fossa of the antihelix. The skin over the tumour is generally of a reddish or bluish-red colour, but may be unaltered at first; the temperature of the ear is sensibly raised; the swelling is very painful and tender; there is no extravasation of blood from the cutaneous vessels; and the tumour is not œdematous. At this stage, the effusion which has taken place between the cartilage and its perichondrium consists of dark-red fluid blood. In rare cases the swelling does not increase further; the inflammatory symptoms subside after about a week; absorption gradually takes place; and only a slight thickening remains. More usually the tumour increases and may attain the size of

a hen's egg; it becomes tense, elastic, distinctly fluctuating, and hot; and is often of a bright red colour. Its prominent anterior wall, consisting of skin, cellular tissue, and perichondrium, is felt to be thinner and less resisting than the posterior, which contains the ear-cartilage. In certain cases, however, owing to the brittle cartilage having split up, and portions of it having adhered to either wall, both walls present irregularly alternating characters. The time which a hæmatoma takes to attain its largest size varies from a week to a month; it then generally involves the whole of the concha, occluding the external auditory meatus; the folds of the auricle are lost, with the exception of the helix (which appears as a band running round the tumour) and the dependent lobule. The weight of the tumour causes the whole ear to fall somewhat forwards and outwards. Sometimes, especially in the presence of constant or repeated irritation, the inflammatory stage may last many weeks, and the deformity which always results from the affection is thereby greatly increased. Unless subjected to violence, it very rarely happens that the tumour opens spontaneously, although its tense and inflamed appearance often seems to indicate that such an occurrence is imminent. If rupture does take place, suppuration ensues; portions of cartilage come away; the cavity closes very slowly; and great deformity results. The most common course is for the inflammatory symptoms gradually to subside. The anterior wall becomes firmer, owing to a new deposit of cartilage upon its inner surface; the sense of fluctuation is gradually lost; and the tumour slowly diminishes in size, often yielding a somewhat doughy sensation to the touch. Occasionally, at this stage, some gaseous contents have been observed in the cavity. The colour of the skin over the tumour becomes gradually more dusky; it then passes into yellow and, later on, into an unnatural pallor. As the fluid contents become absorbed, the tumour becomes harder and smaller; folds again appear in the auricle, but do not correspond to the original ones; and the pinna remains permanently thickened, puckered, and often nodular.

**ANATOMICAL CHARACTERS.**—Many of these have been given above in explanation of symptoms, and do not require to be repeated. A shrivelled auricle, which has previously been affected by hæmatoma, presents, on section, two distinct layers of cartilage; these are of varying thickness, and separated from each other by vascular fibrous tissue, which often contains within it other small isolated plates of cartilage, and sometimes also small portions of bone. The fibrous tissue is the organised product of the original effusion. The two layers of cartilage have been developed upon the inner surfaces of the perichondrium. The loose portions of

cartilage and bone which are occasionally seen, are developed from the fibrous tissue. It used to be supposed that the bone (which is soft, vascular, and contains well-developed Haversian systems) resulted from ossification of the ear-cartilage; but the writer has shown elsewhere (*Brit. Med. Journ.*, Oct. 1873) that this is not the case.

**PROGNOSIS.**—The local affection is in no way dangerous, but it always leaves behind it a permanent characteristic deformity of the auricle. The sense of hearing is only affected by the occlusion of the auditory meatus; but this condition very rarely persists after the acute stage. The occurrence of hæmatoma auris influences the prognosis of the mental disease unfavourably, but does not necessarily indicate the approach of a fatal termination to the case.

**TREATMENT.**—Protection of the part from injury is usually all that is necessary. Cooling applications are useful when the inflammation is excessive. The tumour should not be opened; nor should a portion of the anterior wall be removed, as has been recommended; these procedures only lead to suppuration. It is useless to empty the cavity by aspiration, as it fills again with great rapidity. The treatment by pressure is very painful, and yields no good result.

CHAS. S. W. COBBOLD.

**HÆMATO-PERICARDIUM** (*αἷμα*, blood; *περὶ*, about; and *καρδία*, the heart).—An extravasation of blood into the sac of the pericardium. See PERICARDIUM, Diseases of.

**HÆMATO-THORAX** (*αἷμα*, blood; and *θώραξ*, the chest).—An extravasation of blood into the pleural cavity. See PLEURA, Diseases of.

**HÆMATOZOA** (*αἷμα*, blood; and *ζῶον*, an animal).—This term is of general application to all kinds of animal parasites dwelling in the blood and blood-vessels; but its employment is often restricted to certain of the nematoid entozoa which display this habit in a more marked degree than the other parasites are wont to do. All classes of helminths are liable, at some time or other in the course of their lifetime, to take up their residence in the blood, but in the case of the *Tænia*, or rather of their *proscolicæ*, this period is of very short duration. One or two species only of fluke-worms or trematodes play a similar rôle in man, the most important being the *Bilharzia*, which gives rise to an endemic hæmaturia at the Cape, and elsewhere in Africa (see BILHARZIA). Our knowledge of the nematoid hæmatozoa dates at least as far back as the time of Ruysch (1665), who was acquainted with the strongyles which produce aneurysm in the horse and other solipeds; whilst more than half a century later the subject received additions from the writings of Schulze (1725)

and Chabert (1782); and subsequently from the memoir by Rayer (1843). About the latter period also the observations by Grube and Delafond 'on a verminiferous condition of the blood of dogs, caused by a great number of hæmatozoa of the genus *Filaria*,' excited much attention; but until quite recently it was not so much as suspected that similar microscopic filariæ infested the human body. In 1872 the late Dr. Lewis announced the important discovery of the existence of nematoid worms in the living human subject also. See CHYLURIA; and FILARIA SANGUINIS HOMINIS.

T. S. COBOLD.

**HÆMATURIA** (*αἷμα*, blood; and *οὐρον*, urine).—SYNON.: Fr. *Hématurie*; Ger. *Blut-harnen*.

**DESCRIPTION.**—Hæmaturia is a symptom of many different morbid conditions of the system, and of the urinary tract. The quantity of blood discharged in the urine varies greatly, and the appearance of the urine corresponds. Sometimes it is dark, loaded with clots; sometimes it is merely smoky, or of a faintly pink hue. It is always albuminous, and corpuscles (often altered by the action of the urine) may be discovered by the microscope, sometimes becoming swollen, sometimes shrunken. The following are the best tests for detecting the presence of blood in the urine: (1) *Guaiacum*. When equal parts of simple tincture of guaiacum and oil of turpentine are shaken together to make an emulsion, and the urine is cautiously added, an intense blue colour is produced if blood be present. (2) *Spectrum analysis*. Very minute quantities of blood in the urine show absorption-lines between Fraunhofer's lines D and E in the yellow and green of the spectrum. See SPECTROSCOPE IN MEDICINE.

The blood in hæmaturia may be derived from the urethra. If so, it precedes the stream of urine, sometimes forms a long thin clot, and may escape in the intervals of micturition. Sometimes it is derived from the prostate gland or the bladder. When it has lain in the bladder and been poured out in considerable quantity, it is often in clots; and when the urine is voided, the first part is frequently clear, the last loaded with blood. Blood may also be derived from the ureter or the pelvis of the kidney. Sometimes clots in the form of moulds of these structures may be recognised. At other times the blood is derived from the substance of the kidney, and then is intimately mixed up with the urine, which frequently exhibits blood casts of the tubes.

**ÆTIOLOGY AND PATHOLOGY.**—Urethral hæmorrhage is due to local inflammation or rupture of vessels or to calculus. Prostatic hæmorrhage may be due to tumours, malignant or otherwise, to inflammation, or to scrofulous affection of that organ. Vesical hæmorrhage results from malignant disease,

from simple villous growth, inflammation, ulceration, tubercular disease, or the irritation of a calculus. Hæmorrhage from the ureters or pelvis of the kidney may be due to the presence of calculi, or to other causes. Hæmorrhage from the kidney may be due to malignant disease, tubercle, suppurative nephritis, or to the irritation of crystals or amorphous concretions within the uriniferous tubules. Hæmorrhage occurs also in all the forms of Bright's disease, especially in the early stage of the inflammatory form, and the advanced stage of the cirrhotic. It results from over-doses of turpentine, chlorate of potassium, and cantharides, and from rupture of the kidney. Sometimes it is a manifestation of purpura hæmorrhagica, more rarely of scorbutus; occasionally it occurs in the course of, or as a sequel to, eruptive or continued fevers. It is also occasionally vicarious. Renal hæmorrhage occurs in Egypt, Mauritius, and other localities, in consequence of the existence of a minute parasite, the *Bilharzia hæmatobia*. See ENTOZOA; CHYLURIA.

**TREATMENT.**—The treatment of hæmaturia must vary according to the lesion to which the hæmorrhage is due. But where the symptom is so urgent as to demand treatment for itself, the most important points to be attended to are rest, free relief of the bowels, and the application of ice-bags over the source of the hæmorrhage; along with the internal administration of astringents, especially gallic acid, ergot of rye, perchloride or permanganate of iron, turpentine, or acetate of lead, with or without opium. If these do not succeed, the subcutaneous injection of ergotine is often efficacious. Surgical interference may be required for relief of symptoms due to coagula.

T. GRAINGER STEWART.

**HÆMIC ASTHMA.**—A form of asthma, dependent upon an abnormal condition of the blood. See ASTHMA.

**HÆMIC MURMUR.**—A murmur connected with an abnormal condition of the blood, as in anæmia. See ANÆMIA; and PHYSICAL EXAMINATION.

**HÆMIN.**—See HÆMOGLOBIN.

**HÆMOGLOBIN** (*αἷμα*, blood; and *globus*, a ball).—SYNON.: Hæmatoglobulin.—This substance, the most important pigment in the body, occurs in the red blood-corpuscles, of which, when dried, it forms 90 per cent., this representing 13 to 14 per cent. of ordinary blood. In the corpuscles the hæmoglobin infiltrates the colourless proteid stroma, from which it may be extracted by water, ether, chloroform, or bile salts, thus forming a solution in the plasma; blood so treated is termed 'laky.' It is of an extremely complex nature,

its percentage composition in the dog being estimated at C 53·85, H 7·32, N 16·17, O 21·84, S ·39, F ·43. It is to be regarded as a compound of a proteid substance—globin, and a coloured body—hæmatin, which contains all the iron of the molecule. The former belongs to the globulin group, though its exact nature is not known; and the latter, as a constituent of hæmogoblin, appears to differ slightly from the separated hæmatin presently described. The precise character of the association of these two substances in the compound, whether a simple mixture or a chemical combination, is as yet undetermined.

Hæmoglobin presents a singular exception to the general law of diffusion, inasmuch as, though it crystallises, it will scarcely diffuse through membrane. A considerable variety in the shape of the crystals is met with in different animals, and much difference in the facility with which it crystallises; in man they occur as elongated prisms, and are formed with difficulty.

The most important property of this compound is its affinity for oxygen. In some obscure manner this gas enters into a loose combination with hæmoglobin in the lungs, forming *oxy-hæmoglobin*, and is then conveyed by the red corpuscles throughout the body, separating again from its conveyer in the tissues. The hæmoglobin thus deprived of its oxygen is known as *reduced hæmoglobin*, and is of a purplish colour, whilst the oxy-hæmoglobin is of a scarlet tint. It is thus that the difference in colour between arterial and venous blood is mainly to be accounted for. The spectra of these two kinds of hæmoglobin are characteristic, that of the former being distinguished by two absorption bands of unequal width and intensity between the solar lines D and E (*i.e.* in the yellow and green region of the spectrum), whilst the reduced hæmoglobin presents but a single band occupying a position between the two of the oxidised form. It is to be noted that, whatever be the nature of the combination that exists between the oxygen and its carrier, it is such that the gas retains its properties as a gas, and the union may be roughly compared to a mere solution of the gas in a fluid. There is reason to believe that the oxygen-carrying power is mainly, if not entirely, possessed by the hæmatin constituent, since this substance is capable of being oxidised and reduced in a manner similar to hæmoglobin itself; and, further, this capability is closely dependent on the presence of the iron—iron-free hæmatin having no such power. The 'respiratory pigment' corresponding to hæmoglobin in some of the invertebrata contains copper in place of iron.

Another compound of this substance with oxygen, in which the gas is much more closely combined, is termed *methæmoglobin*; and a still more stable association takes place between hæmoglobin and carbonic oxide, which

thus produces its rapidly poisonous effect by preventing the oxygen-carrying power of the blood. Nitric oxide behaves in a similar fashion.

Certain variations, both quantitative and qualitative, are recognised in disease, but our knowledge in this direction is most fragmentary. In anæmia the amount of hæmoglobin is frequently diminished by a half, and in extreme cases to even less than a quarter of the normal.

The hæmoglobin may be liberated from the stroma of the corpuscles and dissolved in the blood-plasma (*hæmoglobinæmia*), finally appearing in the urine (*hæmoglobinuria*), though chiefly in the form of *methæmoglobin*. This result frequently follows on poisoning by arseniuretted hydrogen, phosphorus, carbolic and some of the mineral acids; also in the course of typhus fever, pyæmia, and scurvy, or after severe burns, and in some cases of jaundice. It is probable that coincident changes in the vascular endothelium favour the diffusion of the pigment.

It is further stated that, in some septic states, pyæmia, cancerum oris, and erysipelas, and also in leukæmia, the blood when drawn from the body shows a remarkable tendency to crystallise, quite contrary to the normal. This has been attributed to the presence in the blood of some peculiar ferment.

The chief derivatives of hæmoglobin may now be briefly referred to.

(a) **Hæmatin** ( $C_{32}H_{32}N_4FeO_4$ ).—Hæmatin may be obtained from red blood-corpuscles by treatment with acids or with strong alkalis.

It is an amorphous dark-brown powder, insoluble in water, alcohol, or ether, but readily soluble in caustic alkalis, in sulphuric or acetic acids, and in chloroform. The acid and alkaline solutions each give a characteristic spectrum. The acid hæmatin is known as hæmatoin. A solution gives a characteristic absorption-band in the spectrum, different from those produced by hæmoglobin. It forms a green solution when boiled with caustic potash.

(b) **Hæmin** ( $C_{32}H_{31}ClN_4FeO_3$ ).—Hæmin, which may be prepared from dried hæmoglobin by treatment with glacial acetic acid, in the presence of an alkaline chloride, is a hydrochloride of hæmatin. It crystallises tolerably readily in needle-shaped prisms or rhombic plates, and thus becomes an easy means of detecting the presence of blood in stains of a doubtful nature. The crystals are insoluble in water, alcohol, chloroform, and ether, and soluble in caustic alkalis and boiling hydrochloric and acetic acids.

(c) **Hæmatoidin** ( $C_{32}H_{36}N_4O_6$ ).—This substance is one of the forms of iron-free hæmatin of natural occurrence as rhombic prisms or needles of a red or greenish-red colour; a fact which shows that the colour of hæmoglobin is not dependent on the iron. It is of considerable pathological interest,

being frequently found in old clots, and in the cavity of ruptured Graafian follicles. It is the cause also of the staining so often seen in the neighbourhood of extravasations of blood, varying from lemon-yellow up to reddish black.

(d) **Hæmatoporphyrin**, also containing no iron, and probably isomeric with the foregoing, may be prepared from hæmatin by heating with fuming hydrochloric acid.

The relationship of hæmoglobin, hæmatin, hæmatoidin, and hæmin to the pigments of the body is of the greatest interest. It would seem that the hæmoglobin is the source of all—biliary, urinary, &c. A constant destruction of red corpuscles is taking place in the spleen, and probably also in the bone-marrow. The hæmoglobin which is liberated from the stroma reaches the liver, where the globin is separated, the iron removed, and bilirubin formed. Normal urobilin, the chief colouring-matter of the urine, is very similar to bilirubin, and is derived from that substance or directly from hæmatin or hæmoglobin itself. The pigments of the bile, fæces, and urine are therefore to be regarded as the excreted waste of hæmoglobin. Bilirubin is closely allied to, if not identical with, hæmatoidin; and a play of colours—the result of oxidation—may be obtained from the latter when treated with nitric acid, similar to that produced by the bile-pigments under the same condition. The injection of hæmoglobin into the blood is followed by the presence of bile-pigments in the urine, and an increase of bilirubin in the bile. Melanin, the black pigment of the retina, hair, and skin, and often found in connexion with new-growths, especially with those of a malignant character, also appears to be directly drawn from hæmoglobin. The colouring-matter of the blood is certainly associated, in some way other than that of its oxygen-carrying function, with the nutrition of the tissues, in connexion with the obscure but unquestionable influence of pigments.

W. H. ALLCHIN.

**HÆMOGLOBINOMETER** (Hæmoglobin; and μέτρον, a measure).

**DEFINITION.**—An instrument for the clinical estimation of the amount of hæmoglobin in blood.

**DESCRIPTION.**—The quantity of hæmoglobin may be ascertained by estimating the amount of iron in the blood, or by observing the dilution necessary to obscure a certain absorption-band in the spectrum (*see* SPECTROSCOPE IN MEDICINE). Neither of these methods is, however, available for clinical use. Simpler methods have therefore been contrived, which depend on a comparison of the colour of diluted blood with that of solutions of carmine and picro-carmine. By this combination the tint of blood, and even its spectrum, may nearly be obtained (Malassez). Coloured discs have been employed

for the same purpose (Hayem). In these methods a given dilution of blood is made, and this is compared with the tint of the standards. In the hæmoglobinometer designed by the writer (and made by Hawksley) the blood is progressively diluted until it reaches the tint of a standard, the colour of which corresponds to a dilution of 1 part of healthy blood in 100 of water. The degree of dilution necessary to make the two correspond represents the amount of hæmoglobin. The apparatus consists of two tubes of exactly equal diameter, and a capillary pipette, holding 20 cubic mm., for measuring the blood. One tube is filled with a standard, consisting of glycerine jelly coloured to the required tint. The other is graduated, each division being equal to the volume of blood taken (20 cubic mm.), so that 100 divisions equal 100 times the volume of blood. The dilution is made by a pipette stopper, and distilled water; the number of degrees of dilution necessary to produce correspondence with the standard indicates the percentage proportion of the hæmoglobin of the blood examined compared with normal blood. For example, the blood of a patient, being progressively diluted, is found to reach the tint of the standard when the amount of water added corresponds to 45 degrees of dilution; the blood examined therefore contains 45 per cent. of the normal quantity of hæmoglobin. The method is no doubt 'rough,' but it is quick and easy. With care there should not be more than 5 per cent. of error. It is well to note not only the point at which the tints become similar, but the number of degrees of dilution through which they appear alike, and to take the mean. The result, being a percentage, is immediately applicable to that obtained with the hæmacytometer. *See* HÆMACYTOMETER.

W. R. GOWERS.

**HÆMOGLOBINURIA** (Hæmoglobin; and οὔρον, the urine).—**SYNON.**: Hæmatinuria.

**DEFINITION.**—A morbid condition of the urine resulting from disintegration of blood-corpuscles, characterised by change of colour of the secretion, in consequence of the presence of granular *débris* containing a large proportion of hæmoglobin; sometimes paroxysmal; sometimes a result of the action of poisons; and sometimes associated with other more dangerous symptoms, and tending to a fatal result.

**ÆTIOLOGY.**—Hæmoglobinuria is not referable to any structural change in the kidneys. It is a result of morbid action in the blood, consisting essentially in the disintegration of the red corpuscles, whereby the hæmoglobin is set free. It has been observed in the course of fevers, particularly ague and scarlatina, also as a result of insolation, and, in the paroxysmal cases, as a result of chill. Outbreaks of this disease have been described

as attacking newly born infants. In that described by Winckel of Dresden, in 1879, twenty-four cases occurred, and twenty-three proved fatal. This was evidently due to the action of a specific poison existing in the Maternity Hospital at the time. Cohnheim also showed that it occurred as a result of severe, but not speedily fatal, burns. Transfusion of the blood of one kind of animal into the veins of another may suffice to produce it. Moreover, many poisons have been found to induce the condition. By far the most important of these is chlorate of potassium. In the case of children, one to two drachms in the twenty-four hours is a dangerous quantity; in the adult, three drachms or upwards. Next to it, but at a long interval, rank carbolic, hydrochloric, sulphuric, and pyrogallic acids, and the inhalation of arseniuretted hydrogen gas. In what way we are to explain the action of these various substances upon the blood-discs is as yet far from clear, but the fact that they do so act is fully established.

**MORBID ANATOMY.**—The kidneys are throughout of a deep chocolate colour, with radiating striæ of a darker tint. The sections show under a low power dark lines corresponding to the tubules, and spots marking the Malpighian bodies. With a higher power one makes out that the tubules are blocked with granular pigment, and that this discharge is evidently from the Malpighian tufts. There are sometimes alterations in the spleen and in the bones, and hæmorrhages into various tissues.

**SYMPTOMS.**—The chief clinical features of hæmoglobinuria are those referable to the urine. It is dark like porter, or like the sediment of port-wine; sometimes brown like coffee, or almost black. This colour is due mainly to hæmoglobin, and usually few, if any, blood-corpuscles are present. Tubercasts and oxalate-of-lime crystals are often present in large numbers. The urine coagulates on heating, or on the addition of nitric acid. Spectroscopically examined, it shows the features characteristic of hæmoglobin, oxyhæmoglobin, and methæmoglobin. Otherwise the patient shows tokens of general discomfort; gastro-intestinal disturbance; jaundice more or less pronounced; sometimes enlargement of spleen, and subcutaneous hæmorrhages; weakness of heart; tendency to hypostatic congestion in cases of a bad type; with other symptoms of depression or even collapse.

**DIAGNOSIS.**—The different forms of hæmoglobinuria have to be diagnosed from each other, and from certain diseases which resemble the paroxysmal forms. See HÆMOGLOBINURIA, PAROXYSMAL.

**PROGNOSIS.**—Cases of this kind rarely terminate unfavourably except when hæmoglobinuria is merely one of many symptoms in connexion with blood-poisoning.

**TREATMENT.**—Protection against septic processes or other causes is of the first importance. When the symptom is developed, ferruginous tonics and chloride of ammonium may be tried. T. GRAINGER STEWART.

### HÆMOGLOBINURIA, PAROXYSMAL.—SYNON.: Paroxysmal Hæmatinuria.

**DEFINITION.**—A paroxysmal affection of the system; manifesting itself by changes in the urine, and particularly the discharge of hæmoglobin; caused sometimes by malaria, and sometimes by other conditions not yet determined; consisting in no anatomical change as yet recognised; and characterised by the occasional occurrence of constitutional disturbance, with discharge of dark, blood-stained urine.

**ÆTIOLOGY.**—The most important extrinsic cause of the tendency to this disease is malarious poison; the most important cause of the paroxysm is exposure to cold or wet; but the tendency may exist without malarial poison, and the attack may occur apart from any special exposure. Amongst intrinsic causes, sex is evidently important, for the disease is almost confined to males. It may occur in children, and may occasionally recur during a period of years.

As in other forms of hæmoglobinuria, the symptom is traceable to disintegration of red corpuscles within the circulating fluid. That this may occur locally has been proved experimentally by Ehrlich. He tied a ligature round the finger of a patient who was liable to the disease, and then chilled the congested part by immersing it for a quarter of an hour in ice-cold water, afterwards transferring it for a like period to tepid water. In this patient it was found that many corpuscles were broken down, while no such effect was produced elsewhere in the body or in healthy people. It is probable, however, that it is chiefly in the liver that the destruction of corpuscles ordinarily takes place; and it is uncertain whether it results from a direct influence operating upon the corpuscles, or through the production of some chemical substance and its action upon them.

**ANATOMICAL CHARACTERS.**—The disease not being fatal, there is no evidence as to the existence of any anatomical change in the kidneys, but there is every reason to suppose that if the kidneys were examined they would show the appearances met with in non-paroxysmal hæmoglobinuria.

**SYMPTOMS.**—This form of hæmoglobinuria is paroxysmal, but not distinctly periodic. It may commence in childhood or during adult life. The attacks may occur once, twice, or thrice a day, on alternate days, once a week, or quite irregularly. The paroxysm may commence abruptly without any premonitory symptom, but is more commonly ushered in by a feeling of uneasiness in the loins and

limbs, by shivering, and general chilliness. Sometimes it is preceded by slight jaundice, furred tongue, and other symptoms of gastric catarrh; and sometimes albuminuria precedes by a few hours or a day the occurrence of hæmoglobinuria. The more abrupt attacks frequently terminate by the discharge of the peculiar urine, the uneasy feelings having subsided before it recurs; and the next urine is normal, or nearly so. In some cases albuminuria precedes or lingers for a time after the discoloration has passed off, and sometimes it replaces the hæmoglobinuria. See BRIGHT'S DISEASE.

The characters of the urine are very peculiar. Its colour is like porter, or like muddy port-wine; its specific gravity ranges from 1015 to 1035; it is acid, or faintly alkaline; it is highly albuminous; sometimes it contains excess of urea. It throws down a copious sediment, which contains very few or no blood-corpuscles, but an immense amount of granular blood-pigment, with numerous tube-casts—hyaline or epithelial, often loaded or coated with amorphous granular matter, and with minute crystals of oxalate of lime. The colour is not due to blood-corpuscles, nor to hæmatin, but to hæmoglobin. In some cases the urine is less affected, being merely albuminous, and not depositing pigment. It may be doubted whether this condition should be admitted to the same category as the disease under discussion, but cases which have come under the writer's observation seem to show that it is entitled so to rank, and it is dealt with in the article just referred to.

A very interesting point in the clinical history of hæmoglobinuria is its relationship to Raynaud's disease. The relationship is neither constant nor exclusive, and the precise link which connects them is as yet not clearly determined. Still the clinical fact remains, that when the one disease exists the other is not infrequently present.

DIAGNOSIS.—The only diseases with which intermittent hæmoglobinuria is likely to be confounded are hæmaturia, and renal calculus or gravel. From the former it is distinguished by the abundance of the blood-pigment and the extreme rarity of blood-corpuscles; from the latter, by the short duration of the attacks, the presence of the characteristic deposit, with the fact that the pains affect both loins, not merely one. It is sometimes important to distinguish the milder forms, in which merely albuminuria occurs, from congestion or from commencing inflammatory Bright's disease. It is not always possible to distinguish these during the early hours of the attack; but the amount of general disturbance, the state of the tongue, the slight jaundice, the suddenness of the onset, and the absence of dropsy, generally suffice to make the diagnosis clear.

PROGNOSIS.—The prognosis is good in

paroxysmal hæmoglobinuria, as to the individual paroxysm. The tendency to the disease is also not infrequently got rid of. It has not proved fatal in any case. But it appears sometimes to usher in, or to constitute an early symptom of, Bright's disease—the cirrhotic form.

TREATMENT.—With a view to preventing the attacks, individuals who are subject to paroxysmal hæmoglobinuria should carefully avoid exposure to cold and over-fatigue of every kind. They should take abundance of good and easily digested food, should avoid free use of alcohol, and should clothe warmly. As the paroxysm is spontaneously recovered from, little need be done, excepting with the view of alleviating the discomfort of the patient. He should go to bed and be kept warm, and have abundance of warm drinks. In respect of diminishing or removing the tendency to the malady, various remedies have been found useful, among which may be mentioned quinine, tincture of cinchona, iron, arsenic, and chloride of ammonium. Antisyphilitic treatment by means of mercury has also been found useful.

T. GRAINGER STEWART.

HÆMOPERICARDIUM.—See HÆMATO-PERICARDIUM.

HÆMOPHILIA (*αἷμα*, blood; and *φιλία*, predisposition for).—SYNON.: Hæmorrhaphilia; Hæmorrhagic Diathesis; Bleeders; Fr. *Hémophilie*; Ger. *Bluterkrankheit*.

DEFINITION.—A congenital disease, often hereditary, characterised by a tendency to immoderate bleedings, whether spontaneous or traumatic, and to obstinate swellings of the joints.

ÆTIOLOGY.—Men are far more liable than women to this disease; the proportion being about eleven to one. Women who suffer from hæmophilia show much less typical specimens of the disease than men, and rarely die from hæmorrhage, although floodings and profuse menstruation are common.

The best-ascertained cause of hæmophilia is hereditary predisposition. No other cause is known with anything like certainty. In a bleeder family, the disease descends to the boys through the mothers, the women remaining quite healthy and apparently free from all disease. The fathers do not seem to transmit the disease to their sons; at least, instances of this are rare. The women of bleeder families are remarkably fertile. Some have thought hæmophilia to be more common in Germany, but this is probably owing to the greater attention paid to the disease in that country. Cases have been met with in the Indian Archipelago, North America, the Scandinavian kingdoms, and elsewhere. The disease is not limited to the Aryan races, as the Jews, a Semitic nation, are singularly liable to it.

**ANATOMICAL CHARACTERS.**—No morbid appearances have yet been found after death with any constancy. The blood-vessels are apparently unaltered. The condition of the blood has not yet been properly investigated. It would be important to ascertain whether the number of white corpuscles is increased or diminished, and whether they are altered in character. The swellings of the joints appear to be due to the extravasation of blood within the articulation.

**SYMPTOMS.**—The first signs of hæmophilia are commonly seen during the first year of life; but sometimes they are delayed until the beginning of the second dentition. Cases on record of a much later appearance of the first symptoms are not trustworthy. It is very rare for bleedings to be noticed at birth. There is nothing about the subjects of hæmophilia, when not suffering from bleeding, to distinguish them from ordinary persons. They look well; and nothing amiss can be discovered by physical examination in chest or belly.

There are three well-marked *degrees* of hæmophilia. The first is the most typical and characteristic, in which there is a tendency to every kind of hæmorrhage, traumatic or spontaneous, interstitial or superficial. The swelling of the joints is well-marked. This degree is scarcely ever seen in women; but it is the most common among men. In the second degree, spontaneous hæmorrhages from the mucous membranes only are present. The third degree, in which the tendency is little marked, is seen only amongst the women of bleeder families; and shows itself only by spontaneous ecchymoses.

**Hæmorrhage.**—Spontaneous bleedings are sometimes preceded by symptoms of unusual fulness and plethora. The mucous membranes supply the blood in this case; in childhood bleeding from the nose is the most common, and also the most fatal, although bleedings from the bowel, mouth, or chest may also occur. There is only one known instance of death from hæmaturia. The traumatic bleedings vary much in intensity, even in the same individual. Death has followed division of the frænum of the tongue, vaccination, leeching, and the extraction of a tooth. This last is a very common cause of death. If abscesses be opened, furious bleeding commonly takes place; and the same occurs if a blood-tumour or extravasation of blood be interfered with.

The bleeding is nearly always capillary, and may kill in a few hours or after some weeks. The quantity of blood lost is sometimes enormous. After the bleeding the patients are extremely anæmic; and this state may last for months.

Besides superficial bleedings, interstitial hæmorrhages, ecchymoses, and blood-tumours may be observed, whether spontaneous or traumatic. A bruise which a healthy person

would not feel may fill the connective tissue of a limb with blood; or the bleeding may be circumscribed, and form a tumour instead.

**Swelling of the joints.**—This chiefly affects the larger joints, the knee being most commonly attacked. The joint, most commonly after some injury, becomes swollen and painful, and apparently filled with fluid; there is fever; and this state may last for many weeks. Relapses are very apt to occur during convalescence.

**DIAGNOSIS.**—The diagnosis of hæmophilia is often easy. If a boy have suffered repeatedly from early infancy from abundant bleedings—especially traumatic—and from joint-affections, there can be no doubt of the diagnosis. It is made more certain by the existence of hereditary predisposition. In women the diagnosis must be made with more care, as they are subject to a hæmorrhagic disorder which first appears about puberty, but which is not hereditary.

**PROGNOSIS.**—The prognosis is not so serious with respect to life as was formerly thought.

**TREATMENT.**—The treatment of hæmophilia includes almost the whole subject of the treatment of hæmorrhage. Surgical interference in the way of the ligation of arteries is, however, contra-indicated in connexion with hæmophilia, as the hæmorrhage in such cases is almost exclusively capillary. For the same reason, the administration of ergot, with a view to bringing about a contraction of the arterioles, would not be likely to prove of service. Treatment ought to be directed entirely to bringing about an increase in (a) the rapidity of coagulation, and (b) in the firmness of the resulting clot. In our present ignorance on the subject of the pathology of hæmophilia, treatment must take cognisance of every factor which is known to influence the coagulability of the blood. The facts that have been ascertained in connexion with this subject are as follows: A defect in coagulability may depend on one or other of the following conditions:—

**A Defect in the Inorganic Substratum of Fibrin.**—This may consist in a diminution of the amount of available calcium salts in the blood. Defective coagulation arising from this cause is to be met by the administration of lime-salts, preferably calcium chloride (liquor calcii chloridi B.P., 1 oz. to 1½ oz. for an adult; in ½ to 1 pint of water). Owing to the disagreeable taste of the salt, it is best administered in the form of an enema. A 1 per cent. solution of calcium chloride may also be applied as a local styptic. Stronger solutions are not to be recommended, as coagulation is retarded after a certain percentage of calcium has been exceeded. In view of the fact that the action of the lime-salt is not instantaneous, the solution must be applied on a tampon in such a manner as to hinder the flowing away of the blood with which it

comes in contact. In all cases the clots obtained by the addition of lime to the blood are characterised by an extraordinary firmness. The coagulative efficacy of a solution of lime-salts can be increased by the addition of fibrin ferment. This last can be obtained by extracting washed fibrin with water.

*A Defect in the Organic Substratum of Fibrin.*—There is little doubt that the coagulation of the blood is brought about by the combination of an element of plasma-fibrinogen with a fibrinoplastic element derived from the breaking down of the white corpuscles. This fibrinoplastic element consists of the substance which is known as Woolldridge's tissue- or cell-fibrinogen. A diminution of the available tissue-fibrinogen in the blood leads to a condition of diminished coagulability. Its addition to the blood in all cases is followed by an immediate increase of coagulability, and an increased formation of fibrin. In view of this fact a local application of tissue-fibrinogen would appear to be indicated when hæmorrhage in hæmophilia does not yield to the application of lime, or lime and fibrin-ferment. Tissue-fibrinogen may be obtained in the following manner: Procure the thymus gland (the chest-sweetbread) or testicle of a calf, lamb, or other animal from the butcher's. Mince it fine in a mincing-machine, and extract with, say, twenty times its volume of water for ten minutes to twenty-four hours. Strain through fine muslin or through flannel, and add to each pint of the strained infusion 1 fluid ounce of diluted or 1 fluid drachm of strong acetic acid (B.P.). A precipitate of tissue-fibrinogen forms almost immediately. Allow it to settle, and get rid of the supernatant fluid by decantation, or, better, collect the precipitate on a filter-paper. In either case dissolve the tissue-fibrinogen thus obtained by the addition of a few drops of alkali (dilute carbonate or bicarbonate of sodium). Apply the somewhat viscid solution on a tampon of cotton-wool to the bleeding point. Escharotics such as ferric chloride are seldom found to be of any service in bleeding in hæmophilia.

In cases of severe hæmorrhage, transfusion may be necessary.

**GENERAL TREATMENT OF THE MORBID CONDITION.**—It is impossible in our present ignorance of the pathology of hæmophilia to lay down a scientific method of treatment. It will, however, be evident that any treatment directed towards rendering the blood more coagulable would be indicated. Thus the administration of lime-salts might prove useful. Further, the means which are at our disposal for increasing the number of white corpuscles in the blood might be employed, with a view of increasing the amount of the fibrinoplastic element, for we have seen that tissue- or cell-fibrinogen is contained in the white corpuscles. While awaiting

further information on this subject, the following therapeutic agents might be brought into requisition: (a) The administration of aromatic substances, such as camphor and turpentine. The administration of these substances increases the number of white corpuscles in the blood (Binz), and they are employed apparently with success in the treatment of internal hæmorrhages. (b) The administration of pilocarpine (Horbaczewski), which increases the number of circulating white corpuscles. (c) The administration of nuclein (Horbaczewski), or nuclein containing substances such as tissue-fibrinogen (Wright), for these substances increase the number of white corpuscles in the blood. They are available in the form of yeast, sweetbreads, lamb's fry, and kidneys. (?) It has, however, not yet been ascertained whether these cell-substances lose their efficacy on boiling.

ALMROTH E. WRIGHT.

**HÆMOPHTYSIS** (*αἷμα*, blood; and *πύρωσις*, I spit).—SYNON.: Pneumonorrhagia; Bronchorrhagia; Fr. *Hæmoptysie*; Ger. *Bluthusten*.

**DEFINITION.**—Blood-spitting having its source in pulmonary or bronchial hæmorrhage.

The restriction of the term 'hæmoptysis,' as thus defined, has the sanction of long usage and convenience. Hæmorrhage from an aneurysm opening through the lung or air-passages would not strictly be included in such a definition.

Spitting of blood, when it arises from other and less important sources, comes under the denomination of *false* or *spurious* hæmoptysis. For *fictitious hæmoptysis*, see FEIGNED DISEASES.

**ÆTIOLGY AND PATHOLOGY.**—The causes of hæmoptysis in the widest sense of the term, and having regard to its pathology, may be thus enumerated:—

**1. Hæmorrhage from the pulmonary artery or its radicles.**

1. Rupture or wound of the lung from external violence.

2. Active hyperæmia of the lungs—inflammatory, vicarious, or induced by violent effort or excitement. The active hyperæmia may be primary as regards the lungs; or may supervene or be attendant upon disease already present in them. See LUNGS, Hæmorrhage into; and LUNGS, Inflammation of.

3. Mechanical hyperæmia of the lungs, secondary to heart-disease, or embolism of one of the pulmonary branches, or to pressure from tumours, such as enlarged bronchial glands. See LUNGS, Hæmorrhage into; BRONCHIAL GLANDS, Diseases of.

4. Necrotic division of vessels in the course of softening of tubercular or other consolidations in destructive lung-diseases—phthisis, tuberculosis, cancer.

5. Aneurysmal dilatation or simple erosion.

of branches of the pulmonary artery, exposed in the course of excavation of the lung, or ulceration of the bronchial mucous membrane.

6. Primary atheroma of the pulmonary artery within the lung.

II. **Hæmorrhage from the bronchial capillaries.**—Capillary hæmorrhage from the bronchial mucous membrane.

III. **Hæmorrhage from the aorta, or one of its great branches.**—Aneurysm rupturing through the lung or into a bronchus.

Details respecting the pathology of these several forms of hæmoptysis will be found under the headings of the principal diseases giving rise to them. There are a few additional remarks, however, that should be introduced here.

The pathology of hæmoptysis occurring in early phthisis—of which it is one of the most frequent symptoms—is still somewhat obscure. Besides the active hyperæmia above referred to—that is, the inflammatory congestion that constitutes the first stage of some forms of phthisis, and tends to recur at different periods of the disease; besides also the necrotic division of vessels, or their aneurysmal dilatation, which more especially account for the hæmorrhage occurring in the later stages of the disease,—there are other conditions present in early phthisis which probably have much to do with the occurrence of hæmoptysis. The minute blood-vessels are importantly concerned in the very earliest stage of phthisical lung-disease in one or other of three ways:—

(a) Their walls become the seat of nuclear proliferation, and hence become softened; and (b) these vessels become more or less extensively blocked, not merely as the result of the inflammatory *stasis* which may affect their capillaries, but by the pressure of surrounding tubercular growth, or by tubercular thickening of their walls intruding upon their calibre. (c) It is very possible that in the ill-developed lungs of small-chested people, who inherit a tendency to consumption, the vessels are also morbidly frail, and are apt to give way in any temporary hyperæmia. Some persons are remarkably subject to irregular distribution of blood; they are liable to chills, cold extremities, and transient flushings; and a *pulmonary blush* is as conceivable as a temporary flush elsewhere, and would favour the occurrence of hæmorrhage.

It has been alleged that the hæmoptysis occurring in all stages of phthisis frequently has its source in hæmorrhage from the bronchial tubes, and that this bronchial hæmorrhage may give rise secondarily to phthisis, or may, when it occurs in the course of that disease, set up fresh centres of mischief in the lungs by inhalation of blood to distant portions. In the entire absence of

reliable pathological grounds for this view, and in the abundance of anatomical evidence, clinical experience, and weight of authority in favour of the pulmonary origin of hæmoptysis, we are, in the opinion of the writer, justified in believing that decided hæmoptysis originating in hæmorrhage from the bronchial mucous membrane is exceedingly rare, excluding, perhaps, syphilitic ulceration of the bronchi. Blood inhaled to distant portions of lung may, however, as shown by Dr. Reginald Thompson, undergo changes resulting in fresh pulmonary destruction.

**DESCRIPTION.**—The quantity of blood brought up in hæmoptysis varies from a mere streak to two or three quarts. When blood is expectorated in large quantity, it is pure and unmixed; and either dark and venous, or bright arterial. The first and last portions are usually more or less aerated. If in small quantity, the blood is most generally bright and frothy, it may be only a speck or two upon the sputum, or it may be in several mouthfuls of pure aerated blood. It commonly happens that this mitigated and more characteristic hæmoptysis precedes the rarer attack of profuse hæmorrhage that may prove instantly fatal. Hæmoptysis is sometimes scanty, dark, and clotted, usually from a small portion of blood having been detained in the lung before being expectorated.

In decided hæmoptysis the shock to the system is always great. The patient is alarmed and anxious, especially on the first attack. The sense of weakness and prostration is, indeed, often prolonged after the attack—not necessarily a severe one as regards quantity of blood lost—has ceased. The face is often flushed; the extremities cold. The temperature is usually depressed; after a few hours it becomes normal, and it may continue so, or it may rise in the course of forty-eight hours or within five days. This elevation of temperature may depend (a) upon return of the previous fever after being temporarily checked by hæmorrhage and shock; (b) upon the cause which has also produced the hæmoptysis; or (c) upon the secondary consequences of the hæmorrhage. It has been clearly shown that the inhalation of blood into the bronchial tubes and pulmonary alveoli sometimes sets up broncho-pneumonia, and thus may give rise to fresh centres of phthisical disease; and cases do occur in which acute miliary tuberculosis of the lungs supervenes upon an hæmoptysis in a manner suggestive of direct infection. The later the rise of temperature up to the fifth day, the more reason we have for regarding it as due to secondary pneumonia.

In the hæmoptysis that occasionally arises in some forms of heart-disease (chiefly mitral stenosis), the amount of blood expelled is usually small, darker in colour, less aerated than in ordinary hæmoptysis, and partially

clotted. This form of hæmoptysis is, moreover, associated with the signs of heart-disease of more or less old standing. See LUNGS, Hæmorrhage into.

**ANATOMICAL CHARACTERS.**—These depend very much upon the nature of the disease which has preceded the hæmoptysis. In cases of death from hæmoptysis there are the usual appearances of death from hæmorrhage—pallor of organs, empty and contracted ventricles, &c. The bronchial tubes of both lungs are found to contain clots. The healthy portions of lung are found inflated from obstructions in the bronchi, impeding the exit of air; they are generally pale, but beautifully speckled by pink spots, marking the lobules into the air-cells of which blood has been inhaled. If death take place several days after profuse hæmoptysis, any blood which remains in the bronchi is dark and disorganised, and patches of consolidation may sometimes be found, having for their centres the stained appearance attributable to inhaled blood. More or less bronchitis is also noticeable. It is rare to find fatal hæmoptysis without the presence of cavities, which are also, more or less, filled with blood-clot. In almost every instance of fatal hæmoptysis in phthisis, sufficient diligence will discover either an aneurysm of a pulmonary vessel within a cavity, or ulcerative erosion or laceration of an exposed vessel. The writer has found such a condition in fifteen cases at various ages, including one infant seven months old.

**SIGNIFICANCE OF HÆMOPTYSIS.**—No modification in our views respecting the nature of phthisis can lessen the significance of hæmoptysis as being one of its most important positive signs. Thus regarded, it is a warning that may sometimes save, and very often prolong life, by drawing our early attention to a condition that might otherwise remain too long concealed; but, lightly considered and carelessly treated, it is but the precursor of destructive disease.

**DIAGNOSIS.**—Genuine hæmoptysis can rarely be mistaken by a skilled observer present at the attack. The gurgling in the bronchi, the loose cough, and repeated expectoration of *bright frothy* blood are quite characteristic. The blood is distinctly expectorated with cough, not vomited; and its quality is distinctly fresh, not changed. In hæmatemesis, with which hæmoptysis is sometimes confounded, the blood is brought up by vomiting; is more or less mixed with the contents of the stomach; and, save when the hæmorrhage is very rapid and abundant, presents a dark grumous appearance, owing to the action of the gastric fluid upon it (see HÆMATEMESIS). It may, too, be observed that the blood does not come from the nose, unless a small quantity be projected through the nostrils with the spasmodic cough. If, as often happens, no medical attendant is at

hand at the moment of the attack, the appearance of the blood is usually sufficient for diagnosis; and if the hæmoptysis have been at all copious, any further expectorations for the next few hours are sanguineous. The fact of this sanguineous coloration of the sputa having existed for some hours, or a day or two after an hæmoptysis, is positive evidence of the hæmoptysis having been genuine. In cases of *very copious* hæmorrhage from a large pulmonary branch, the blood brought up is dark and venous.

It is very important in investigating the cause of hæmoptysis to be as gentle as possible in physical examination. We can listen to the lungs in front and to the heart without moving the patient or requiring him to breathe deeply; we can make a note of the temperature; and at the time we should do no more.

The throat should always be examined in doubtful cases of hæmoptysis, as the blood will sometimes be found to have issued from an ulcerated tonsil, or even from enlarged vessels on the posterior wall of the pharynx. *Spurious hæmoptysis*, however, may be defined as *the escape of a blood-stained mucus from the throat or gums*. In cases of spurious hæmoptysis there is usually distinct evidence of a morbid condition of these parts. The mucous membrane of the fauces is relaxed; the gums are spongy, and often bleed when the teeth are brushed in the morning. Sometimes this condition of gums arises from insufficient attention to the teeth, carious stumps and much tartar round the teeth causing irritation and sponginess of the gums. The patients are usually anæmic and short-breathed, and often complain of morning cough, but there is no evidence of lung-disease. The blood-stained mucus is usually ejected in the morning, on waking; and often escapes from the mouth during the night, staining the pillow. On examination, it is found to be a pink, watery mucus, uniformly stained, and containing comparatively few blood-corpuscles.

**TREATMENT.**—Absolute rest is the first thing to be observed in the treatment of all attacks of hæmoptysis. The patient should lie down with the head and shoulders raised by pillows. He should not talk. The room should be kept quiet and cool; the bedclothes should be light, but sufficient; and warmth should be applied to the feet. A little ice in the mouth, or some iced water to sip, will ease the cough and tend to check hæmorrhage. The patient must, if possible, be reassured as to the absence of immediate danger, and the shock to the system allayed without the use of stimulants. Sometimes opium may be usefully given for this purpose, due regard being had to the habits and idiosyncrasies of the patient. In copious hæmorrhage, attended with shock, a small subcutaneous injection of morphine is some-

times of great value. Astringent medicines are not always needed in hæmoptysis, especially in attacks in which the hæmorrhage is presumably capillary; they may, however, be given in such cases in small doses. To be really useful in hæmoptysis, astringents must be given in full doses. Those most used are acetate of lead, four grains, every three or four hours; alum, twenty grains, with diluted sulphuric acid, thirty minims, every four hours; gallic acid, twenty to thirty grains every half-hour or hour, for two or three doses, followed by ten-grain doses every three hours; oil of turpentine, thirty minims every two hours in sweetened mucilage or in milk, for a couple of doses, then in half or third doses; liquid extract of ergot in half-drachm or drachm doses, every two or three hours; ten- or fifteen-minim doses of the permittate or persulphate of iron solution, or thirty or forty minims of the perchloride freely diluted. Of the above-mentioned astringent remedies, alum and sulphuric acid, gallic acid, and ergot are the best in the greater number of cases. Acetate of lead is less applicable to cases of lung-hæmorrhage than in bowel or kidney lesions. Ergot is to be preferred in those cases in which we infer that a considerable vessel has given way. It may also be given in the form of ergotin—one-half to two grains hypodermically; but thus given it sometimes causes much local inflammation. Oil of turpentine is certainly one of the most powerful of astringents in pulmonary hæmorrhage, and may be usefully held in reserve. When freely administered, the condition of the urine must be carefully noted. Cases of hæmoptysis only rarely occur in which it is well to give the iron astringents. As a rule, they tend to increase pulmonary congestion; but in some cases, in which hæmoptysis has a tendency to continue after the patient has been brought to a state of profound anæmia from the first outburst, they may be usefully given, their effect being carefully watched. Digitalis is a drug sometimes of great value in hæmoptysis; it is best adapted to those cases in which there is much excitement of the circulation. In cases, for instance, in which the hæmoptysis has been determined by intemperance, or in full-blooded people by effort, tincture of digitalis in twenty- or thirty-minim doses may be given. In such cases, in which the portal system is usually congested, sulphate of magnesium and diluted sulphuric acid may be combined with the digitalis, and given every four or six hours for a couple of days or so. In the treatment of hæmoptysis it is usually necessary to counteract the effect of the astringent upon the bowels by purgatives or laxatives. An enema may be necessary to enable the bowels to act easily and without straining. The use of the bed-pan must of course be enjoined.

Counter-irritation is of great value in the

treatment of many cases of hæmoptysis, chiefly in those cases in which the hæmorrhage is not great, and occurs in the course of phthisis, being due either to local pulmonary congestion or to active hyperæmia of the walls of a cavity. Blisters are, as a rule, best avoided in copious hæmoptysis during the stage of shock. Continuous cold to the chest is decidedly to be deprecated. The intermittent application of cold may sometimes be employed, but is a measure of doubtful expediency.

The temperature and pulse of the patient should be carefully watched during an attack of hæmoptysis, and for days afterwards.

The diet must at first be restricted to cold nutritious fluids. The tendency is to keep patients suffering from hæmoptysis on cold slops and cold solids for an unnecessary time. In most cases, after forty-eight hours, warm, not hot, food may be allowed. Stimulants, except in special cases, must be interdicted.

The treatment of *false* or *spurious hæmoptysis* depends upon its cause. In most cases, the cause being anæmia, with a relaxed and morbid condition of the mucous membranes, an acid preparation of iron containing some chlorate of potassium and glycerine will speedily cure the malady. Tincture of the acetate or perchloride of iron, added to a small 'lemon-squash,' is also an excellent remedy. When the gums are spongy, the addition to a chalk or charcoal tooth-powder of finely powdered kino or catechu, or the addition of some glycerine of tannin to the tooth-water, will prove efficacious. Astringent gargles will suggest themselves in fitting cases; and fruit and vegetables must be added to the diet.

R. DOUGLAS POWELL.

**HÆMORRHAGE** (*αἵμα*, blood; and *ῥήγνυμι*, I burst forth).—SYNON.: Fr. *Hémorrhagie*; Ger. *Blutfluss*.

**DEFINITION.**—The escape of blood from any part of the circulation, and its discharge from the body.

When blood escapes from any of its natural reservoirs, it is either extravasated into the neighbouring organs or cavities, or flows from one of the surfaces or orifices of the body. In the latter case only is the term 'hæmorrhage' strictly applicable (see EXTRAVASATION). This distinction, however, is not always carefully observed; and such expressions as 'cerebral hæmorrhage,' 'hæmorrhage into the pericardium,' and 'hæmorrhagic eruptions' are employed in connexion with what are more correctly called *extravasations* of blood.

**ÆTIOLGY.**—Hæmorrhage is almost always due to a solution of continuity of some part of the circulatory system, whether by injury or by disease. Hæmorrhage from the external surface of the body is, with few exceptions, the result of wounds or other form of injury; but ulceration—either simple or

malignant—frequently lays open a vein or an artery; and aneurysm of the great vessels, and even of the heart, may also give rise to external bleeding. Hæmorrhage from internal organs, on the contrary, is most frequently the result of disease, as, for instance, ulceration or aneurysm, or of circulatory disturbance, such as congestion. Great excitement and severe exertion are two of the chief determining causes of hæmorrhage, by producing great or sudden rise of the general blood-pressure, whether by cardiac disturbance or by obstruction of the terminal vessels; they may lead even to the rupture of healthy vessels. Local disturbances of the blood-pressure give rise in the same way to congestion or hyperæmia, and finally to hæmorrhage. The principal causes of hæmorrhages of this class are diseases of the heart, of the great vessels, of the respiratory organs, and of the liver, and sudden variations in the atmospheric pressure or in the temperature; and such hæmorrhages are also in some instances undoubtedly of a *vicarious* nature. Certain conditions of the blood are believed to predispose to hæmorrhage, as, for example, in scurvy, purpura, the malignant fevers, chronic Bright's disease, and alcoholism. There is, probably, however, a lesion of the vessel-walls in some of these diseases. A peculiar condition of body is known as the *hæmorrhagic diathesis* or hæmophilia; the subjects of which exhibit, amongst other forms of debility, a remarkable tendency to profuse hæmorrhage from trivial causes. See HÆMOPHILIA.

SEATS AND VARIETIES.—Bleeding from the surface of the body, whatever its origin, is simply called *hæmorrhage*, and its exact source and situation are otherwise defined. On the contrary, when blood escapes by any of the natural openings of the body, and is derived from an internal organ, the hæmorrhage is described by a special name. Hæmorrhage from the nose is called *epistaxis*; from the ear *otorrhagia*; from the stomach *hæmatemesis*; from the respiratory tract *hæmoptysis*; and from the urinary tract *hæmaturia*. Blood passed *per anum*, after undergoing certain changes to be referred to immediately, is called *melæna*. *Menorrhagia* and *metrorrhagia* are the names given to profuse bleeding from the genital organs in the female—at the menstrual period and at other times, respectively.

DESCRIPTION.—The local phenomena which characterise hæmorrhage from the external parts of the body, vary with the source of the blood. If it proceed from an artery of considerable size, it escapes in the form of a jet, which is strengthened at each beat of the pulse as an active spurt. Blood discharged from an opened vein either flows in an abundant continuous stream, or wells up from the depth of the tissues in which the vessel lies. Capillary hæmorrhage is gene-

rally much less profuse, and is most frequently seen in the form of oozing. Blood proceeding from internal organs is variously discharged by the several outlets. Sometimes it is immediately expelled, especially if in quantity; or it may escape even when in small quantity, owing to gravitation, as in hæmorrhage from the nose. Most frequently the blood is retained for a time, and then, acting as a foreign body, is ejected; for example, in bleeding from the stomach, bowels, or bladder.

*Characters*.—Blood flowing from the surface of the body usually presents its familiar characters. The colour of the blood will be scarlet when it is derived from an artery; purple, with a tendency to become scarlet on the surface, when it flows from a vein; and of a tint intermediate between these two colours when the hæmorrhage is capillary. In all cases, the blood coagulates more or less abundantly around the seat of hæmorrhage.

In hæmorrhage from internal organs, on the contrary, the blood is frequently coagulated; more or less mixed with the fluids of the part; or otherwise altered during its transit. The following are the principal changes that blood undergoes in its passage towards the several outlets of the body:—

Blood flowing from the *external ear*, or from the *anterior nares*, is frequently thin and watery, if the hæmorrhage have lasted for any length of time. Blood discharged from the *posterior nares* is often coagulated, black, and mixed with thick mucus. In hæmorrhage from the *mouth*, the blood may be derived from that cavity, from the fauces, from the respiratory passages, or from the upper portion of the digestive tract—especially the stomach. When the blood flows from some part of the *mouth*, such as the gums, tongue, palate, or cheeks, it is more or less intimately mixed with frothy saliva and mucus. In hæmorrhage from the *respiratory passages*, or from the *stomach* through the mouth, the ejected blood varies extremely in different instances (see HÆMOPHTYSIS; and HÆMATEMESIS). Hæmorrhage from the genito-urinary tract is also specially described. Hæmorrhage from the bowels is not peculiar, if the source of the blood be near the anus, or if the bleeding be very profuse; taking the form sometimes of a gush of fresh, warm blood, at other times of a mere red streak upon the fæces. If the blood be derived from a higher part of the intestines, the appearance of it, when discharged, will be different. Generally it is so far altered by the action of the various intestinal contents, as to be converted into a black tarry-looking mass (see MELÆNA). Less frequently, the bleeding may be so profuse as to fill a considerable portion of the bowel, and clots of blood may then be passed, as in some cases of hæmorrhage in typhoid

fever. The peculiar characters of *menstrual* blood are described elsewhere. Hæmorrhage from the *uterus* in large quantity consists of unaltered blood, whether coagulated or not.

**COURSE AND TERMINATIONS.**—Hæmorrhages caused by wounds are generally most profuse at first; and, unless they prove fatal speedily, or are artificially arrested, gradually cease. On the contrary, hæmorrhages due to disease are frequently insignificant at first, and increase in amount; or they recur again and again in variable quantity.

Many natural causes contribute to the arrest of hæmorrhage, the most important being weakening of the force of the heart by the loss of blood; the contraction of the coats of the vessels at the seat of lesion; the coagulation of the blood—first around, and then within the open vessel; the pressure of the extravasated blood in the surrounding tissues; and the increased coagulability of the blood after the flow has continued for some time. The relief of the local disturbance of pressure by the occurrence of the hæmorrhage is alone sufficient in many instances to arrest the flow.

**EFFECTS.**—The effects of hæmorrhage are chiefly exerted upon the system generally; and they are therefore remarkably uniform, whatever may be its locality. The circumstances that more especially affect the intensity of the effects or symptoms are two, namely, the amount of blood lost, and the rapidity of the flow. When hæmorrhage is at once free and persistent, syncope rapidly ensues, probably accompanied by convulsions, and ending speedily in death unless the bleeding either spontaneously cease or be artificially arrested (*see* SYNCOPE). The sudden loss of one-half of the total amount of blood in the body is believed to be sufficient to cause death. On the other hand, enormous quantities of blood may be lost without a fatal result if the hæmorrhage be slow, or frequently repeated with considerable intermissions. The condition of the subject of the hæmorrhage then becomes one of anæmia (*see* ANEMIA). More moderate bleedings frequently repeated produce faintness, and may cause a certain degree of pallor, which shortly disappears. Moderate hæmorrhages from certain situations, for example from the nose, rectum, or even the lungs, may sometimes remove congestion, and be attended with a feeling of relief and with real benefit.

Local effects do occur in some forms of hæmorrhage. Hæmoptysis is frequently accompanied by inhalation of blood, and may lead to inflammation or more complex disease of the lungs. Hæmorrhage into the uriferous tubules causes plugging of these, and discharge of blood-casts. Blood retained in the generative or in the respiratory passages occasionally becomes decomposed. The special symptoms of the several forms of

hæmorrhage are described under the head of each.

**TREATMENT.**—The treatment of hæmorrhage from external parts is a subject of purely surgical interest; but it may be said that, in cases of emergency, moderate pressure, with the finger or other means, on the seat of the bleeding, is generally practicable and successful. Hæmorrhage from internal parts requires special treatment according to the particular organ from which it is proceeding. The reader is, therefore, referred to the several articles on EPISTAXIS; HÆMATEMESIS; HÆMATURIA; HÆMOPTYSIS; MELÆNA; MENSES OR MENSTRUATION, Disorders of; HÆMOSTATICS; and STYPTICS.

J. MITCHELL BRUCE.

**HÆMORRHAGIC** (*αἷμα*, blood; and *ῥήγνυμι*, I burst forth).—Associated with hæmorrhage. The word is applied to certain inflammatory products or to effusions when they contain blood, as in *hæmorrhagic peritonitis*; and to varieties of certain diseases in which extravasations or hæmorrhages from free surfaces occur, for example *hæmorrhagic small-pox*, *hæmorrhagic measles*, and *hæmorrhagic purpura*.

#### HÆMORRHAGIC DIATHESIS.

*See* HÆMOPHILIA.

**HÆMORRHOIDS** (*αἷμα*, blood; and *ῥέω*, I flow).—**SYNON.**: Piles; **Fr.** *Hémorrhoides*; **Ger.** *Hæmorrhoiden*.

**DESCRIPTION.**—The hæmorrhoidal vessels, and especially the veins distributed to the lower part of the rectum, are very liable to become dilated and varicose, giving rise to a disease termed *hæmorrhoids* or *piles*. When the plexus beneath the mucous membrane within the external sphincter is thus affected, the hæmorrhoids are said to be *internal*. When the veins beneath the integuments outside the muscle are enlarged, the hæmorrhoids are called *external*. Internal and external hæmorrhoids very frequently co-exist.

*External Hæmorrhoids.*—We may distinguish two kinds of external piles—(1) the *venous*; and (2) the *cutaneous*.

(1) The *venous tumour* consists of a softish elevation of the skin near the margin of the anus, of a rounded form, and a livid or slightly blue tinge. On cutting into it, we find it is formed by a dilated vein or veins, which after inflammation will be occupied by a dark-coloured coagulum.

(2) The *cutaneous excrescence* consists of a flattened prolongation of skin, due to hypertrophy of the cutaneous layers, and is commonly pedunculated. It is generally the result of the first form—a projecting skin tag left after absorption of the coagulum having undergone some hypertrophy. Often there is only a single broad flat excrescence at the side of the anus, but sometimes there are two—one on each side; and occasionally there are

several encircling the anus. They are apt to become inflamed and œdematous. Similar excrescences occur as the result of irritating discharges from the bowel, and are common in stricture and chronic ulceration of the rectum.

*Internal Hæmorrhoids.*—Internal piles may be divided into three classes—(1) the *venous*; (2) the *columnar*; (3) the *navoid*.

(1) The *venous pile* resembles the external venous pile, except that it is covered by mucous membrane and is within the sphincter.

(2) The *columnar pile* consists essentially of hypertrophy of the folds of mucous membrane which surround the anal opening—the pillars of Glisson. They have a red, almost vermilion, colour, an elongated form, and contain within them one of the descending parallel branches of the superior hæmorrhoidal artery (Hamilton). This pile is very vascular, bleeds copiously, and is made up of hypertrophied submucous tissue with many dilated arteries and veins.

(3) The *navoid pile* is bright red, spongy and villous, and has been well compared in aspect to a strawberry. In structure it resembles a capillary nævus. It may bleed almost continuously.

Internal piles seldom attract attention until they have become so developed as to protrude at the anus in defæcation. They then exhibit a remarkable diversity of appearance, according to their number, size, and condition. The protrusion may consist of only one large pile, found usually towards the perinæum, especially in women. More commonly there are three distinct prominent growths, differing in size, one at each side of the anus, and a third in front—the latter, the perineal, being the largest. In old cases they may be more numerous—as many as four or five. The distinction between them is commonly well-marked, but not always, for the piles sometimes merge into each other, so that the protrusion forms nearly a circular prominence. The aspect of extruded piles depends much upon their condition, whether congested, inflamed, or constricted by the sphincter. In an inactive state, and in a relaxed condition of the sphincter, they form softish tumours of a granular appearance, presenting just at the orifice of the anus; but when protruded and congested, they constitute large tense tumid swellings, of a deep red colour and smooth surface, which readily bleed. When hæmorrhoids are of large size, the integuments at the margin of the anus become everted, and form a broad band girding the base of the tumours. The skin thus everted is liable to be mistaken for external piles, and to be excised in operations—an error very likely to be followed by serious contraction of the anus.

*ETIOLOGY.*—Hæmorrhoids are a disease of middle and advanced age. They rarely occur before puberty; and but few persons in after-life altogether escape them. All circum-

stances which determine blood to the rectum, or which impede its return from the pelvis, tend to produce this disease. There is in many persons a natural predisposition to the complaint, which may be hereditary. But a predisposition is most frequently acquired by sedentary habits, indulgences at table, and neglect of the bowels. Hæmorrhoids, though a common disease in both sexes, occur more frequently in males than in females. Few women bear children without becoming in some degree affected by them; but the urinary and genital disorders of the other sex, combined with freer habits of living, leading to congestion of the liver, are still more fertile sources of piles. It must be remembered that piles may be a symptom only in certain cardiac, hepatic, or uterine diseases.

*SYMPTOMS.*—The symptoms produced both by external and internal piles vary greatly in different subjects, and in different stages of the complaint.

*External* piles cause a feeling of heat and tingling at the anus. A costive motion is followed by a burning sensation, and the excrescence becomes swollen and tender on pressure, so as to render sitting uneasy. This congested state of the pile may pass off; or it may lead to inflammation accompanied with considerable enlargement of the hæmorrhoid, forming an oval tumour, red, tense, and extremely tender. In such case phlebitis has ensued, and the pile cavity is occupied by a coagulum. The inflammation may subside or go on to suppuration. When the matter is discharged, the clot of blood escapes with it, the abscess closes, and the dilated vein is usually obliterated, the pile being reduced to a small flap of integument. External piles rarely give rise to bleeding.

*Internal* piles, when slight, may exist for years, causing little inconvenience besides slight bleeding after a costive motion; with occasionally a feeling of fulness, heat, and itching just within the anus. If only small, they protrude slightly with the mucous membrane in defæcation, returning afterwards within the sphincter. When of large size, the piles always protrude at stool, and require to be replaced, the patient usually pushing them up with his fingers. In a lax state of the sphincters, and in a loose hypertrophied condition of the mucous membrane from which they spring, hæmorrhoids come down, even when the patient stands or walks about, so as to prove exceedingly troublesome, and to interfere with his taking walking exercise. In consequence of the irritation from pressure and friction to which the protruding piles are liable, their mucous surface becomes tumid and abraded, and furnishes a free mucous discharge tinged with blood, which soils the linen. They are often so sore that the patient is obliged to keep the recumbent posture, the pressure in sitting causing more or less uneasiness.

Persons subject to piles frequently suffer no inconvenience from them until irritated by an unusually costive motion, or by a smart purgative; or when, under the excitement of alcohol, the growths become congested and inflamed, and cause spasm of the sphincter muscle. Then they have what is termed an 'attack of piles'—that is to say, they experience a sensation of heat, weight, and fulness just within the rectum, followed by considerable pain at stool, and sometimes irritation about the bladder. These symptoms, which are often attended with febrile disturbance, arise from inflammation and swelling of the piles, which afterwards subside, but seldom without leaving some enlargement of the growths. Anæmia may follow upon continued bleeding; and reflex pains in the back, groin, and genitals are often complained of.

*Strangulation.*—When internal piles of some size protrude at the anus and are not returned, they are liable to be constricted and strangulated by the external sphincter. The contracted muscle impedes the return of blood, and occasions inflammatory swelling of the piles, which may become strangulated and gangrenous. In this way hæmorrhoids of large size have been known to slough off, the patient being relieved of the annoying complaint by a sort of natural process. An occurrence of this kind is attended with a good deal of pain and suffering, but is free from danger. In general, the extremities only of one or two of the larger growths perish, and the patients, though experiencing relief, are by no means cured of the disease.

*Hæmorrhage.*—One of the most common symptoms of internal hæmorrhoids—indeed that from which the name of the complaint is derived—is hæmorrhage, which occurs when the bowels are evacuated. The bleeding varies greatly in amount. Sometimes the motions are merely tinged with a few drops of blood; in other instances the quantity lost is considerable, several ounces being voided at stool. The bleeding may be irregular, occurring only after costive motions, or in certain states of health; or it may take place daily, going on even within the bowel, and producing the usual symptoms of derangement from continued losses of blood. The character of the bleeding also varies with the structural character of the pile, being, however, usually venous. There are persons who are liable to discharges of blood from the hæmorrhoidal veins, either at regular periods, or when, from good living or want of exercise, some plethora is induced. In these cases from three to six ounces of blood, or even more, pass away at stool, following the evacuation; and the blood which is voided is of a dark colour and evidently venous. Such discharges must not be rashly interfered with. They relieve congestion of the liver and kidneys, help to ward off attacks of gout,

and prevent fits of apoplexy; so that in certain of these plethoric persons they are rightly regarded as safety-valves. The bleeding from internal piles is undoubtedly in some cases arterial. Sometimes the blood may be observed to escape from the pile in jets. This is not evidence that it is arterial, but may be due to a regurgitant stream of blood forced through a rent in a vein by the pressure of the abdominal muscles. That bleeding from piles is always good for the health is quite a mistaken notion; and it is important that the practitioner should distinguish the bleeding taking place as a consequence of local disease, from that which arises from plethora, congestion of internal organs, or such a disease as cirrhosis of the liver.

*TREATMENT.*—When piles are small and cause but little inconvenience, the treatment is very simple. Persons with this complaint should take stimulants in great moderation, if at all; and in most instances they would do well to abstain entirely from alcohol. Many individuals never suffer from piles except after taking a glass of spirit-and-water, or a few glasses of wine. Such persons should become water-drinkers. Active exercise in the open air should be taken daily; and the patient should avoid sitting too long at the desk, because it is by prolonged sedentary occupation and the neglect of the rules of health that hæmorrhoidal complaints are encouraged. Chairs with cane seats are to be recommended. The bowels must be carefully regulated, so as to avoid hard and costive motions, as well as too frequent action. Irritating the rectum by active and repeated purging is more hurtful even than constipation. In regulating the bowels, a simple enema of half a pint of cold water, injected after breakfast, answers most admirably, and is more efficacious than purgatives. If aperients be needed, preference may be given to confection of senna, cascara, or the compound liquorice powder. Carlsbad salts, or the foreign mineral waters—the Pullna, the Friedrichshall, or the Hunyadi János—taken in the morning fasting, answer well with many persons, and ensure a comfortable relief. The diet must be simple and moderate, and the habits of the patient regular. The relief afforded by this palliative treatment is often remarkable.

Ordinary bleeding may be met by injections of cold water, or by some astringent injection or ointment; among these may be named a solution of sulphate of iron (10 grs. to ℥j), or preparations of tannic acid or rhatany. When the bleeding is copious, injections are not so successful, and operative treatment often becomes necessary. As a local application, the ointment of galls and opium is of value, or a combination of morphine and tannic acid.

External piles when large and troublesome,

and internal piles when they protrude at stool, and are not easily reduced, or are subject to inflammation, ulceration, and frequent bleeding, can be removed only by operation. It is seldom desirable to operate upon piles developed during pregnancy, or upon hæmorrhoids symptomatic of grave constitutional disease.

T. B. CURLING. FREDERICK TREVES.

**HÆMOSTATICS** (αἷμα, blood; and σπάρτός, stopped).

**DEFINITION.**—Internal remedies and local applications which arrest hæmorrhage.

**ENUMERATION.**—The chief hæmostatics are: the Ligature; Pressure; Rest; Cold; the Actual Cautery; Astringents; and the whole class of Styptic drugs.

**ACTIONS AND USES.**—When taken in their widest sense, it is evident that hæmostatics must include all the various means which have been devised to stop bleeding. Externally we must vary our plan of treatment according to circumstances. No surgical principles are better founded than those which enjoin us to tie a wounded artery, and to apply pressure to a vein; and for the absolute arrest of hæmorrhage from any readily accessible part a most powerful aid has been provided in Esmarch's elastic bandage. When the bleeding depends on general capillary oozing, the application of ice may often prove effectual; and where this fails, recourse must be had to some of the numerous articles of the Pharmacopœia, already referred to, which possess styptic properties (see STYPTICS). An example of the successful application of a hæmostatic is the arrest of uterine hæmorrhage by means of injections of perchloride of iron. In the case of undue hæmorrhage from a leech-bite, if milder remedies, such as pressure, do not succeed, we may apply the solid nitrate of silver, or include the bleeding point in a loop of twisted suture.

Absolute rest is essential for the successful treatment of hæmorrhage; and the regulation of the diet and of the bowels is equally to be attended to. For the details of treatment in each particular form of hæmorrhage, the reader is referred to HÆMATEMESIS; HÆMOPHILIA; HÆMOPHTYSIS; MELÆNA; &c.

ROBERT FARQUHARSON.

**HÆMOTHORAX.**—See HÆMATOTHORAX.

**HAIR.**—The intimate anatomical relations which exist between the hair and the cuticle, on the one hand, and the skin-follicles on the other, naturally influence or determine the diseases to which the hair is liable. These diseases may be divided into three chief groups: those of nutrition and growth, including atrophic, hypertrophic, and pigmentary changes; diseases associated with inflammation of the follicles, such as sycosis

and other forms of folliculitis; and parasitic affections. The two latter groups will be found described under their respective heads, so that in this place we have to deal chiefly with the first group.

**Diseases involving the Nutrition and Growth of the Hair.**—Most of the alterations in the growth and colour of the hair are atrophic in their nature. The more common forms of atrophy produce various kinds of baldness; and in addition we have greyness or loss of the natural colour of the hair, and certain structural alterations, such as those met with in trichorexis nodosa; these latter alterations, though not simple atrophies, yet belong to the atrophic group.

**Alopecia or Baldness.**—Baldness is of two kinds—*congenital* and *acquired*. *Congenital* alopecia is very rare; it is a malformation rather than a disease, and is generally associated with a defective development of the teeth. This condition is permanent.

*Acquired* baldness arises from many different causes. It may result (1) from *various forms of disease*; (2) from *simple defective nutrition and growth*; or (3) from *atrophic senile changes*.

(1) *Baldness from various forms of disease.*—Different diseases produce baldness in different ways, and they may be conveniently divided into four groups: acute febrile diseases; follicular inflammations; nervous affections of the skin; parasitic diseases.

The baldness which follows *fevers* is always temporary, though often severe. There is apparently an almost sudden death of the hair at the time of the fever, and this is followed by its falling off some two or three months afterwards. Parturition and some other illnesses besides fevers produce a similar result, though generally more slowly and persistently. The falling off of the hair which accompanies secondary syphilis is apparently produced in a similar way, and often occurs without any visible eruption on the scalp.

*Folliculitis*, or inflammation of the hair-follicles, is one of the commonest causes of baldness. We meet with it in seborrhœa capitis, in sycosis, and in various pustular eruptions involving the hair-follicles. When produced in this way, alopecia may be temporary or permanent, according to the nature or the severity of the disease. All diseases of the skin which leave scars or obliterate the follicles lead to permanent alopecia of the parts affected. Erythematous lupus, scleroderma, and some forms of syphilis, are examples. It is especially these forms of alopecia resulting from folliculitis which are so much aggravated by the stimulating applications almost universally recommended by hairdressers, without regard to the cause of the malady.

The changes in the *nervous system* which produce baldness may be general, as in some

cases of shock; or local, as in alopecia areata. Falling of the hair, which sometimes follows a blow or other injury, belongs to this class. See ALOPECIA AREATA.

The *parasitic diseases* which lead to partial baldness are tinea tonsurans and favus, which are fully described under their proper headings.

(2) *Baldness from defective nutrition*; and (3) *Senile baldness*.—Premature baldness from defective nutrition differs little from senile alopecia, and is often hereditary. The changes, however, in these cases are said to differ from those due to old age, inasmuch as there is no thinning or atrophy of skin such as we meet with in senile baldness. This view is, however, certainly erroneous with regard to the great majority of cases, which are quite indistinguishable from senile baldness, not only in the part of the head affected, but also in the atrophic changes of the scalp itself. The two cases, in short, differ only in degree and not in kind.

The treatment of this kind of alopecia, as might be expected, is not very satisfactory. Some good, however, may be done in the early stages by stimulating remedies. One of the best is a lotion consisting of vinegar of cantharides, f. ℥vi.; glycerine, f. ℥j.; spirit of rosemary, f. ℥ij.; rectified spirit, f. ℥v. This should be dabbed on the scalp daily with a small sponge. Frequent and excessive sweating is certainly an exciting cause of premature baldness in those predisposed to the malady, and hence its frequent occurrence in Europeans who live in tropical climates. See BALDNESS.

*Pigmentary changes*.—Canities, or greyness, results from the deficient development of the natural pigment of the hair. It may be congenital or acquired. The extreme form of congenital canities is met with in albinos, in whom there is an absence of pigment in the skin and choroid as well as in the hair. In an irregular form it sometimes exists as a congenital white lock on the scalp or face, the surrounding hair being of a normal colour.

As an acquired alteration in the hair, canities is well known as a simple senile change. Premature greyness, not differing essentially from the senile change, is often met with in those suffering from any chronic disease, such as phthisis; it is also more or less hereditary. In all these cases the greyness is symmetrical. In an unsymmetrical form it is met with in several diseases of the skin, especially in area, leucoderma, and chronic neuralgia of the scalp. The distribution of the grey hair and the history of the case will serve as a means of differential diagnosis.

In many cases of canities any treatment is obviously useless. In those cases which admit of rational treatment, cantharides is by far the best local stimulant, as it has a

tendency to excite the natural development of colouring matter, as may be often seen in the skin where a blister has been applied. Arsenic is usually the best form of tonic for the same reason.

*Excessive growth of hair*.—Writers have generally stated that hairs may increase in numbers and size. With regard to an increase in their numbers, the statement cannot be accepted. Downy and almost invisible hairs may grow into strong and visible ones, but there is never any increase in their number; it is really a great increase in their size which leads to the erroneous belief that they have multiplied. Sometimes the old hair is held in the follicle after the new hair has grown up, but this is not a true increase of numbers. An excessive growth of hair is not uncommon, and is chiefly inconvenient on the face in women. The ordinary depilatories which are used to get rid of this excessive growth are worse than useless. See DEPILATORIES.

*Structural change in the hair*.—*Trichoclasia*, or *trichorexis nodosa*, is the most important and best-known change in the structure of the hair. The affection was first described by the late Erasmus Wilson and Beigel. To the naked eye the hairs appear to be marked with two, three, or more small bulging spots, somewhat resembling nits. The hairs very easily break at these points, which, examined under the microscope, seem to consist of spindle-shaped swellings of the shaft of the hair. In a more advanced stage this swelling partially bursts near its most distended part, and finally the hair breaks with a ragged fracture, so that the divided hair has the appearance of two brushes, the bristles of which are interlocked. When the separation is complete, a stumpy hair is left with a frayed or brush-like free extremity. Trichorexis is chiefly met with in the hairs of the face, especially those of the chin.

*Lepothrix*.—Although this curious affection is a concretion rather than a structural change of the hair, yet it has that appearance to the naked eye, and may be conveniently referred to under this head. The hairs affected with this disease are met with in the axillæ and on the scrotum. Indeed, warmth and moisture appear to be essential to its development. The concretions, seen under the microscope, have usually a rounded and nodulated form apparently surrounding the shaft of the hair, but not symmetrically. Sometimes the concretion extends continuously for some distance along the shaft of the hair, giving it a feather-like appearance. The hairs themselves are abnormally brittle. The exact nature of these curious concretions has not yet been determined, but they are associated with a micrococcus. See LEPOTHRIX.

ROBERT LIVEING.

**HALL**, in Austria.—Common salt waters, with iodine. See MINERAL WATERS.

**HALLUCINATION** (*hallucinator*, I blunder).—A false perception of an organ of sense, for which there is no external cause or origin (see ILLUSION); as when a man in total darkness thinks he sees an object. Hallucinations of all the senses occur, the most frequent being those of sight and hearing. They may be found in persons not insane, but indicate a disordered state of brain.

**HAMMAM-MELOUAN**, in Algiers. Thermal muriated saline waters. See MINERAL WATERS.

**HAMMAM-MESKOUTINE**, in Algiers (the *Aquæ Tibilitina* of the Romans).—Highly thermal saline springs, weakly mineralised. See MINERAL WATERS.

**HAMMAM-R'HIRA**, in Algiers.—Highly thermal waters. See MINERAL WATERS.

**HANGING**, Death by.—Hanging is the effect of suspension of the body by the neck by means of a ligature or noose, the constricting force being the weight of the body, wholly or in part, or the weight multiplied by the distance through which the body falls. The mode of death varies according to these circumstances. With a long drop, the method now usually employed in judicial hanging, and particularly if the knot is under the chin, death is not infrequently due to fracture, or displacement, of the cervical vertebræ, and injury to the medulla oblongata. Death may also occur, without such anatomical lesion of the cervical vertebræ, from shock or syncope, or, as it is termed by Casper, neuro-paralysis.

When death does not occur in either of these ways, it is the result of asphyxia from occlusion of the air-passages, or rather of asphyxia in combination with coma, caused by compression of the cerebral blood-vessels. Though compression of the carotids and jugulars may be maintained for a considerable time without a fatal result, if the trachea is open below the point of constriction—whereas death speedily ensues if the air-passages are also occluded—yet death may result from the disturbance of the cerebral circulation alone; and the two causes operate conjointly in every case, in varying proportions.

**PHENOMENA**.—When death is not instantaneous, as in cases of injury to the medulla, or from neuro-paralysis, convulsive movements of the type seen in asphyxia may continue for some minutes after suspension, and the heart may continue to beat for a considerable period after all other vital movements have ceased.

Subjects who have been partially hanged

have described various sensations, more or less pleasurable, similar to those of cerebral congestion and narcotic stupor.

**POST-MORTEM APPEARANCES**.—The appearances found after death by hanging are not uniform or constant; and there is no single sign invariably present diagnostic of this mode of death. Indicative of suspension, but not necessarily of death so caused, is the mark of the cord on the neck. Usually it is above the hyoid, passing obliquely upwards behind the ears, and losing itself in the occiput. But the position may vary according to the tightness of the noose before suspension, or the position of the head and direction of the pressure. It is generally single, but if the cord should have been twisted twice round the neck, two marks may be found, one circular and the other oblique. The characters of the mark differ somewhat according to the texture and thickness of the ligature. Usually it is a shallow groove or furrow, of a whitish or brownish hue and parchmenty consistence, occasionally abraded, rarely ecchymosed; but it may have livid edges or a chocolate tint. The appearances may vary in different parts of the same mark. The subcutaneous cellular tissue is compressed and silvery. Occasionally minute extravasations are seen in the deeper layers of the skin. The middle and internal coats of the carotids are sometimes lacerated; and where the momentum has been great, lacerations of the cervical muscles, hæmorrhage in their vicinity, fracture of the larynx, rupture of the thyro-hyoid ligaments and fracture of the hyoid bone, and fracture or dislocation of the cervical vertebræ, with injury to the medulla and effusion into the spinal canal, have been found. All the appearances usually found in the neck in cases of hanging may be produced by suspension of the dead body, especially if the legs are pulled forcibly downward.

The face is sometimes, but not commonly, distorted and expressive of suffering. Usually it is placid and pale, though if the body have hung for some time, it becomes very livid. The eyes are sometimes very prominent, and the pupils are usually dilated. Frothy mucus may be found at the mouth and nostrils. The tongue is pressed against the teeth and indented, or it may be clenched between the jaws. The base of the tongue is injected. The hands are often tightly clenched, the nails even being driven into the palms. Erection, or semi-erection, of the penis in men, with expulsion of semen or prostatic fluid, and vascular turgescence of the genitals in females, with sanguinolent effusion, are occasionally observed. Expulsion of the contents of the bladder and rectum is likewise common. The condition of the brain varies. Congestion of the meninges is sometimes pronounced, at other times not very marked. The mucous membrane of the larynx and

trachea is congested, and mucous froth is present. The lungs are at times pale and distended; at other times collapsed. The condition of the heart and venous system characteristic of asphyxia is common. Marked redness of the mucous membrane of the stomach, simulating irritant poisoning, has been occasionally noted. The determination of the fact of death by hanging depends on a consideration of these various phenomena, and the absence of other causes of death.

**ACCIDENT, SUICIDE, OR HOMICIDE?**—Hanging is rarely homicidal. It signifies great disproportion of strength between the assailant and the victim; and therefore, in the absence of this condition, there will be injuries indicative of a struggle. Apart from collateral circumstances, homicide can only be argued from the presence of such injuries as could not have been self-inflicted or caused accidentally during the act of suspension. Occasionally hanging is accidental, as in foolish experiments and insane imitation. It is not necessary that the body should be entirely off the ground to cause death by hanging. Many instances are recorded of suicide by hanging in most extraordinary positions calculated to throw the greater part of the body-weight on the noose.

**TREATMENT.**—This is rarely called for, except in accidents or attempted suicide. The body must be cut down, and artificial respiration employed. Venesection may be had recourse to for relieving cerebral congestion. See **ARTIFICIAL RESPIRATION**; and **RESUSCITATION**.  
D. FERRIER.

**HARKANY, in Hungary.**—Thermal sulphur waters. See **MINERAL WATERS**.

**HARROGATE, in Yorkshire.**—Saline, chalybeate, and sulphur waters. See **MINERAL WATERS**.

**HASTINGS, on the South-East Coast of Sussex.**—A mild climate. Mean winter temperature, 40° F. Exposed to the east, but sheltered from the north. See **CLIMATE, Treatment of Disease by**.

**HAUT MAL (Fr.)**—A synonym for epilepsy gravior. See **EPILEPSY**.

**HAY FEVER.**—**SYNON.**: *Catarrhus astivus*; Bostock's Catarrh; Hay Asthma; Fr. *Asthme d'été*; Ger. *Frühsummercatarrh*.

**DEFINITION.**—A catarrhal affection of the mucous membrane of the eyes, nose, mouth, pharynx, larynx, and bronchi, accompanied by dyspnoea; induced in persons predisposed to it by the action of the pollen of various plants, chiefly of the Gramineæ; prevalent during the hay season, but subsiding at its close; and varying in severity according to certain atmospheric conditions, and the amount of pollen present in the air.

**ÆTIOLGY.**—Hay fever exists in Europe

and North America, and it is especially common in England, where the number of cases is annually double that of any other country. The Anglo-Saxon race appears more liable to it than other races. It prevails more among men than women, probably because the former are most exposed to the atmosphere; and inhabitants of towns visiting the country are more liable to attack than the country-people themselves. Dr. J. N. Mackenzie of Baltimore assigns hay fever to an exalted condition of the nasal erectile tissue, and especially the portion covering the posterior end of the inferior turbinated bone and the septum immediately opposite, rendering it more sensitive to the action of pollen and other irritants; and he maintains that destruction of this erectile tissue by the galvano-cautery cures the disease.

It has been ascribed by some writers to the sun's heat in the summer months, also to certain odours, vegetable and animal; but the experiments of Mr. C. H. Blackley show it to be due to the specific influence on certain mucous membranes of the pollen-grains of the following natural orders of plants: Ranunculaceæ, Papaveraceæ, Fumariaceæ, Crucifere, Violaceæ, Caryophyllaceæ, Geraniaceæ, Leguminosæ, Umbelliferae, Rosaceæ, Liliaceæ, Compositæ, Gramineæ, and others, both exotic and native. Different kinds of pollen were applied to the mucous membrane of (1) the nares, (2) larynx, trachea, bronchial tubes (by inhalation), (3) conjunctivæ, (4) tongue, lips, and fauces; and in all these cases it produced the symptoms of hay fever, the pollen of grasses being most potent. Amongst these, secale cereale, or rye, exercised most marked effects, though greater influence is generally attributed to *anthoxanthum odoratum*.

It has been found that large quantities of pollen float in the air during the summer months; and that the number of cases of hay fever depends on the amount present, which increases in warm damp weather, decreases when it is very dry and hot, and often nearly disappears after heavy rain. Cold weather reduces the number of sufferers by checking the inflorescence of plants. The higher strata of the atmosphere appear to contain more than that immediately overlying the soil; and Mr. Blackley found the greatest number of pollen particles at between 1,000 and 1,500 feet above the earth's surface, whither they are probably carried by aerial currents. The number of pollen grains present reaches its maximum in June, when Mr. Blackley collected 880 in a day on a square centimetre of glass. The size and forms of the pollen grains vary greatly in the different species, but this does not seem to influence their action, which appears to depend on the pollen sac absorbing moisture from the contiguous mucous membrane and bursting,

when the minute granules it contains are thus extruded, and cause irritation.

**SYMPTOMS.**—An attack of hay fever generally occurs without any premonitory disturbance, immediately on the application of the pollen to the mucous surfaces—for instance, when the person enters a hay-field. The first symptoms are itching of the parts with which the particles come in contact, beginning with the hard palate and fauces, and then extending to the nostrils, the eyes, and face, though, if the wind be strong, the eyes may be first attacked. The catarrhal stage follows, marked by violent fits of sneezing, and running from the eyes and nose, with occasional pains in the head and in the frontal sinuses; then the submucous tissue of the nares swells; and in a short time both nostrils become blocked and impervious to air. A change to the recumbent position, however, if the patient lies on one side, will often re-open the uppermost nostril, while the other, as the result of gravity, becomes still more occluded. The sneezing will continue without fresh application of pollen, as at night, when the subsidence of the swelling restores, or even exaggerates, the sensibility of the Schneiderian membrane. The *alae nasi* become red and inflamed, and occasionally bleed. The discharge after this becomes less in quantity, inspissated and puriform, and finally subsides. In the eyes, the swelling of the submucous tissue causes closure of the lachrymal canals and nasal ducts; and considerable injection of the conjunctival capillaries is apparent. Sometimes, but rarely, œdema of the eyelids follows. Similar to the nose-symptoms are those occurring in the throat, some swelling taking place in the pharynx, which gives rise to partial closure of the Eustachian tubes, and hence to a certain degree of deafness. Slight feverishness is occasionally present, the pulse quickening to 100, and the temperature slightly rising; but in a large number of cases pyrexia is entirely absent. The changes which take place in the mucous membrane of the air-passages give rise to asthmatic symptoms, such as tightness of the chest, difficult and wheezy breathing, with prolonged expiration, and some dry cough, followed, at the close of the attack, by expectoration.

The catarrhal symptoms are, however, more characteristic than the asthmatic, which are not invariably present.

The liability to attack lasts generally from three to four weeks in summer, but its duration depends on the presence of the exciting cause, which, if not removed, may cause the malady to last for months. A fall of rain will diminish the disorder by clearing the air of pollen; exercise, which increases the number of respirations and, therefore, of pollen grains inspired, will render it more severe; while each attack makes the individual more susceptible to this subtle influence, and conse-

quently augments the probability of other seizures. As a rule, hay fever has no complications, and passes away completely on the removal of the exciting cause. Constant recurrence of the attacks has been noticed to lead to deafness, owing to catarrh of the Eustachian tubes.

**DIAGNOSIS.**—The diagnosis of hay fever from other affections is easy, as the fact of the catarrhal symptoms occurring only in summer separates it from an ordinary 'cold in the head'; while their combination with dyspnoea in hay fever prevents it being mistaken for spasmodic asthma arising from other causes, in which there is usually no catarrh.

**PROGNOSIS.**—The prognosis is favourable if the patient be removed from the exciting cause, as the asthmatic symptoms seldom, if ever, lead to pulmonary emphysema, or to any permanent change in the bronchi.

**TREATMENT.**—The most obvious course in the treatment of hay fever, but not always the most easy one, is to avoid exposure to pollen. Mr. Blackley notices that a small amount of this material might exist without giving rise to hay fever, but if ten particles of pollen were detected on the glass slide exposed to the air for twenty-four hours, symptoms were sure to appear in persons liable to it. Sufferers from this complaint should avoid hay-fields, hay-ricks, and much exposure and exertion in the country during the hay season, and should remain to a great extent within doors; but, where circumstances admit, change to the seaside is highly desirable, and it frequently effects a speedy cure. Even on the coast some care must be taken to select a locality free from vegetation; for if the wind blows from the land, and hay-grass be flowering at the time, an attack may be induced. Choice should be made of a seaside place backed by high cliffs, and where the prevailing winds are from the sea. Another method of avoiding attacks is by sea voyages or short yachting cruises, undertaken during the hay season, and many sufferers enjoy complete exemption in this way. Of other localities, high mountain stations, where there is more grazing than hay-growing, and closely inhabited cities with few parks or grassy squares, are to be preferred. Carbolised cotton-wool and other available respirators are sometimes used with advantage.

The medicinal treatment consists, first, in combating the general predisposition to the complaint by tonic measures; and, secondly, in allaying the local irritation.

The first object is best achieved by shower baths, and by such tonics as iron, quinine, nux vomica, sulphate of zinc, and arsenic. Lotions of sub-acetate of lead or sulphate of zinc applied to the eyes and inner surface of the nostrils give some relief; but the writer has found the most successful results from brushing the interior of the nostrils and pharynx with a solution of hydrochlorate of

cocaine (20 per cent.) with a curved brush. Sir Andrew Clark strongly recommends the application of a combination of glycerine of carbolic acid, quinine, and perchloride of mercury to the floor of the nares and pharynx. Various sprays are exceedingly useful, such as those of solutions of carbolic acid (eight grains to an ounce), sulphurous acid (equal parts with water), sulphate of quinine (two grains, with acid, to an ounce), and tannic acid (four grains to an ounce); but the best of all is a solution of cocaine (8 to 10 per cent.), which reduces the sensibility of the parts affected. The spray may be applied, with proper precautions, to all the irritated surfaces—eyes, nose, throat, and larynx—with great relief.

C. THEODORE WILLIAMS.

**HEADACHE.**—SYNON.: Cephalalgia; Fr. *Douleur de tête*; Ger. *Kopfschmerz*.—Pain or uneasiness in the head is very variable in its nature, and is produced by a great number of causes. It is present at some period or other in the course of most acute, and many chronic diseases; and may be associated or not with organic change in the brain, or in other organs of the body.

**SYMPTOMS.**—Headache presents many varieties. It may be slight or most intense; superficial or deep-seated. It may be more or less confined to particular parts, as the forehead, the temples, the occiput, or vertex. Sometimes the pain is limited to one spot, producing the sensation as if a nail were being driven into the head, when it is called *clavus*. It may extend over one side of the head, as in hemicrania, or *mégrim*; or be generally diffused. Headache presents every variety of character—dull, sharp, cutting, &c. Its accession may be sudden or gradual; and the paroxysms may be of the shortest possible duration, or may extend over hours, days, or months. The pain may be simple, or associated with various perverted sensations, such as giddiness, tingling in the limbs, disordered hearing, or disturbances of vision.

**VARIETIES.**—For practical purposes headaches may be arranged in the following order:—

1. *Structural headache*, or headache dependent upon disease within the cranium.
2. *Congestive headache*.
3. *Nervous or sick headache*—hemicrania or *mégrim*.
4. *Toxæmic headache*.

1. *Structural headache*.—This may be due to any of the many forms of disease of the brain, or of its membranes, such as meningitis, cerebral softening, abscess of the brain, cerebral tumour, &c.; or it may be premonitory of cerebral softening. It is nevertheless often wanting in these disorders, and the locality of the pain, when present, by no means corresponds with that of the lesion. As a rule, the pain of organic disease is fixed

and habitual, though sometimes, as in abscess or cancerous tumour, it may be of an intermittent character. If there be sickness associated with it, the sickness occurs without any apparent gastric disorder, and the pain continues after the sickness ceases. Stooing, and even the recumbent posture, aggravates the pain, whilst it is lessened by elevating the head. If organic disease be suspected, the collateral symptoms must be carefully scrutinised and weighed. It rarely happens that organic disease needs to be inferred from pain alone.

2. *Congestive headache*.—Many forms of headache depend upon a greater or less degree of congestion of the vessels of the brain; the congestion being either active or passive.

Active congestion may be caused by hypertrophy of the left ventricle of the heart, general plethora, catamenial irregularities, mental or emotional excitement, and other conditions. The pain in such cases is of an obtuse character, affecting the whole or a part of the head, particularly the forehead and occiput. It is accompanied with a sense of pulsation in the ears, flushed face, glittering eyes, and giddiness on stooing.

Passive congestion may be produced by dyspnoea, by asthma, by valvular disease of the heart, or by disordered action of the liver, bowels, and skin; it may be an after-effect of drunkenness; or may result from any cause which can produce a state of debility in the vessels of the brain, such as general anæmia, exhaustion from fatigue, loss of blood, leucorrhœa, or that following over-excitement, mental exertion, or bodily fatigue—causes which all favour congestion. When the headache is induced by anæmia, debility, or indigestion, the pain occurs generally across the forehead or at the top of the head.

3. *Nervous or sick headache, hemicrania or mégrim*.—This disorder is discussed in a separate article. See *MÉGRIM*.

4. *Toxæmic headache*.—The headache which attends all fevers and inflammatory disorders, though due in some measure to cerebral congestion, is chiefly caused by the action of the blood, altered in character and elevated in temperature, on the nervous elements of the brain. In uræmia likewise the headache, which frequently precedes or accompanies the other symptoms pointing to the existence of renal disease, results from the morbid condition of the blood. So also, in some persons, breathing the impure air of a crowded room, or the products of the combustion of gas, will, by the imperfect decarbonisation of the blood, speedily produce a headache.

**DIAGNOSIS.**—Besides the above varieties of headache, pain about the head external to the brain may be produced by *rheumatic* affections of the scalp, with tenderness of the skin and rheumatism in other parts; by *sphilitic* affections of the periosteum or bone; by *inflammation* of the scalp, com-

mencing erysipelas, &c; and by trigeminal and other varieties of *neuralgia*. Headache may be discriminated from neuralgia by its mode of accession; by the generally longer duration of the attack; and by the more complete intermissions.

**TREATMENT.**—The treatment of headache must necessarily depend upon the peculiarities of each individual case. In organic or toxæmic headache, the disease with which it is associated, and not the symptom itself, will of course be the object of consideration, and the treatment will be found discussed in connexion with these special morbid states. The same remark applies to many forms of congestive headache, such as those produced by disease of the heart, asthma, &c. If catamenial irregularities or uterine disorders are the exciting cause, these must be treated by appropriate measures. If anæmia or debility be present, then, in the intervals between the paroxysms, iron in some form, either alone or in combination with quinine or some vegetable bitter, must be given. During the paroxysms a little sal volatile, a cup of soup or strong tea or coffee, or some weak alcoholic stimulant, may be of service. Where anæmia is not a prominent symptom, or when the disorder assumes a periodical or intermittent character, quinine alone, in doses of two or three grains twice or three times a day, may be given; and if this fails to afford relief, arsenic is often of signal service. Great benefit is frequently afforded in the latter cases by the careful administration of alcohol or other alcoholic stimulant during the paroxysm. Except when the headache is associated with general plethora or active congestion, strong purgatives are to be avoided, and the bowels are to be regulated by the mildest aperient which will answer the purpose. The patient's habits and mode of life must also be strictly regulated; and care must be enjoined as to diet, sleep, clothing, and exercise, especially if the headache be associated with dyspepsia. If dyspeptic symptoms are prominent, or if the pain be connected with a gouty diathesis, then these disorders must be treated with their appropriate remedies.

If during the paroxysms the head be hot and the face flushed, warm or cold lotions, iced water, or eau-de-Cologne may be applied; a warm douching may be useful in some cases. Occasionally in severe attacks a few leeches may be placed on the temples or behind the ears with advantage, or a blister to the nape of the neck, but never if the face be pale and the pulse feeble. Compression of the temporal arteries with a pad, sustained pressure around the head, or holding the arms high above the head, will sometimes relieve severe congestive headache.

The treatment of sick headache is discussed under the article MEGRIM.

P. W. LATHAM.

**HEALING SPRINGS**, in Bath County, Virginia, U.S.A.—Thermal waters. See MINERAL WATERS.

**HEALTH**, Maintenance of.—See DISEASE, Causes of; PERSONAL HEALTH; and PUBLIC HEALTH.

**HEARING**, Disorders of.—These disorders may be grouped under three classes, namely: (a) *Partial or complete loss of hearing*, or *deafness*; (b) *Exalted hearing* (so-called); (c) *Perverted hearing* or *tinnitus*. They may be due to various conditions quite independent of any actual disease of the auditory apparatus, and only such causes of disordered hearing will be considered in the present article as are not due to changes in the conducting portion of the ear, which can be demonstrated by the different methods of examination, or to recognised affections of the nervous apparatus connected with hearing. These will be found discussed under the article EAR, Diseases of.

(a) *Partial or complete loss of hearing.*—Perhaps the deafness due to accumulations of cerumen, which so frequently interfere for a time with the hearing of persons whose ears are free from disease, should be regarded as disordered hearing, rather than as a symptom of a pathological condition. As nothing more energetic than careful syringing is required to remove such obstructions, it will be sufficient to observe that in this proceeding the nozzle of the syringe should be directed along the roof of the external canal. Amongst a very large number of people with the organs of hearing in an apparently healthy state, some few will be found upon whom, throughout their lives, certain notes produce no response. They will not, for example, be able to hear the sounds made by grasshoppers, or the singing of some birds—the call of a partridge, for instance; and in most persons, as age advances, the very high notes are lost. To prove this, it is only necessary to blow one of Mr. Galton's whistles in a room full of people, when a considerable proportion of the assembly will fail to catch the high notes, which are distinctly heard by the rest; and although this failure is also noticeable in many nervous affections, all other sounds will perhaps be heard quite normally by these individuals. Emotional influences play a very large part in the destruction or suspension of hearing, and this is especially observable in the case of women. The unexpected sight of a dead husband, hearing of the death of a dear friend, the proposal of a severe surgical operation on a relative, a quarrel, an alarm of thieves, and witnessing a carriage accident, have each within the knowledge of the writer been followed by intense and sudden deafness, which has only been partially recovered from. The same effect has been noticed

with men who have been subjected to prolonged mental strain, in connexion with literary work, or during commercial crises. It has been recorded that a deaf-and-dumb child has suddenly recovered hearing, after the discharge from the bowels of eighty-seven lumbrici and a large number of oxyurides (*Journ. of Med. Soc.*, 1844). Complete loss of hearing, extending over several months, was on one occasion followed by perfect hearing in a girl of fifteen, on the first appearance of menstruation. The temporary effect of quinine and salicylic acid on the hearing is well known, but when quinine has been administered in large doses, and for a long period, this special sense is not unusually injured permanently. Amongst the diseases which often induce a lasting deafness, without any perceptible local change in the conducting portion of the ear, may be included mumps, many of the fevers, and diphtheria; for although in the two latter instances the middle ear often suffers, this is not always the case, and the immediate cause of the deafness must be sought in the products of inflammation which have been left within the cranium. The same explanation is probably also the correct one in those instances where children lose for ever all hearing power after cerebral excitement or congestion. Habitual and obstinate constipation is sometimes attended with loss of hearing, which returns after the action of purgative medicine. A clot of blood within the cranium, whilst causing hemiplegia of the opposite side, may destroy the hearing on the same side as the effusion; and a case is on record in which closure of the cerebro-spinal foramen gave rise to this symptom.

(b) *Exalted hearing*.—What is termed 'exalted hearing' will generally, on careful examination, be found to be not so much a definite change in the capacity of the hearing apparatus to receive impressions, as an inability on the part of the patient to receive such impressions without an undue effect on the nervous centres being produced. Thus in many inflammatory states of the brain or its membranes this symptom is often a prominent one. It is also not uncommonly met with in hysterical and nervous persons.

(c) *Perverted hearing*.—Attendant on most of the above examples, and closely allied to deafness, is the often persistent tinnitus; but there are conditions in which this distressing symptom is the chief and solitary trouble. Thus tinnitus, with a feeling of pulsation in the ear, is occasionally the first warning of an intracranial aneurysm; whilst a furious tinnitus and the hearing of strange noises sometimes precede an attack of acute mania. Patients who have been the subjects of malarial fevers and sunstroke often complain of tinnitus; and as in all cases of disease of the ears, when present, it is the most intractable of symptoms, so it is when the ears

have not been the seat of any malady or injury.

**TREATMENT**.—Since all the above states of disordered hearing may strictly be said to be due to causes which are in themselves abnormalities of one part or another of the organism, it is to these that the treatment will naturally be directed rather than to alterations in hearing which in truth are merely symptoms. See EAR, Diseases of; and TINNITUS. W. B. DALBY.

**HEART, Diseases of**.—The study of this class of diseases has reference to the immediate pathological changes which occur in the heart itself, and to the consequences or results of these changes upon its function, that is to say, upon the circulation of the blood. The latter portion of the subject will be found discussed in the articles which have reference to disorders of the special organs of circulation; and it will therefore be necessary in this place to summarise only the morbid changes which affect the heart itself, and this merely as an introduction to the full description of those changes contained in the following articles, or which will be found in other parts of this work.

1. The heart may be *displaced, misplaced, or malformed*. 2. Its various textures, including the coverings, the lining membrane, the valves, and the walls, are liable to *acute and chronic inflammation* and their effects. 3. The organ itself as a whole may be increased in size, either by general *dilatation* of one or of more of its cavities, or partially, as by *aneurysm* of the walls; or by an addition to its volume, by *hypertrophy* of its muscular structure, of the *fatty* tissue which exists beneath the pericardium, or of the *connective tissue* which binds the muscular fibres together. 4. Its volume may be diminished by simple or general *atrophy*; or by the walls of one or more of its cavities being wasted and thinned. 5. Its walls are liable to various forms of *degeneration*—more especially *fatty, granular, calcareous, and pigmentary*. 6. They may be the seat of *fibroid disease*, and of various *morbid growths*, such as *cancer, tubercle, syphilitic formations, or hydatids*. 7. *Congestion* and *hæmorrhage* may occur in the walls of the heart. 8. These are liable also to such injuries as *rupture*—whether spontaneous or as the result of violence; and to various kinds of *wounds* and their effects. 9. The nervous apparatus of the heart may be affected, as in *angina pectoris*. 10. And, lastly, the reader will find discussed under the head of *functional disorders* of the heart, certain disturbances in its action and sensibility which cannot be clearly referred to any distinct lesion. RICHARD QUAIN.

**HEART, Abscess of**.—See HEART, Inflammation of; and HEART, Pyæmic Abscess of.

**HEART, Aneurysm of.**—DEFINITION.

A simple depression or a sacculus formed in the walls of the heart, and communicating by an orifice, varying in size, with one or more of its cavities.

The term 'aneurysm of the heart' has not been always used in this sense. It was first applied by Lancisi and subsequently by Bouilleau to any dilatation of the heart, whatever its cause or its character. Cases of the disease, as the term is now understood, were published by Galeatti and John Hunter in 1757. In this country, Dr. Thurnam, Dr. Peacock, more recently Dr. Wickham Legg, and others have treated the subject fully. In France aneurysm of the heart has been described specially by Breschet and by Pelvet; whilst in Germany, Lobstein, Löbl, and Hartmann have written upon it at length. A full account of the researches of these and several other writers will be found in the work of M. Pelvet—*Des Anévrysmes du Cœur*, Paris, 1867—and in the Bradshaw Lecture at the Royal College of Physicians by Dr. Wickham Legg.

**ÆTIOLOGY AND PATHOLOGY.**—The essential condition which leads to aneurysm of the heart is a change in a portion of the heart's texture, by which the resisting power of the affected part against the pressure of the blood from within the cavity is diminished. Under such circumstances a simple depression, corresponding to the weakened spot, may be first formed on the inner surface of the heart. This gradually extends through the cardiac wall towards the external surface, where the resistance becomes less, and where a pouch or sac is then formed, communicating with the cavity of the heart, it may be by a neck. The weakened condition referred to is attributable in different instances to inflammation of the substance of the heart, whether acute or chronic; to syphilitic or other growths; to fibroid and to fatty degeneration.

(a) *Inflammation* of the heart, affecting either the endocardium or the substance of the heart itself, may lead to softening and ulceration; and both conditions have been found in connexion with aneurysm. Inflammation may also lead to the formation of pus in the walls of the heart; and cases are recorded in which, the contents of the sac thus formed having been discharged into the circulation, the cavity became converted into an aneurysmal pouch (Dr. Wilks, *Path. Soc. Trans.*, vol. xii.) Cases of aneurysm of this character may be regarded as originating in acute inflammation of the heart.

In a still larger number of cases, in which the endocardium and pericardium, as well as the muscular walls, are involved, we find a development of fibroid tissue—a cirrhosis, as it were, of the wall of the heart, as the result of chronic inflammatory action, and as some writers suppose of disease of the coronary

arteries. In these cases the fibroid tissue is stretched at each systole of the heart, and it returns less and less to its former dimensions, owing to its want of elasticity. Thus by degrees the portion of the heart affected yields and is pushed outwards, forming a sac of a more or less globular shape.

(b) *Growths* in the heart, whether syphilitic or tuberculous, undergoing the process of softening, may lead to the formation of aneurysm, as in the conditions just described.

(c) *Fatty degeneration* may give rise to the formation of aneurysm. First, a circumscribed portion of softened tissue in the wall of the heart, the result of fatty degeneration, yields without rupture to the pressure of the blood from within, and thus allows of the formation of an aneurysmal pouch. Secondly, partial rupture may take place in the muscular wall, and hæmorrhage occurring at this point, constitutes what is called 'cardiac apoplexy' (see HEART, Hæmorrhage into the Walls of). The clot thus formed undergoes the changes usual in extravasated blood; and a cyst results, which may ultimately communicate with one of the cavities of the heart. Meriedec Laennec, who wrote on aneurysms of the heart, believed that this form of disease was almost exclusively thus developed.

With reference to the relative frequency of the various causes of cardiac aneurysm just enumerated, the writer finds that out of a total of 56 cases, the histories of which were collected by himself, in 21 the walls had undergone fibroid changes, which may have occurred before or during the progress of the aneurysmal formation; in 6 there was fatty degeneration; in 3 the disease was the result of ulceration; in 2 cases it appeared to have originated in abscess; and in 24 the materials were not sufficient for arriving at an accurate conclusion.

*Age.*—With regard to the age at which this disease occurs, in 62 cases collected by the writer the oldest was eighty-two, the youngest a child of twelve. Three cases occurred between ten and twenty years of age, 10 between twenty and thirty, 14 between thirty and forty, 10 between forty and fifty, 7 between fifty and sixty, 10 between sixty and seventy, 6 between seventy and eighty, and 2 between eighty and ninety years of age.<sup>1</sup>

*Sex.*—Of 62 cases, 45 were males and 17 were females.

**ANATOMICAL CHARACTERS.**—Keeping in view the several conditions under which aneurysm of the heart can occur, we may expect to find a corresponding variety of morbid appearances. On laying open the pericardium in cases in which aneurysm of the heart exists, adhesions which are more or less universal, or which may be limited

<sup>1</sup> Sacs connected with the cavities of the heart have been found at birth occasionally. They must be regarded as malformations.

to the seat of the disease, are very frequently found. The heart itself is generally enlarged; and where the aneurysm projects externally it is altered in shape, so much so in some instances that the organ looks like a double heart. The sac may project from the walls as a rounded or conical tumour; or, as in an instance that came under the notice of the writer, it may assume the appearance of an elongated sac winding round the base of the aorta. Again, nothing abnormal may be observed until the heart is laid open, when a depression or an opening may be discovered in the walls of the ventricle, or in the septum. In some instances more than a single pouch is formed: there may be several, each communicating with the cavity of the heart by a separate or by a common opening. The size of the tumour may vary from that of a bean to that of an average-sized cocoa-nut. The opening leading into the pouch may be the widest part of the sac, the aneurysm being a mere depression like a watch-glass or half an egg; or there may exist a constricted or defined neck, leading to a tumour bulging from the walls. The size of the opening may vary from a couple of inches across, to one capable of admitting only a probe. The neck is, in a few cases, described as hard and cartilaginous; in others, as being smooth and regular, or jagged and irregular. The walls of the tumour may be formed by the dilated and thin walls of the heart; or by the walls considerably thicker than natural, and altered in texture. A very usual condition to find is that the walls of the sac consist, proceeding from within outwards, of endocardium; of fibroid tissue, with or without portions of muscular tissue; and of pericardium. This condition was described as occurring in 11 cases. In 8 cases the walls were said to consist only of the endocardium and pericardium, with fibroid tissue between; but it was found that at the base of the tumour, in all these cases, all the layers of the heart-tissue were present, and that it was only towards the apex of the swelling that the muscular layers disappeared. In 3 cases the walls of the aneurysm were said to be composed of a thin membrane, which appeared to consist of endocardium and pericardium only. In 3 cases the walls were of cartilaginous consistence, with bony plates interspersed in the tissue. The thickness of the walls of the sac varied from that of paper to three lines or more. In 14 cases the wall of the tumour was strengthened by an adherent pericardium. The aneurysmal cavity in the majority of cases was lined by smooth membrane; but in a few instances, apparently of acute formation, the walls consisted of muscular fibres, torn and separated by the blood which had been extravasated amongst them. The contents of the sac are generally in the form of blood-clots or layers of fibrin,

the outermost layers of which may be more or less organised and adherent to the wall.

*Seat.*—Of the 56 cases already alluded to, 52 were in the left ventricle, 3 in the right ventricle, and 1 in the right auricle. Of the 52 cases in which the aneurysm was situated in the left ventricle, 22 occupied the apex, 11 the base, and 17 were in intermediate situations. Several cases have been recorded in which the aneurysm was situated in the muscular septum between the ventricles; in the ‘undefended space;’<sup>1</sup> or at the base immediately below the aortic valves. A case of the last-named form was recorded by the writer in the third volume of the *Transactions of the Pathological Society*. These two last forms are generally associated with endocardial inflammation and ulceration.

*SYMPTOMS.*—In 13 of the 56 cases referred to, the aneurysm was not discovered until after death; no mention being made of signs or symptoms of its previous existence. In the remaining cases, symptoms, more or less marked, indicative of heart-disease, were present. These symptoms were chiefly—pain, dyspnoea, lividity of the surface, palpitation, and irregularity of the pulse. In 10 cases murmurs were heard, accompanying or replacing the sounds of the heart. We thus see that the symptoms of aneurysm are such as may exist in common with other lesions of the heart; and it is extremely doubtful, except in presence of some special circumstances indicative of this condition, whether we have at our command the means of diagnosing the existence of cardiac aneurysm. The writer not long since saw, with Dr. Holman, late of Reigate, a case of grave heart-disease, in which extended dulness to the left, and below the usual situation of the apex-beat, with a feeble impulse in the same situation, led to a suspicion of the probable existence of cardiac aneurysm.

*PROGRESS, DURATION, AND TERMINATIONS.*—Pathological testimony fully justifies the inference that certain cardiac aneurysms—such as those which originate in inflammatory softening, ulceration, or the opening of cysts into the cavities of the heart—are acute in their formation. But the like evidence further testifies that the formation of most other aneurysms, and the progress of all, are of a slow or chronic character. Still it would seem to be difficult, if not impossible, to determine the duration of the disease in any given instance, inasmuch as many cases, for example, have been found in the *post-mortem* room, which had not given rise to any special symptoms; whilst in other cases the pre-existence of heart-disease before the formation of aneurysm rendered it equally impossible to fix a date for the development of the latter special disease.

<sup>1</sup> A case of this character, apparently congenital, was described by Dr. Angel Money in a child five years old. See *Path. Soc. Trans.*, vol. xxxviii. p. 97.

Death may result from the disturbance of the heart's action, induced by the presence and the extent of the disease; from the aneurysm opening into the pericardium; or from its burrowing in the wall of the heart, and opening into another cavity of the organ different from that in which it originated. Lastly, one or two cases are recorded in which a cure of the disease had apparently been effected by the walls of the sac becoming indurated or calcified.

**DIAGNOSIS AND PROGNOSIS.**—Seeing how extremely obscure the clinical history of these cases is, it would be impossible to speak more definitely either as to the diagnosis or the prognosis of the disease than has been done under the preceding head.

**TREATMENT.**—The treatment of cardiac aneurysm must be such as would be adopted in any other form of grave heart-disease, and according to the special circumstances of, and indications in, each case. We can only seek to mitigate the more urgent symptoms, whether local in the heart itself, or more generally affecting distant organs.

RICHARD QUAIN.

**HEART, Apoplexy of.**—See HEART, Hæmorrhage into the Walls of.

**HEART, Atrophy of.**—**DEFINITION.**—A diminution in the size and weight of the heart as a whole; or a diminution in size of one part of the heart in relation to the whole organ.

**ÆTIOLGY.**—The causes of atrophy of the heart are either *general* or *local*. With respect to the *general* causes of atrophy, the heart is found reduced in volume together with the other organs of the body, in cases of marasmus, of phthisis, of syphilis, cancer, &c. Probably one of the smallest hearts on record—one which weighed but  $3\frac{1}{8}$  ounces—was found by Dr. Church in the body of a woman aged forty-seven, who died of cancer of the pylorus, after an illness characterised by 'gradual starvation' of more than seventeen months' duration. With reference more especially to phthisis, as affecting the size of the heart, an analysis of 171 cases, made by the writer at the Brompton Hospital, showed that this organ was below the average weight in 54·4 per cent. Diseases of a sub-acute character, such as typhoid fever when protracted in its course, may lead likewise to wasting of the heart. The heart is also occasionally congenitally small.

The *local* causes of atrophy of the heart are chiefly two—namely, (1) pressure by pericardial adhesions upon the heart, by mediastinal tumours, by fatty growth beneath the pericardium, and other like conditions; and (2) interference with the circulation in the coronary arteries, as in the conditions just enumerated, or as a result of malformation or of disease of the vessels themselves.

Partial atrophy of the heart, when it occurs,

may be said to be referable to insufficient blood-supply from vascular disease or from local pressure; or from fatty infiltration.

**ANATOMICAL CHARACTERS.**—The heart in simple atrophy presents a general uniform diminution in size, as regards both its walls and its cavities; and in its weight. In local atrophy, a portion of the cardiac wall, more or less extensive, or of one of the divisions or cavities of the heart, may be found to be below the ordinary dimensions. The colour of the atrophied heart may be normal; it is frequently pale; and it is occasionally of a deep reddish-brown. The pericardium, not shrinking proportionately with the muscular substance, may present a puckered, opaque, and cedematous aspect, 'like a withered apple' (Laennec); and for the same reason the coronary vessels may be tortuous and prominent. The consistence of the walls is generally firmer than natural; and the muscle may be even tougher, except where the atrophy is due to the presence of fat, in which case the fibres are friable, and on microscopical examination present the appearances of fatty degeneration. In *simple* atrophy of the heart, the muscular fibres undergo diminution in volume; and they may also be actually reduced in number. Atrophy of individual muscular fibres is also found as the result of interstitial fatty or fibroid growth; and this when extensive has been somewhat erroneously named '*yellow atrophy*.' Another variety, which is most frequently found in the marasmus of old age, is known as *brown atrophy* of the heart. In such cases the muscular tissue is of a dark or dirty reddish-brown colour, which proves on microscopical examination to be due to the presence of numerous shining yellow or brown pigment-particles within the muscular fibres, and specially abundant either around the nuclei or between the ultimate fibrillæ.

**SYMPTOMS.**—The symptoms and signs of atrophy of the heart are those which might be expected to result from diminished size and power of the organ. The characteristic phenomena are those of feeble circulation. The physical signs are chiefly diminished præcordial dulness; a feeble impulse, the apex-beat being within and above the usual situation; diminished area of audible sounds; and a small, weak pulse.

**DIAGNOSIS.**—The above signs and symptoms, in association with general wasting, afford sufficient grounds for the diagnosis of atrophied and feeble heart. Emphysema, pericardial effusion, and other causes of diminished cardiac dulness and weakness of impulse, must be excluded by the ordinary modes of investigation. There are no special means by which *partial* atrophy of the heart can be diagnosed, except, possibly, that the presence of this condition may be assumed where the functions of the organ are disturbed in the absence of valvular or other of the

more common forms of cardiac disease, sufficient to explain the symptoms.

**TREATMENT.**—The treatment of atrophy of the heart is the treatment of the primary disease upon which it depends, so far as is possible.

RICHARD QUAIN.

**HEART, Calcification of.**—See HEART, Degenerations of.

**HEART, Cancer of.**—See HEART, Morbid Growths in.

**HEART, Cirrhosis of.**—See HEART, Fibroid Disease of.

**HEART, Congenital Misplacement of.**—**SYNON.**: *Ectopia Cordis* (Breschet); *Ectocardia* (Alvarenga).

The heart is occasionally found to occupy a wrong position, and such misplacement may be either within the cavity of the thorax, or external to it.

1. Of the *internal* malpositions—*ectopia cordis intrathoracica* or *ectocardia intrathoracica*—the most common is that to which the term *dextiocardia* has been applied, in which the heart is in a very similar position on the right side of the chest to that which it should occupy on the left. This condition may coexist with transposition of the other viscera of the body, or it may occur alone. Instances of the former kind have long been placed on record, cases having been met with in Rome in 1643, in Paris in 1650, and in London in 1694. When the heart is misplaced, the aorta generally follows an irregular course, crossing the right bronchus and passing down to the right side of the bodies of the vertebræ; and the right carotid and subclavian arteries are given off as separate vessels, while the brachio-cephalic trunk is situated on the left side. In some instances, however, the vessels at the arch are not transposed; whilst in others the aorta, after passing over the right bronchus, crosses the spine and follows its usual course to the left of the bodies of the vertebræ. In cases of transposition the heart itself may be well-formed; or it may be very imperfectly developed.

In another kind of misplacement, *mesocardia*, the heart is situated more in the median line than natural—a position which it occupies in the fœtus at the earlier periods. Cases have also been recorded in which the organ occupied a transverse, and an antero-posterior direction.

2. Of the *external* misplacements, those in which the heart is situated external to the thoracic cavity—*ectopia* or *ectocardia extrathoracica*—the most common is that in which, from deficiency of some part of the sternum, the organ lies in front of the chest—*ectopia cordis* or *ectocardia pectoralis*. In other cases, from deficiency in some portion of the diaphragm, the heart is placed in the abdomen, either lying in the cavity, or

if the integuments are partially defective, in a sac in the præcordia—*ectopia cordis* or *ectocardia abdominalis*. In a third form the heart lies at the root of the neck—*ectopia cordis* or *ectocardia cephalica*. Of these forms, examples are related or referred to in the memoirs of Breschet and Alvarenga, and various others have been published since the appearance of the memoir by Breschet.

**SYMPTOMS, DURATION, AND TERMINATIONS.**

—When the heart is well-formed, its malposition within the thorax does not necessarily cause such interference with its functions as to be productive of symptoms, or materially to curtail the duration of life. Indeed, cases are on record in which the heart and other viscera have been transposed in persons who had never presented any signs of disorder of the circulation, and who lived to very advanced ages. When, however, the organ is also defective, and especially when the displacement is external to the thoracic cavity, life is usually only of short duration—though some remarkable cases of external displacements are on record, in which the patients survived to advanced ages.

**Malformation of the Pericardium.**—

Closely allied to the cases of misplacement of the heart are those in which the organ, though occupying its natural position, is not covered by the pericardium, but lies in contact with the lung in the left pleural cavity. Of this form of anomaly various instances are recorded—the first undoubted case of the kind being probably that represented by Dr. Baillie, in 1778. The defect seems to consist in the pericardium, which is apparently reflected from the external coat of the aorta, not being prolonged so as to cover the front of the heart and become attached to the diaphragm. The imperfectly developed membrane is represented by a kind of loose fold, or pocket, which is found on the right side or upper part of the heart.

**EFFECTS.**—This condition does not seem materially to interfere with the functions of the organ. Cases are recorded in which the subjects lived to middle age; and the writer has himself seen it in a man who died of heart-disease at seventy-five.

T. B. PEACOCK.

**HEART, Congestion of.**—Attention

was first directed to this morbid condition of the heart by Sir William Jenner (*Med.-Chir. Trans.*, vol. xliii., p. 199). The coronary veins, like the veins of other parts, are subject to engorgement when the flow of the blood from them into the right auricle is interrupted. The most common cause of this interruption is dilatation with distension of the cavities of the right side of the heart, which conditions are themselves usually due either to emphysema or to valvular disease of the heart. Disease of the

trunks of the coronary veins and pressure upon them may be regarded as less frequent causes of cardiac congestion.

*Post mortem*, congestion of the heart is recognised, when recent, by fullness of the veins on the surface of the organ; by œdema of the loose connective tissue at the base; and ecchymosis of the pericardium and endocardium. The pericardial sac contains some serous or sero-sanguinolent effusion; and the mouth of the coronary sinus may be found to be dilated. When the congestion is slight, gradually developed, and of long standing, the venous fullness gives rise to an increased formation of connective tissue in the walls of the heart, which become, in consequence, tough and indurated; whilst the dilatation of the cavities, with which the congestion is associated, is rendered permanent by the same cause. When divided with the knife, the cardiac walls do not fall inwards; their substance feels like a piece of leather; and the section has a smooth homogeneous appearance. Microscopically, the connective tissue seems to be increased in quantity; and the muscular fibres are in a condition of granular, fatty, and pigmentary degeneration.

Congestion of the heart possesses no direct clinical relations; but its existence should never be overlooked in estimating the effects on the circulation of mechanical congestion, which must impair the functional activity of the heart in the same way as it impairs that of the liver, stomach, and kidneys.

J. MITCHELL BRUCE.

### HEART, Connective-Tissue Hypertrophy of.—SYNON.: False Hypertrophy.

**DEFINITION.**—An excessive development of the connective tissue which exists between the muscular fibres of the heart, causing an increase in the size of the organ.

**ANATOMICAL CHARACTERS.**—In connective-tissue hypertrophy the heart is enlarged more or less uniformly as regards the walls of its several cavities, and usually to a great extent, the organ weighing in some instances as much as forty ounces. The thickness of the walls is increased, as in simple or true hypertrophy; their density and consistence are such as to present a firm, tough, leathery character. When cut the edges do not collapse, but continue stiff and prominent. The colour of a heart in this condition may vary from pale buff to deep purple, according to the amount of connective tissue, and of blood present in the vessels. Microscopically there is seen—not the usual limited amount of intermuscular fibrillar tissue and connective-tissue cells, but a decided hyperplasia of these elements, in the form of connective-tissue, of which all stages of development may sometimes be observed, from the round and spindle-shaped cell to the complete bundle of fibrille. Between the individual

bundles of this tissue lie the muscular fibres, which are also hypertrophied, but which are more or less compressed, and are occasionally in a condition of granular or fatty degeneration (fig. 59).

There is a certain amount of anatomical resemblance, but a very clear pathological distinction, between this form of heart-disease and the change described by Sir William Jenner as fibroid disease of the heart resulting from congestion (*see* HEART, Congestion of). Connective-tissue hypertrophy may also to some extent be compared with the fibrosis described by Sir William Gull and Dr. Sutton as existing in the walls of arteries and other tissues.

**ÆTIOLOGY.**—Cases of hypertrophy of the heart have been described by several writers which were remarkable for the absence of disease of the valves, or of the blood-vessels, or of the kidneys, which, if present, could explain its origin, but which the writer believes were due to the morbid change above described, and not to hypertrophy of the muscular fibres alone. A remarkable specimen is preserved in the museum of St. George's Hospital, consisting of a heart weighing forty and a-half ounces, which was removed from the body of an under-butler. In the *post-mortem* examination of whom nothing was found which could satisfactorily explain the occurrence of the enlargement.

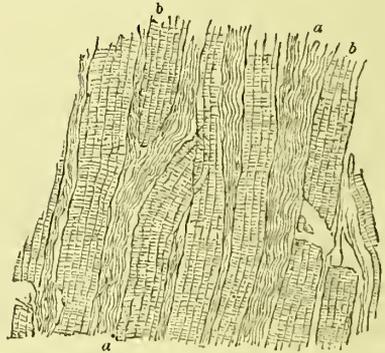


FIG. 59.—Connective-tissue Hypertrophy of the Heart.—*a a*, Connective-tissue fibrillæ. *b b*, Muscular fibres.  $\times 250$ .

The writer is indebted to Dr. Whipham for an opportunity of examining this specimen, which was found by his friend, Dr. Mitchell Bruce, to possess the microscopical characters above described and figured (fig. 59). This and similar cases exhibit no appearance of chronic inflammatory action, and thus differ altogether from examples of that form of fibroid degeneration which is described under a separate heading (*see* HEART, Fibroid Disease of). In the cases now described there is a simple hyperplasia of connective tissue, the origin of which cannot be fully explained. In Germany a similar enlargement of the heart

is said to have been found more especially in *gourmands*, and hence it derived a characteristic appellation. The existence of connective-tissue growth being thus determined, the effect of its presence on the muscular tissue is obvious. The connective tissue, surrounding, as it must do, the muscular fibres, interferes with their free action, to overcome which there will be a natural tendency to increased action, and consequent hypertrophy of the muscular fibres themselves. It is very probable that it is to these two processes going on simultaneously that the great increase in the size of the heart is due.

**SYMPTOMS AND DIAGNOSIS.**—In a remarkable case recorded by the late Dr. Hyde Salter, which the writer believes to have been of the nature here described, acute or severe cardiac dyspnoea and hæmoptysis, from which the patient had suffered for several weeks, were the most prominent symptoms. The heart-sounds were natural, except that the first was dull and defective. The pulse was 84. The symptoms increased in severity, and were aggravated by excessive epistaxis. The patient died after being in hospital for fourteen days. At the *post-mortem* examination the heart was found to be of great size, and there was no disease either of the valves or of the vessels or of the kidneys to account for this. In the case of the butler in St. George's Hospital, it is recorded that he continued going about until within a few days of his death. These and some like cases indicate that we can do little in the way of diagnosis beyond recognising the presence of cardiac hypertrophy by the usual signs; and if the hypertrophy be considerable, and if there be no valvular disease and no kidney-disease, we might not be far wrong in considering that the hypertrophy was caused by increase of some other element as well as that of the muscular fibres.

**TREATMENT.**—If the opinion be correct that this form of disease finds its origin in excessive alimentation, it would be well to place the patient in such circumstances as would prevent this, giving attention at the same time to other hygienic conditions. The more aggravated symptoms of cardiac disease must be treated on general principles.

RICHARD QUAIN.

**HEART, Coverings of, Diseases of.**  
See PERICARDIUM, Diseases of.

**HEART, Degenerations of.**—The degenerations that affect the heart may be enumerated as follows: (1) Fatty; (2) Parenchymatous; (3) Albuminoid; (4) Pigmentary; (5) Cartilaginous; (6) Calcareous; and (7) Vitreous. The condition which has been called 'fibroid degeneration' of the heart is described under the heads of HEART, Fibroid Disease of; and HEART, Syphilitic Disease of.

1. **Fatty.**—This form of degeneration of the heart being of special importance is

discussed in a separate article. See HEART, Fatty Degeneration of.

2. **Parenchymatous.**—**SYNON.**: Granular Degeneration; Cloudy Swelling; ? Parenchymatous Inflammation.

**ÆTIOLOGY.**—This form of degeneration of the heart is generally met with in the acute specific fevers, especially typhus, and typhoid fever, diphtheria, and septicæmia; and is probably referable to the action either of the poison or of the high temperature attending the disease-process upon the muscular substance. See DEGENERATION.

**ANATOMICAL CHARACTERS.**—The disease generally attacks the heart as a whole. The organ appears somewhat enlarged, extremely soft—flabby as well as friable, and of a dirty greyish-red colour. The pericardium is ecchymosed, dull, and swollen, and the epicardial fat has more or less completely disappeared. Microscopically, the muscular fibres are found to be dull and granular, swollen, and variously ruptured; their striations are indistinct; and the addition of acetic acid removes many of the granules from the fibres, whilst it brings more distinctly into view a few fatty globules, and frequently an increased number of pigment-particles.

**SYMPTOMS.**—Inasmuch as parenchymatous degeneration of the heart is usually but a complication of some acute specific disease, the condition of the patient is one of great febrile prostration with cardiac asthenia. The physical signs, which are regarded as more distinct evidence than the symptoms of the condition of the heart, are—feebleness, advancing to complete absence, of the apex-impulse, or more rarely palpitation; and progressive weakening, and finally loss of the first sound. The pulse has been described as corresponding with the condition of the heart, indeed in some cases it is imperceptible although associated with cardiac palpitation.

**COURSE AND TERMINATIONS.**—The course and terminations of granular degeneration of the heart are inseparable from those of the primary disease. In typhus the average date of the appearance of the symptoms and signs just described is the sixth day of the fever; and they usually cease on the fourteenth day, but a large proportion of cases prove fatal before that time.

**PROGNOSIS.**—The existence of this kind of degeneration of the heart adds seriously to the gravity of a case of fever; and the danger increases with the rate and weakness of the pulse, and the feebleness of the cardiac impulse and first sound. The re-appearance of the first sound under observation justifies a favourable prognosis.

**TREATMENT.**—The treatment of parenchymatous degeneration of the heart is in no respect different from that of the fever in which this condition originates. The appearance of the characteristic symptoms and signs of the cardiac affection is, however, to

be regarded as an important indication for the use of alcoholic stimulants, which are, as a rule, well borne in such cases, and act very beneficially.

3. **Albuminoid.**—This kind of degeneration has been said, with a certain amount of possibility, to have been found in the heart. It is certainly excessively rare.

4. **Pigmentary.**—Pigment-granules, in the form of shining yellow particles, are almost invariably found in the muscular fibres of the heart in chronic cardiac disease. In certain cases of atrophy known as 'brown atrophy,' as well as in the granular degeneration just described, these pigment-particles are decidedly increased in number, and collected towards the axis of the fibres. A somewhat similar appearance is seen in the heart in jaundice.

The condition is of purely pathological interest.

5. **Cartilaginous.**—Portions of the myocardium have frequently been described as 'cartilaginous' or 'fibro-cartilaginous,' but it would appear that in these instances the muscular tissue was replaced by dense firm fibroid tissue only. See HEART, Fibroid Disease of.

6. **Calcareous.**—Calcification of pericardial adhesions is not very rare; and in some of the recorded instances of this condition, plates of the same material have been found projecting into the substance of the heart, appearing as if formed within the myocardium. Besides this class of cases, instances of true deposit of calcareous particles within the individual muscular fibres have been described. The change appears in the form of small, pale, gritty deposits, taking the place of the normal muscular tissue on the surface. On microscopical examination, the muscular fibres are found to have become solid and opaque, whilst hydrochloric acid or sulphuric acid removes the opacity with the evolution of gas, the addition of the latter acid also producing gypsum crystals. It is probable that, in other instances, the calcareous particles are situated outside the muscular fibres, and may be products of a caseous nodule, whether syphilitic or 'tubercular' in origin.

This form of disease appears to possess no special clinical relations.

7. **Vitreous.**—Vitreous, waxy, or colloid degeneration, as described by Zenker, occurs in the myocardium, as it does in the voluntary muscles.

J. MITCHELL BRUCE.

**HEART, Dilatation of.**—DEFINITION. Dilatation of the heart may occur in two forms. In the one it involves only a limited portion of the cardiac walls and constitutes an aneurysm; in the other there is uniform enlargement of one or more of the heart's cavities, and dilatation in the usual acceptation of the word is present. To this latter

condition, however, the names 'aneurysm' and 'passive aneurysm' of the heart were formerly applied. Dilatation is probably always associated with hypertrophy.

ÆTIOLOGY.—The occurrence of dilatation implies that the walls of the heart which yield are too weak to resist successfully the internal pressure to which they are exposed. This defective relation may be due either to actual enfeeblement of the heart's walls, which renders them unequal to the task normally devolving on them; or to excessive blood-pressure, which even the healthily constituted walls are unable to withstand. The enfeeblement of the heart may be a consequence of fatty or other degeneration; or, as is probably more frequently the case, may be inherent but unconnected with visible textural disease. The excessive blood-pressure may be dependent on actual obstruction to the circulation, which the heart is called upon to surmount; or on undue rapidity of action, which (other things being equal) implies an unwonted expenditure of force. As a matter of fact, however, dilatation and hypertrophy are generally if not always associated; and the processes by which these combined conditions are attained are more complicated than the foregoing statement might lead one to suppose. It will be convenient, therefore, to consider certain cases *seriatim*.

1. In obstructive disease at the aortic orifice; in general stricture of the minute systemic arteries, such as occurs in connexion with contracted granular kidneys; and indeed in all cases in which resistance is offered to the free discharge of blood from the left ventricle, progressive hypertrophy of the walls of that ventricle takes place. But the hypertrophy is complicated even from the beginning with dilatation. The hypertrophy, at any rate at first, is simply compensatory, and may be taken as a measure of the excess of resistance which the heart is called upon to overcome. The dilatation, however, is in no sense compensatory, and is probably to be regarded as a measure of the inability of the walls to cope successfully with the extra work required of them. It is, moreover, obvious that the occurrence of dilatation, by increasing the area of resistance to the endo-ventricular blood-pressure, increases *pro tanto* the muscular effort requisite for the propulsion of the blood into the aorta; and by enlarging the capacity of the ventricular cavity and consequently the amount of blood to be discharged from it, on that account also throws additional labour on the muscular walls of the ventricle. Thus the hypertrophy and dilatation react on one another; and the hypertrophy, which was probably at first simply compensatory of the mechanical obstacle to the discharge of the normal contents of the ventricle, ends by becoming—or

rather striving to become—compensatory not only of this but of the virtual weakness of the heart which dilatation entails.

2. In regurgitant disease at the aortic orifice, hypertrophy and dilatation of the left ventricle also take place. But in this case, while the hypertrophy probably reaches a higher degree of development than in simple obstruction, dilatation preponderates from first to last; and the ventricle attains larger dimensions than in perhaps any other form of disease. But to what are the hypertrophy and dilatation due in this case? There is no impediment to the escape of blood through the aortic orifice, and therefore *primâ facie* no need for compensative hypertrophy. There is no doubt that here hypertrophy waits on dilatation. The immediate effects of regurgitation are, that during diastole the ventricle becomes more rapidly and completely filled with blood than it does under other circumstances; that hence the subsequent contraction of the auricle tends to distend it unnaturally with blood; and that during the whole of this period the walls have also to sustain the backward pressure of the systemic arterial blood. The result is that, on the principles above enunciated, the walls of the ventricle have to encounter a larger area of pressure, and to expel a larger amount of blood, than natural, and hence are called upon to make excessive effort, and hypertrophy ensues. Thus the tendency to dilatation causes the tendency to hypertrophy; and both, acting continuously, promote the progressive increase in the capacity of the ventricular cavity and in the thickness of the ventricular walls.

It is probable in both cases, but more especially in the latter of them, that ere long the ventricle fails to expel the whole of its contents into the aorta at each contraction, and that the retention of this residual blood becomes an important factor in promoting dilatation.

3. The effects of continued violent action of the heart, whether caused by nervous influence or by muscular effort, are much the same as those of obstructive disease. For both increased rapidity of contraction and increased amount of blood to be expelled at each beat (other things being equal) imply increased expenditure of force; and the persistence of either or both of these conditions, therefore, the supervention of hypertrophy and dilatation.

4. The above discussion relates especially to dilatation and hypertrophy of the left ventricle. But, *mutatis mutandis*, it applies with equal force to dilatation and hypertrophy of the other sections of the heart. Thus, in mitral valve disease, the left auricle undergoes hypertrophy and dilatation—the dilatation preponderating in regurgitant disease of the valve, the hypertrophy preponderating in obstructive disease.

5. In pulmonic valve disease the right ventricle becomes hypertrophied and dilated—the dilatation being greatest where there is pulmonic regurgitation, the hypertrophy being greatest where the disease is obstructive.

6. In tricuspid valve disease the right auricle suffers, becoming chiefly dilated in the presence of tricuspid regurgitation, chiefly hypertrophied when there is obstruction. And thus, also, just as when the systemic circulation is impeded the left side of the heart suffers, so when the pulmonic circulation is obstructed, the right side of the heart undergoes enlargement.

In all cases, therefore, hypertrophy and dilatation seem to result concurrently; but whether the one or the other condition preponderates, depends partly on the particular nature of the cause to which the hypertrophy and dilatation are due, partly on the inherent strength or weakness of the cardiac walls. In all cases, too, the other cavities of the heart, besides that primarily and directly implicated, suffer according to their position from the effects of the greater or less work which sooner or later is cast upon them. It may particularly be pointed out, that cavities situated behind the one primarily implicated tend sooner or later to share in its hypertrophy and dilatation, and that in cases of obstructive disease of either of the auriculo-ventricular orifices the corresponding ventricle, in consequence of receiving less than its due amount of blood, and of thus having less work thrown upon it, tends to undergo atrophy.

The temporary dilatation which is described as occurring in acute febrile disorders, such as typhus, is due mainly to enfeeblement of the cardiac walls.

ANATOMICAL CHARACTERS.—In dilatation of the heart, the cardiac walls may be either thinner or thicker than natural, or may retain their normal thickness. It is a question, however, whether, excepting in the case of partial dilatation or aneurysm, dilatation ever takes place independently of hypertrophy; for even as regards the auricles, where dilatation with attenuation is chiefly observed, there is reason to believe that the attenuation is not commensurate with the extension which accompanies it, and consequently that the total bulk of muscular tissue is increased. When dilatation is associated with no apparent change in the thickness of the walls, hypertrophy is of course present.

It must be mentioned, however, that it is often very difficult to determine on *post-mortem* examination the true relation between the thickness of the cardiac walls and the capacity of the cardiac cavities. For their apparent relation is largely dependent on the condition of the cavities as to systole or diastole at the moment of death; and

on the state of the heart as to cadaveric changes at the time of *post-mortem* examination.

The form which the heart assumes in dilatation is the same as that which it assumes in hypertrophy; and indeed, as the two conditions are probably always associated, it is needless to endeavour to establish any distinction between them in this respect. If the dilatation be general, the form of the heart remains unchanged, but its size is uniformly augmented. If the left ventricle be mainly affected, the heart appears not only enlarged but elongated, the left ventricle taking more than its due share in the formation of the cardiac apex. If the right ventricle be specially implicated, the heart becomes enlarged in its transverse diameter; it is more rounded in its contour as seen from the front than it should be; and its apex is obtuse, and either bifid, from the fact that the apices of both ventricles take an equal share in the formation of the cardiac apex, or formed wholly by the right ventricle. If the auricles be dilated, they constitute large masses on both sides of the root of the aorta and pulmonary artery.

The walls of the dilated heart vary not only in thickness, but also in quality. Thus they may be preternaturally firm or preternaturally soft; they may be healthy in structure, or may present more or less degenerative change.

**CONSEQUENCES OF DILATATION.**—Dilatation of the ventricles, especially if it be considerable, is apt to disarrange the mechanism of the auriculo-ventricular valves. It was shown many years ago by Mr. Wilkinson King that even in mere temporary distension of the right ventricle a kind of safety-valve action of the tricuspid valve took place, in consequence of which regurgitation of blood was permitted from the ventricle into the auricle. And since his time it has been clearly demonstrated, both by clinical and by *post-mortem* evidence, that established dilatation of the right or left ventricle is liable to be attended with persistent regurgitation of blood through the corresponding auriculo-ventricular orifice. The defaulting valve under these circumstances has a natural aspect; but careful examination shows either that the orifice has undergone dilatation in company with the ventricle—the valve itself presenting no corresponding increase; or that there is a want of relation between the size of the muscoli papillares and chordæ tendinæ on the one hand, and the capacity of the ventricle on the other, which interferes with the due closure of the valve.

It is obvious that if regurgitation becomes established, the usual consequences of regurgitation will presently ensue—namely, in connexion with affection of the left side of the heart, dilatation and hypertrophy of the left auricle, and subsequently congestion of the lungs and pulmonary apoplexy; and in

connexion with affection of the right side of the heart, dilatation and hypertrophy of the right auricle, fulness of the systemic veins, anasarca, nutmeg liver, and congested, indurated kidneys. It is also obvious that, even if no regurgitant condition be developed, dilatation of heart, which implies feebleness of heart and imperfect circulation, must ultimately induce the ordinary remote consequences of heart-disease. A further consequence of dilatation and other cardiac affections attended with feeble circulation is the formation of thrombi during life, both in the heart itself and in other parts of the vascular system. Mr. Wilkinson King has demonstrated that dilatation of the left auricle may cause compression of the left bronchus.

**SYMPTOMS.**—Since dilatation of the heart rarely, if ever, exists alone, but is associated with hypertrophy, valve-disease, degenerations, and other conditions, it is almost impossible to make any definite statement with regard to the signs and symptoms by which its presence may be recognised. Still there is no doubt that dilatation is one of the most important factors of heart-disease, clinically considered; and that its supervention materially affects the patient's condition, and prospect of life. Dilatation implies weakness, and as a rule over-distension of the implicated cavities with blood, which probably never becomes wholly expelled.

The *physical signs* of dilatation are necessarily in many respects the same as those of hypertrophy. The præcordial dulness is increased in area—the extent and form of this area, and the situation of the apex-beat, being determined by the general size of the heart, and the relative dimensions of its component parts. In proportion, however, as dilatation preponderates over hypertrophy, the impulse of the heart becomes weak, and possibly to some extent diffused. In extreme dilatation, as in extreme weakness from other causes, the sounds of the heart, and especially the first sound, are enfeebled. And it may be asserted that generally the tendency of dilatation is to shorten the first sound, and to give it the characters of the second sound. It has nevertheless been observed over and over again that it is in the concurrence of hypertrophy and dilatation that the cardiac sounds are apt to attain their greatest intensity. The feebleness of the heart's action is generally attended before long by more or less irregularity; and even in the absence of valve-disease, a mitral or tricuspid systolic murmur, implying regurgitation, is apt to be established.

The symptoms of dilatation are to a large extent those of cardiac obstruction, and more especially of mitral disease. The patient complains of weight, oppression or uneasiness in the cardiac region, with probably a sense of fluttering there, and of a tendency

to sighing respiration. He becomes short-breathed, and may have extreme dyspnoea. His face is apt to become livid; his surface pale or ghastly; his extremities cold and blue; and his pulse weak and irregular. Dilatation of the systemic veins arises sooner or later; and subsequently general anasarca, pulsation of the veins in the neck, epigastric pulsation, and pulsation of the liver, together with the other usual consequences of heart-disease. The chief of these are—congestion of the lungs, with pulmonary apoplexy, cough, and expectoration of blood; congestion, enlargement, and tenderness of the liver, with jaundice; and congestion of the kidneys, attended with the discharge of scanty, high-coloured, heavy urine, containing albumen and possibly blood. Other symptoms referable to the nervous and digestive organs, which need not be enumerated here, are also liable to supervene.

The symptoms will vary, of course, according as the left or the right ventricle is mainly affected. In the former case we are liable to have at first irregularity and feebleness of pulse with tendency to faint; then pulmonary complications; and at a later period, symptoms referable to the systemic venous circulation. The latter case is one of considerable interest; because in a large number of instances it is, in its most marked form, a consequence of emphysema of the lungs, or of other analogous conditions, and moreover is apt to come on very rapidly. Under these circumstances, there is necessarily much dyspnoea, but the systemic venous and capillary systems speedily become over-loaded; extreme cyanosis often develops rapidly; and, before long, all the other symptoms referable to disease of the right side of the heart become established: namely, pulsation of the veins in the neck, epigastric pulsation, pulsation of the liver; general anasarca, with perhaps petechial extravasations, jaundice from nutmeg liver, and albuminuria from congestion of the kidneys.

**PROGNOSIS.**—There is no doubt that some degree of dilatation of the heart, and more especially of the right ventricle, may arise either from over-exertion, or from functional disturbances, and in connexion with pulmonary disorders. But such dilatation is for the most part temporary or remediable; and only by continuance of its cause becomes established and a matter of serious importance. In the same way there is no doubt that the dilatation which comes on in the course of organic disease of the heart or lungs, or of other organic diseases which influence the action of the heart, is remediable within certain limits by due attention to the conditions under which it arises. Nevertheless it is certain that the presence of dilatation of the heart in connexion with other diseases, more especially those of the heart

itself, lungs, or kidneys, is a grave source of danger; and that in the great majority of cases it is of fatal omen, aggravating the patient's cardiac symptoms, and hastening his death.

**TREATMENT.**—The treatment of dilatation of the heart merges in that of the other cardiac conditions with which it is associated, and in that of other diseases in the course of which it may have supervened. It may be stated, generally, however, that the treatment is that of cardiac debility, and of distension of the heart with blood.

The chief indications, therefore, are rest of mind and body; avoidance of exposure to cold and wet; the exhibition of ample, nutritious, and readily digestible food; due attention to the action of the bowels, kidneys, and skin; and the employment of medicines likely to regulate and strengthen the action of the heart. For the last purpose digitalis in small doses is universally acknowledged to be of great value. And it is in many cases desirable to combine the digitalis with iron, or some vegetable tonic. Ammonia and other diffusible stimulants are often called for, and are of great service. In cases where there is much lividity, and evidence of stagnation of blood in the right side of the heart, removal of blood by venesection will often prove of great service.

When the dilatation is due to pulmonary disease, this of course requires primary and especial treatment. J. S. BRISTOWE.

**HEART, Displacements of.**—Besides the displacements of the heart that occur as the result of disease, there are certain changes of position which this organ undergoes in health. The most important of these physiological displacements of the heart are—first, its vertical movements in respiration; and, second, the alterations in its situation corresponding with changes in the bodily posture. The present article, however, will deal only with the former class, or abnormal displacements of the heart.

**ÆTIOLOGY.**—The heart may be congenitally displaced—a condition which is described under the head of **HEART, Congenital Displacement of**. These cases being excepted, the causes of displacement of the heart may be arranged in two classes—namely, (1) conditions that exert *pressure*, and (2) conditions that exercise *traction*, upon the heart.

1. The heart is *pressed* or pushed out of position by effusions of fluid—inflammatory, serous, or bloody—into either pleural cavity; by pneumothorax of either side; by intrathoracic tumours—whether mediastinal (including aneurysm and abscess), pulmonary, or parietal; by hypertrophous emphysema, or other causes of enlargement of the lungs; by extensive pneumonic consolidation; or by abundant pericardial effusion of any kind. Certain conditions of the abdominal contents

produce a similar effect—for example: gaseous distension of the stomach and intestines; enlargement of the liver and other solid organs; abdominal tumours of all kinds; the pregnant uterus; and ascites, when considerable. Hernia of the abdominal viscera through the diaphragm, and abscesses connected with the diaphragm, also cause displacement of the heart.

2. The heart suffers *traction*, or is drawn out of position, during absorption of pleuritic effusion with imperfect expansion of the lung, on either side; by the contraction of pleuro-pericardial adhesions, of pulmonary cirrhosis, or of cavities in phthisis; in collapse of either lung from pressure on the main bronchus; and in some forms of deformity of the chest from curvature of the spine.

**MECHANISM OF DISPLACEMENT.**—The causes just enumerated constitute in each instance what may be called the *displacing force*. When this force belongs to the first or *pressure* class, it acts against the surface of the pericardium and heart that is opposed to it, and presses or pushes it, *a tergo*, away from its own seat, in the direction of least resistance. Thus the heart is, speaking broadly, pushed towards the left by effusion into the right pleural cavity; towards the right by similar disease on the left side; downwards by tumours in the region of the base; and upwards by gaseous distension of the stomach.

On the other hand, when the displacing force is of the nature of *traction*, the heart is drawn *a fronte*, that is, towards the seat of the force. Thus, when a cavity in a phthisical lung is contracting, the pericardium and heart, as well as the walls of the chest, are displaced towards the healing area. It must be observed, however, that in this class of cases, actual traction, in the strict sense, is rare, and that the ordinary displacing force is, in reality, the atmospheric pressure; the heart and the other organs being ‘sucked’ towards the potential vacuum, in the same way as water is ‘drawn’ into a syringe. Still, in a very small number of cases, the pericardium does actually become involved in a healing process in the lungs; and it and the heart are dragged towards the cicatrix.

Besides the displacing force, there are at work in dislocation of the heart certain other agencies, which contribute to the result, whether their effect be to increase or to diminish that of the chief cause:—

(a) The weight of the heart manifestly favours displacement in different directions, according to the posture. Thus, in the erect posture, it favours downward, and limits upward displacement. However, the effect of the weight of the heart is comparatively insignificant, and may be practically disregarded.

(b) The resistance, positive or negative, of neighbouring parts must be taken into account. The heart when disturbed from its

position will move in the direction of least resistance. Thus it cannot be displaced to any extent either forwards or backwards; but is moved with comparative ease towards either pleural cavity. The resistance inferiorly is greater under the right half of the diaphragm than under the left. On the other hand, the resistance around may become negative; for example, in left pleural effusion the corresponding half of the diaphragm is pushed downwards, and the accompanying downward displacement of the cardiac apex is thus increased.

(c) The heart is attached at its root; and, speaking broadly, this is a fixed point, at the right and upper extremity of the long cardiac axis. This attachment will limit and otherwise modify displacements of the heart in all directions, especially downwards. Round this point as a centre, and with the long axis as the radius, the apex of the heart would describe an arc of a circle, cutting the surface of the chest in the left axilla, the left submammary region, the epigastrium, the right submammary region, and the right axilla.

(d) The tendency that the heart has to rotate or roll on one or other of its axes is also affected by its attachment at the root. If the heart lay free in the pericardial cavity, there would be no limit to such rotation under the influence of pressure or of traction. The base being fixed, rotation is greatly limited, and does not occur to any extent except around the longitudinal axis; the left ventricle, for example, being rotated more forwards or more backwards, as the case may be. Rotation round the transverse-horizontal and the antero-posterior-horizontal axes is very limited.

**ANATOMICAL CHARACTERS AND EFFECTS.**—The only essential change that the heart is found to have undergone in displacement is an alteration of its relations to the surrounding parts. The softer parts of the cardiac wall, however, such as the auricles, are sometimes compressed to a moderate degree. The pericardium is partly dislocated and partly stretched. The great vessels at the base of the heart and at the root of the neck may be elongated, shortened, twisted, or bent, according to the particular form of displacement; and the circulation within them impeded. The neighbouring organs are variously displaced and compressed. One of the effects often seen after displacement is permanent fixation of the pericardium and heart in their new position, for example, in the pleural cavity, on the disappearance of the original cause.

The effects of displacement of the heart upon its functions differ greatly in the two classes of dislocation to which we have referred:—

In displacement due to *pressure*, the heart is compressed between the displacing force and the resistance in other directions, and the dislocation is generally rapid. For-

tunately, in most cases of such displacement the resistance is slight; and the heart, if healthy, suffers little or no real compression of its substance or cavities between the two forces, the mobile and compressible lung especially yielding before it. But if the heart be diseased—and especially if its walls be weak, degenerated, or dilated—moderate compression, as in flatulence, may cause embarrassment of the cardiac action and even fatal paralysis; and the rapidity or even suddenness with which displacement generally occurs when due to pressure—for example, in pneumothorax—is another and perhaps the principal cause of this embarrassment.

On the other hand, when the heart is drawn out of its normal situation towards a pthysical cavity, or towards either pleural cavity from which an inflammatory effusion is being absorbed, the displacement occurs, not because there is want of space, but because there is excess of space within the thorax. The process is also very gradual. The effects, therefore, upon the functional activity of the heart may be said to be few, though the unusual pulsation may be a source of inconvenience, and even of anxiety, to the patient. In very rare cases, the heart and pericardium, when thus displaced, may be involved in the fibrotic process going on in the lung or pleura, and the adhesions thus established may ultimately interfere with the cardiac action.

**SYMPTOMS.**—In displacement of the heart, special symptoms are frequently slight or altogether wanting; or they are inseparable from the symptoms of the original cause. This may be said to be almost invariably the case when the displacement is due to gradual traction, as in pthisis. In the pressure class of cases, on the contrary, there are frequently developed, and that rapidly or suddenly, symptoms due to compression of the heart, such as a sense of distress, stifling, and pain over the præcordia or at the epigastrium, or even true angina; dyspnoea, perhaps amounting to orthopnoea; palpitation; blueness of the surface; and irregularity and feebleness of the pulse. When the displacement is due to upward pressure from gaseous distension of the stomach and intestines, the above symptoms may be associated with flatulence or 'spasms,' and are relieved by the erect posture, eructation, vomiting, and the administration of carminative and absorbent remedies. If this condition be not removed within a short time, it may become aggravated, pass into a state of collapse, and end in death.

**VARIETIES AND PHYSICAL SIGNS.**—The varieties of cardiac displacement, according to the direction in which the dislocation occurs, may, for clinical purposes, be said to be as follows: towards the *left*, towards the *right*, *downwards*, *upwards*, *backwards*,

and *forwards*. It must be observed, however, that this is only a broad general classification, and that the heart is very rarely displaced in an absolutely horizontal, or in an absolutely vertical plane. The exact direction taken in each variety will now be described, as well as its special causes, and the physical signs by which it may be recognised.

**1. Displacement towards the Left.**—This, the most common variety of marked cardiac dislocation, is most frequently caused by contraction of the left lung from any of the conditions already enumerated, and effusions into the right pleural cavity. Right pneumothorax, and tumours connected with the right side of the chest, with the mediastinum, or with the right lobe of the liver, are less common conditions leading to the same result. The distance towards the left to which the heart is dislocated varies, the extreme limit being probably the vertical axillary line. During its progress towards the left, the heart is rotated around its longitudinal axis, so that the right ventricle is more exposed anteriorly; and the apex is moved, at first somewhat downwards, and afterwards upwards.

The visible and palpable impulse is found to the left of its normal situation, and either lower or higher than it, or on the same level with it, according to the degree of displacement. In some cases due to contraction of the left lung, the impulse may be found in any one or in all of the left intercostal spaces from the base to the apex of the heart, and of different rhythm in the different spaces. If the displaced heart be the seat of valvular disease, thrill may be felt in an unusual situation, for example, in the left axilla. The area of percussion-dulness is altered in outline, being invaded on the right side either by the dulness due to effusion there, or by resonance due to pneumothorax or to encroachment of the right lung-border; whilst it is either transposed towards the left axilla, or blended with unnatural dulness over the left lung. The cardiac sounds are reduced in loudness over the normal præcordia, whilst they are unnaturally loud towards the left axilla and up the left front. Structural murmurs if present are similarly transposed, as regards the seat of their greatest intensity and the lines of their convexion. A systolic murmur may be developed at the base of the heart from distortion of the great vessels.

**2. Displacement towards the Right.** This form of dislocation of the heart is the result of effusion into the left pleural cavity; of contracting processes connected with the right lung or pleura; of left pneumothorax; and of tumours of the left side of the chest or in the mediastinum. The heart may be displaced towards the right side until the impulse is found in the axillary region, the true apex at the same time being depressed

towards the epigastrium. During its lateral movement, the heart is rotated on its longitudinal axis in such a manner that the left ventricle is more exposed.

The physical signs correspond closely with those enumerated under left displacement—the two sides being, of course, exactly reversed. The cardiac impulse is most frequently transferred to the epigastrium. New areas of pulsation are developed in the region of the right nipple, and in the second and third right interspaces, close to the sternum; they indicate the displaced position of the right auricle, if præ systolic, or of the aorta, if systolic and followed by palpable shock in diastole. The description of the auscultatory phenomena, as regards both sounds and murmurs, does not require to be repeated.

**3. Displacement Downwards.**—This is an exceedingly common form of cardiac displacement, though seldom extreme in degree. It is the constant result of hypertrophic emphysema of the lungs; and may also be caused by the downward pressure of tumours at the base of the heart, such as aneurysm, and by collapse of the stomach and intestines. Displacement of the heart downwards is limited by the diaphragm, and by the attachment of the pericardium and great vessels at the root of the heart. At the same time the apex may either move somewhat towards the left in its descent if the downward pressure be uniform, as in emphysema; or it may ascend somewhat towards the left if the pressure be exerted chiefly upon the base.

The ordinary cardiac impulse is generally quite imperceptible in this form of displacement, on account of enlargement of the lungs; or it is greatly weakened, and situated in the sixth left space, or lower, to the left of its normal position. A new area of systolic pulsation is perceptible in the epigastrium, generally well marked, and connected with the right ventricle. The præcordial dulness is usually completely replaced by pulmonary resonance; or, more rarely, confused by the dulness of some form of mediastinal tumour. The cardiac sounds are feeble, or absent, over their usual seat; and are heard, instead, over the epigastric triangle and the lower left cartilages.

**4. Displacement Upwards.**—The many abdominal causes of this form have been already mentioned; so have the symptoms due to compression of the heart which characterise it when so produced. The heart, as a whole, is moved upwards in the chest, and at the same time the apex passes more or less towards the left, and the right ventricle may become somewhat more exposed anteriorly.

The cardiac impulse is elevated until it is found on the nipple-level, or even higher; or it is lost, along with the area of percussion-dulness, behind the inferior border of the left lung. The sounds of the heart are transposed

upwards, and weakened. The displacement of the cardiac apex towards the left axilla in pericardial effusion is described elsewhere. See PERICARDIUM, Diseases of.

**5. Displacement Backwards.**—This variety of displacement of the heart is very uncommon; and when it does occur, is generally referable either to abundant pericardial effusion, or to backward curvature of the spine (kyphosis) in the dorsal region. A certain amount of backward displacement is, however, not so rare in extensive excavation of the left lung, in association with other forms of dislocation. The base of the heart is then the part most transposed into the left paraspinal groove, and the apex is tilted somewhat forwards as well as elevated.

The physical signs of backward displacement are those of the cause of the malposition rather than any that can be referred to the condition itself.

**6. Displacement Forwards.**—Displacement forwards is also very rare, although it is frequently simulated by bulging of the præcordia in enlargement of the heart. The chief cause of it is the presence of a tumour in the mediastinum—especially aneurysm of the descending aorta, or enlargement of the bronchial glands. The amount of actual transposition is necessarily exceedingly small, the anterior border of the lungs being compressed or pushed aside, but the further progress of the heart forwards being arrested by the anterior wall of the chest.

The physical signs are, therefore—increase of the area and strength of pulsation and of percussion-dulness over the præcordia; bulging of the same in young subjects; and increased loudness of the cardiac sounds in that situation.

**7. Complex Displacements.**—It has already been indicated that displacements of the heart, strictly speaking, occur almost without exception in more than one of the directions described; and they may all, therefore, be said to be generally more or less complex. Dislocation at once upwards and towards either side is especially common, as the result of contracting processes in the apex of the lung.

**DIAGNOSIS.**—After the full account that has been given of the several forms of displacement of the heart, there ought to be no great difficulty in diagnosing them from each other, as well as from the conditions which simulate them. These must be carefully remembered. The chief of them are: (1) Physiological displacements, already referred to; (2) cardiac enlargement, especially when attended with bulging of the præcordia; (3) pulsating tumours of the chest and abdomen, particularly aneurysm of the aorta; (4) adhesion of the pericardium; and (5) atrophy of the lungs from any cause.

**TREATMENT.**—The rational treatment of

displacement of the heart would be to remove its cause; but when the cause is of the nature of traction, treatment is very rarely called for, even if it were possible. In displacement due to pressure, on the contrary, treatment is often urgently indicated, perfectly practicable, and highly successful. The unpleasant sensation of pulsation complained of in some instances of displacement—for example, in phthisis—is frequently relieved by an assurance on the part of the physician that the palpitation is of no import; and by the application of a simple plaster, containing iron, belladonna, or opium.

J. MITCHELL BRUCE.

**HEART, Embolism of.**—See **EMBO-  
LISM**; and **HEART, Thrombosis of**.

**HEART, Fatty Degeneration of.**—  
**SYNON.**: FR. *Dégénérescence graisseuse du  
Cœur*; GER. *Fettige Metamorphose des Her-  
zens*.

**DEFINITION.**—The process by which the muscular fibres of the heart are converted into a granular fatty matter. The term is also used to express the state of the heart in which this change has been accomplished.

**ÆTIOLGY AND PATHOLOGY.**—The process by which the protein elements of animal bodies, including muscular fibre, are converted into granular fatty matter, as well as the circumstances under which this change occurs, have been already so fully discussed under the head of **FATTY DEGENERATION**, that it is unnecessary to repeat the information which will be found there. It will suffice to say here, that when the process of nutrition is interfered with in the tissue of the heart, this change takes place, and is best illustrated in the *local* or *limited* form of disease, which occurs when the coronary circulation is obstructed. This is seen in cases in which the orifice of the coronary arteries is contracted or where there is thickening or calcification of one of the trunks, or of the branches of these vessels, and is more marked by reason of the fact that the coronary arteries do not anastomose as freely by their primary and secondary branches as do arteries in other situations. Still, as Dr. Samuel West has well shown (*Lancet*, vol. i. 1883), there does exist free communication. The fatty change is found to occur in the more *diffused* or *general* form in those diseases in which the vital powers are lowered, as in certain forms of chronic cachectic disease, especially in cases of pernicious anæmia, as described by Dr. Sidney Coupland (*Brit. Med. Journ.*, vol. i. 1881), in poisoning by phosphorus, or after loss of blood. In certain other conditions, such as acute specific fevers, the tissue of the heart becomes softened, and under the microscope presents a granular appearance, which is believed by some pathologists to be an incipient stage of fatty degeneration. We might

also refer to the more or less diffused form of fatty degeneration which takes place in cases of enlarged heart, the result, not, as Rokitsansky supposed, of a disturbance of the nervous functions, but of the fact that these enlarged hearts require a larger supply of the materials for nutrition than can be furnished to them by the coronary arteries, which in such cases are frequently themselves diseased, both at their origin and in their course. Lastly, fatty degeneration of the heart is found to occur after delivery in some instances, in which the organ had become enlarged during pregnancy, thus explaining certain cases of sudden death after parturition. Dr. Bedford Fenwick has described (*Lancet*, vol. i. 1888) fatty degeneration occurring in consequence of the pressure exercised by the presence of large ovarian tumours; and he accounts for the collapse which sometimes occurs after operation for the removal of these tumours by the presence of this lesion of the heart's texture.<sup>1</sup>

Certain other circumstances connected with the origin of the disease require to be mentioned here. In reference to *sex*, the disease is more frequent in males, in the proportion of nearly two to one. With respect to *age*, in his original memoir on this subject the writer found that nearly one-half of all the cases observed were over sixty years of age. In the late Dr. Hayden's valuable work on *Diseases of the Heart*, the proportion stated of cases under sixty years of age shows a larger number of young persons whose hearts have undergone this change as a result of wasting disease—a result which is evidently due to the greater care with which microscopical examinations of the heart have been made in recent times. As regards *social position*, of thirty-three cases formerly noted by the writer, the subjects of the disease are stated to have belonged to the higher ranks in nine cases; to the middle class in eight cases; and to the lower class in sixteen cases. This enumeration contrasts with the proportion in which fatty growth appears on the heart; seven of fifteen cases belonging to the first class; six to the second; and only two to the third. Fatty degeneration and fatty growth on the heart are thus seen to occur under markedly different conditions. The latter is the result of the accumulation in the blood of the elements of fat; the former is the result of decay and disintegration of tissues.

**ANATOMICAL CHARACTERS.**—In fatty degeneration the heart was found to be enlarged in about two-thirds of the cases recorded both by Dr. Hayden and the present writer. It is not infrequently simply dilated. To find a heart thus affected of an average size,

<sup>1</sup> For much very instructive and valuable information, see an article, by Dr. F. W. Mott, on 'Cardio-Vascular Nutrition and its Relation to Sudden Death,' in the *Practitioner*, vol. xli. p. 161.

or even occasionally below it, is not an exceptional occurrence. The colour of the heart's substance is pale, sometimes as pale as 'a dead leaf,' but more generally it is of a yellowish-brown or buff, or muddy pink colour. This discoloration is generally seen in spots or in patches; and though the whole heart may be pale, the spots, being still paler when seen beneath the endocardium, give the tissue a mottled look. The same appearance may be observed beneath the pericardium, and in the substance of the heart. With the progress of disease the spots run together, giving portions of the walls a uniform buff-coloured character, whilst the rest of the organ retains its ordinary aspect. The consistence also varies from that of mere flabbiness or softness, to such a condition as permits of the tissue being torn like wet brown paper. The organ then feels like a piece of wet chamois leather, or a wet glove. In other cases the heart retains in appearance much of its ordinary solidity, but the tissue breaks down on pressure, as does a lung consolidated by pneumonia. This is a state which occurs more frequently in hearts which are hypertrophied. In addition to these changes in size, colour, and consistence, others have to be noted. The fibrous character of the heart's structure, even to the naked eye, disappears; in some cases the tissue resembles that of a fatty or a boiled liver. In other instances the cut or torn surface has a granular appearance, not unlike that of the lung in an early stage of grey hepatisation. These different appearances may in a great measure be due to the greater or less fluidity of the oily matter present, as well as to the extent and degree to which the disease has advanced. Further varieties in appearance may be caused by the presence of a larger or smaller quantity of blood, or of its colouring matter, in the heart's texture or in its cavities, by which the lining membrane may

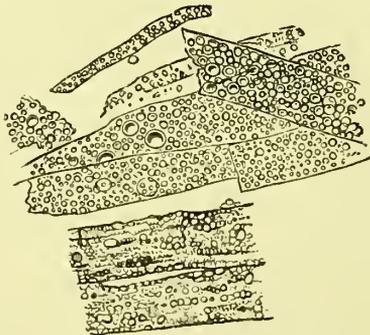


FIG. 60.—Fatty Degeneration of the Heart.  
x 400 diam.

in the latter case be dyed of a deep purple colour.

*Microscopical characters.*—The microscopical characters of this disease will be found

so fully described under the head FATTY DEGENERATION, that it is needless here to do more than refer the reader to that article, and to the annexed figure (60), in which these characters are well seen.

All parts of the heart are subject to fatty degeneration, but not equally so. It is most frequently found in the left ventricle; next in the right ventricle; then in the right auricle; and least frequently in the left auricle. It is generally more evident in the columnæ carneæ, and in the inner layers of the muscular walls, than elsewhere.

*EFFECTS.*—Of the *structural* lesions which occur in the heart when it is the seat of fatty change, one of the most important is *rupture*, which was found in twenty-five out of sixty-eight cases of fatty or softened heart, the histories of which were collected by the writer. Partial rupture, leading to some effusion of blood in the tissue and the formation of what has been called *cardiac apoplexy*, is another condition which has been described. The clot in such cases, if it lose its colour, may produce the appearance of an encysted abscess; and a consecutive false aneurysm of the walls of the heart may be thus formed, as well as by the simple yielding of a portion of the softened cardiac wall. The involvement of the columnæ carneæ may lead to imperfect action of the valves. Valvular disease itself is not often present in connexion with fatty heart. Dr. Henry Kennedy, in a late interesting work on this subject, pointed out that the valves are affected only about once in nine cases; and he further shows that, when the valves are affected, it is chiefly the aortic valves that are involved.

Of the effects of fatty degeneration upon the *functions* of the heart, the most prominent are those which exhibit the weakened powers of the organ. *Coma*, preceded or not by giddiness, has been described by several writers in connexion with feeble powers of the circulation. The late Dr. Adams of Dublin has mentioned as many as twenty attacks in one of his cases of fat heart; and the writer has noticed the occurrence of even more frequent seizures. *Syncope*—'cardiac syncope'—is a term very frequently used by the older writers; and it is a term which may be well applied to the condition of faintness which is frequently found in connexion with fatty heart. In some cases the feeling of syncope amounts to little more than a simple sense of faintness—that the patient must fall if he do not lay hold of something. In other instances this symptom is accompanied by a feeling of impending death; and such patients do frequently die. In the cases collected by the writer, thirteen out of thirty-three died of what he proposed to call *syncope lethalis*, or 'fatal syncope;' and it would be possible, no doubt, now greatly to extend the number of cases that have proved fatal in this way. Death may result in such cases

from cardiac failure, as indicated after death by a flabby heart containing blood in the left ventricle; or, where there is a less amount of degeneration, death may result from irregular action or spasm, and the ventricles may be found empty.

*Pain* is also an effect of degeneration. It may occur independently of syncope; or, being associated with syncope, constitutes what is named *syncope anginosa*. See ANGINA PECTORIS.

The *respiration* is always affected in cases of fatty degeneration, and we find either simple breathlessness, even on slight exertion, or that peculiar form which has been called *Cheyne-Stokes respiration*.

**SYMPTOMS AND DIAGNOSIS.**—There is no doubt that many cases occur in which fatty degeneration is found in the heart after death, without its presence during life having been suspected. This result is found more especially in those instances of exhausting disease in which the heart participates. In such cases the requirements of the system may not be out of proportion to the powers of the heart; and death may come on slowly and insidiously without our attention being attracted to the state of this organ. In a second class of cases, in which the heart suffers from some local cause, as, for example, from disease of the coronary arteries, whilst the system generally maintains its powers more or less fully, the balance between the system and the function of the heart is lost, and diagnostic evidence of the change that has occurred in the central organ, sufficiently clear and pointed, may be traced without difficulty. Amongst the symptoms of the disease we then observe various modifications of the phenomena of drowsiness and coma; faintness and syncope; disordered respiration; pain in the region of the heart; and disturbed pulsation. For example, the patient complains in the earlier stages of being easily exhausted, particularly by mounting ascents; he feels, he says, faint on reaching the top of a flight of stairs; though not giddy, he feels he must fall; though not breathless or fainting, he sighs deeply and seeks the air. Any unusual excitement, or a heated or a close atmosphere, produces the like effects. At the same time there is often experienced an uncomfortable feeling of choking or fullness in the chest. In the intervals the individual may be fairly well.

As the disease advances, the attacks, of whatever kind, become more frequent and severe, and often disturb and distress the patient at night. The temper is observed to become irritable. The expression of the features frequently appears anxious, and the countenance sallow. Copious perspiration from very slight causes, sometimes coldness of the extremities and swelling of the ankles, appear amongst the incidents of the disease. The pulse is generally affected; but how

must no doubt depend in a great measure upon the part of the heart affected, and on the extent and degree of the disease. In the writer's opinion, intermittence and irregularity are the more frequent alterations; weakness is another; and slowness—often remarkable—is a third. Quickness of the pulse, more especially when it increases with age, has been dwelt upon by Dr. Kennedy as a symptom deserving of attention in the diagnosis of fatty degeneration. The irregularity may be constant, or it may appear and disappear. The writer has seen it present during the slight disturbances above described; and he has seen it disappear altogether when the patient was in tolerable health, to return as the effect of any depressing cause, the more marked because that cause may be far too inefficient to affect a sound heart. The frequency with which the pulse is affected in cases of dyspepsia, more especially in those in which there exists a gouty diathesis, should not be overlooked in the diagnosis of fatty heart.

The breathing is always more or less affected in cases of fatty degeneration of the heart. In some instances the disturbance is represented as a sense of choking or suffocation; the person feels as if he were breathing through a sponge. The difficulty in some instances is so slight as scarcely to be regarded; in others so severe that the smallest effort, particularly in mounting ascents, is most painful. A peculiarity sometimes observed is that the ascent of a gentle height is distressing, while the person can read aloud without inconvenience. A character of the respiration first described by Dr. Cheyne of Dublin, and afterwards by Dr. Stokes, is by some regarded as diagnostic of fatty degeneration of the heart. It is thus described by Dr. Stokes: 'A form of respiratory distress peculiar to this affection (fatty degeneration of the heart), consisting of a period of apparently perfect apnoea, succeeded by feeble and short inspirations, which gradually increase in strength and depth until the respiratory act is carried to the highest pitch of which it seems capable, when the respirations, pursuing a descending scale, regularly diminish until the commencement of another apnoeal period.' Dr. Hayden, in writing on this subject, mentions a case in which during the period of apnoea there was no change in the heart's action; a second case in which the action of the heart and the pulse underwent no change during the period of apnoea and dyspnoea; whilst in a third case, during the paroxysm of dyspnoea, the heart's action was remarkably irregular. It should be stated with regard to this symptom that, though frequently present, it is by no means characteristic of fatty degeneration only. It is by some said to be more frequently associated with disease of the aorta. Various explanations of this phenomenon have been given. Dr. Little (*Dublin Journ.*

of *Med. Sci.*, No. 91) believes that it is due to derangement of the dynamic adjustment between the right and left ventricles of the heart. Dr. Hayden (*op. cit.*) connects it with atheromatous or calcareous change with dilatation of the arch of the aorta, involving loss of elasticity in its walls. The late Professor Laycock thought (*Dublin Journ. of Med. Sci.*, July 1873) that this phenomenon depended upon 'sentient palsy of the respiratory centre,' or 'a paresis of reflex sensibility of the mucous membrane of the lung.' This respiratory action, though suggestive of some grave and serious source, may pass away. See RESPIRATION, Disorders of.

Another phenomenon, said to be diagnostic of fatty degeneration of the heart, is arcus senilis—a pearly crescentic opacity of the upper and lower portions of the circumference of the cornea, which must be distinguished from an opaque annulus which occasionally surrounds the entire cornea, and which is not the result of fatty degeneration. The late Mr. Canton was the first to describe the nature of this change as fatty degeneration. It is quite true that when fatty degeneration is present in the cornea it may possibly be found in the muscular fibres of the heart and in the arteries. Still it by no means follows that the degeneration must exist in any other particular part or organ, and therefore this appearance in the cornea cannot be at all regarded as pathognomonic of fatty degeneration of the heart.

As the disease progresses the symptoms become more marked; the various effects of feeble and languid circulation further show themselves; angina may perhaps become fully developed; or the patient may be cut off suddenly by one or other of the effects connected either immediately or remotely with the lesion itself. Of eighty-three cases of 'fatty disease' of the heart collected by the writer, sixty-eight had died suddenly.

**PHYSICAL SIGNS.**—The physical signs that characterise fatty disease are neither many nor marked. They are—a feeble impulse of the heart, proportionate to the extent and the degree of the disease; a feeble muffled first sound, under like conditions, sometimes scarcely audible. When the heart is enlarged, the impulse, though weak, will be found extended, and so likewise will be the dullness on percussion. A murmur may be present, as suggested by Rokitansky, from degeneration of the columnæ carneæ. The second sound is often distinct and clearly accentuated, as compared with the first.

**DIAGNOSIS.**—The diagnosis of the presence of this degenerative change, in the absence of any alteration in the size of the heart, must be founded upon a consideration of the symptoms and physical signs above described. When the heart is hypertrophied or dilated only, the presence of fatty degeneration is more difficult of diagnosis by its physical

signs. We must then seek to trace how far the usually well-marked signs of hypertrophy of the organ are modified by those we have described as being present in, and characteristic of, fatty degeneration. The same observations will apply to dilatation or thinning of the walls. This special condition has its own well-marked phenomena, which will be found described elsewhere (*see* HEART, Dilatation of). These signs will be more or less modified in proportion to the degree and extent of any fatty change that may be present. They must be carefully studied so that they be not confused with like conditions originating in disordered state of other organs, as the lungs, the liver, &c.

**PROGRESS, DURATION, AND TERMINATIONS.**—It is impossible to determine the duration of a disease the date of origin of which is in most cases very obscure. Still there are grounds for believing that persons with a certain amount of degenerated tissues in their hearts have gone on living during periods extending over many years. On the other hand, death has occurred from fatty degeneration of the heart, determined *post mortem*, in which the entire absence of symptoms until a few months before the fatal event justified the opinion that the duration of the disease had not much exceeded the period just mentioned. When fatty degeneration occurs as the result of phosphorus-poisoning, or of certain exhausting diseases, the progress of the change, which can then be determined, is rapid. In such cases, the morbid process is not confined to the heart alone, and therefore when death occurs, it cannot well be attributed solely to the condition of this organ.

Death is frequently sudden, the proportion being as five to one compared with other modes of death from fatty disease of the heart. The immediate modes of death in these cases are those which have been already alluded to when treating of the effects of the disease, namely, by syncope, angina, coma, and rupture of the heart; the first and last of these contributing nearly the whole number of those who die suddenly. Such facts indicate very strongly the necessity of avoiding any mental excitement or physical exertion which might lead to these results. Here it might also be well to remember, with reference to the administration of anæsthetics, that chloroform has an especially depressing effect on the heart's action; and that when the heart's power is enfeebled by the disease which we are here describing, a very small dose of this anæsthetic, which would have little or no effect on a healthy heart, may prove fatal. This opinion was first expressed by the writer many years ago, in 1850, and it has been fully confirmed by numerous cases of death which have since occurred during the administration of chloroform.

**PROGNOSIS AND TREATMENT.**—The prognosis of fatty degeneration of the heart must depend in a great measure upon a knowledge of its cause and its extent. In cases where the disease originates in constitutional causes, such as in phosphorus-poisoning, and in cases where it is of the nature of involution—for example, after parturition—there is good ground for believing that, the cause being removed, the effect will cease, and a fairly healthy condition of the organ be restored. On the other hand, when the coronary arteries are obstructed, and degeneration is thereby set up, or when nutrition generally is impaired, and all the tissues are more or less undergoing this change, the prognosis must be in the highest degree unfavourable, more especially so if in the latter case the patient cannot be placed in a condition by which this degenerative tendency may be counteracted.

The treatment consists in the adoption of all the measures calculated to improve the general health—such as pure fresh air, wholesome food, and temperance, together with moderate exercise (either carriage, riding, or walking), if it can be accomplished without causing pain or breathlessness. Everything which may tend to lay stress on the heart's action, such as walking uphill or making efforts, or any mental excitement, should be avoided. With reference to drugs, such tonics as can be best tolerated by the patient might be given. We may mention iron—especially dialysed iron—phosphorus in small doses, and strychnine. Special attention must be paid to the condition of the excretory organs, such as the kidneys and liver, which are liable to become congested when the cardiac action is feeble. Lastly, it may be said that in cases of syncope, in addition to the administration of the usual stimulants, galvanism applied from the back of the neck to the præcordia by the interrupted current, has in a few instances been known by the writer to be useful.

For further information on the subject of Fatty Degeneration of the Heart the reader may consult the complete and very valuable article by the late Dr. Hayden in his work on *Diseases of the Heart*; and a memoir by the writer in the 33rd vol. of the *Med.-Chir. Trans.* (1850). RICHARD QUAIN.

**HEART, Fatty Growth on.**—SYNON.: Fr. *Hypertrophie graisseuse du Cœur*; Ger. *Fettige Infiltration des Herzens*.

**DEFINITION.**—The growth of fat on the surface and in the substance of the heart, in quantity sufficient to interfere with its functions, and thus to constitute a disease.

**ÆTIOLOGY.**—In our inquiries concerning the cause of this condition, we are met with the problem, still to be solved, Why are certain individuals, and certain parts of the body, more prone to the formation of fat than other

persons and other parts? We can ascertain with some degree of certainty the circumstances which promote the formation of fat in general; and observations collected by the writer and others show that when fat is thus formed throughout the system, the heart is likely to partake largely of the accumulation. We may accordingly refer to the article **OBESITY**, in which the causes of fatty growth in general will be found discussed.

Of fifteen cases of extreme fatty growth on the heart collected by the writer, eleven occurred in very fat individuals, and only one in a person who was described as being 'thin.' Age seems to exert a decided influence upon the formation of fat upon the heart. It is very scanty in infancy, and is rarely present in any quantity before the thirtieth year. Corvisart, however, quotes from Kercking the case of a child whose heart seemed wanting, so great was the quantity of fat in which it was embedded. Of the fifteen cases just referred to, thirteen were above fifty years, and one only under that age. Males, according to the same data, are more liable to accumulation of fat on the heart than females, the respective numbers being as twelve to three.

**ANATOMICAL AND PATHOLOGICAL CHARACTERS.**—A certain amount of fat-tissue, which is not inconsistent with health, occupies a definite position in the structure of the heart. It is seen most abundantly in the groove between the auricles and the ventricles; and as the distribution of this tissue bears a relation here, as in other parts, to that of the blood-vessels, it first appears in the course of the primary branches of the coronary arteries; then in the course of the secondary branches—that is, in the groove over the septum, which marks the boundary between the ventricles; and, lastly, it follows the distribution of the small lateral branches. These branches are more superficial over the right ventricle than over the left; hence the former is found always and more abundantly covered with fat. A fringe of fat is also found at the apex of the heart; and frequently around the margins of the auricles. A mass of superabundant fat will of itself be sufficient to press on and embarrass the action of the heart; but fat rarely exists in this abundance on the surface of the organ without insinuating itself between, and encroaching on, the muscular fibres. In this way the muscular portions of the walls of the organ become thinner and thinner, until the columnæ carneæ may appear to arise from a mass of fat. This state constitutes what was once regarded as fatty degeneration of the heart, and which has also been called 'fatty metamorphosis;' but it is in many cases nothing more in reality than a simple hypertrophy of fat. In parts of hearts which are less affected, that is, where fat is not very abundant, simple striæ of yellow tissue will be observed lying amongst

the muscular fibres—an appearance often found in the auricles.

*Microscopical appearances.*—When a portion of heart suffering from fatty growth in a high degree is examined with the microscope, it will be found that where the growth is most advanced, that is, towards the external surface, very few muscular fibres can be seen, and that the very wide intervals between them are occupied by fat-cells (see fig. 61). Proceeding inwards, the muscular fibres become more evident; the fat-cells become fewer; and, finally, we reach the muscular fibres beneath the endocardium, with a few fat-cells lying here and there amongst them. It is worthy of note that the fibres, though overwhelmed by fat, may still retain their organisation. In all cases, however, the course and direction of the fibres are more or less modified and distorted. The fact that the fibres still exist, though concealed, affords an explanation of the persistence of the heart's action in those instances in which the muscular walls appear to a greater or less extent replaced by fat. It might also be mentioned that small masses of fatty tissue sometimes

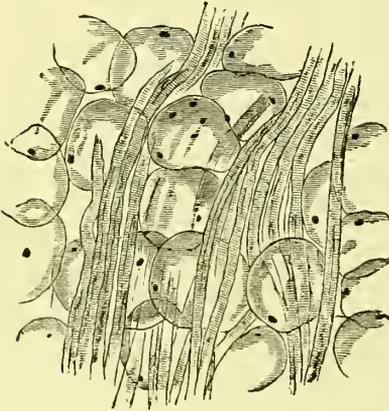


Fig. 61.—Fatty Growth in the substance of the Heart.  $\times 400$  diam.

appear beneath the endocardium, varying in size from that of a pin's head to that of a pea. The writer has seen these little fatty tumours in cases where there was a considerable, but not an excessive, amount of fat upon the surface of the heart.

*EFFECTS.*—The fat accumulated on the heart and in its substance may be supposed to act mechanically; and by its pressure upon the muscular fibres, on the nerves, and on the blood-vessels, to impede the function of the organ, embarrass its nutrition, and produce those effects which may be briefly enumerated as—a languid and feeble condition of the circulation, with a sense of uneasiness and oppression in the chest; embarrassment and distress in breathing, drowsiness, even coma; syncope; perhaps angina

pectoris; it may be death. Rupture of the heart sometimes results. Such an enumeration of evils, in which there is no evidence of any other lesion of the heart's texture, save an accumulation of fat, would suffice to render this condition a source of very grave import; but we cannot always be quite clear on the subject, because in the particular cases quoted it is possible that some degeneration of the muscular fibres may co-exist. It would be well, therefore, not to dwell too much on such effects as proceeding from and dependent on fatty growth alone.

*DIAGNOSIS.*—The presence of an excessive deposit of fat about the heart must, in a great measure, be a matter of inference during life. Where one or more of the effects mentioned above as having been noticed in cases of this kind are present; when the pulse is small and weak; when the first sound of the heart is feeble, and the impulse weak; when the extent of dullness on percussion is increased; and when these phenomena occur in a fat person, it may be inferred that the heart is too fat.<sup>1</sup> But, on the other hand, it must not be overlooked that these symptoms and signs may be found in cases of fatty degeneration of the walls of the heart, in cases where the amount of fat-tissue is but moderate. Nay, more, as already stated, both conditions are often present in the same heart, thus rendering distinctive diagnosis impossible. It is said that the presence of water in the pericardium may be confounded with the presence of fat upon the heart; but the history and general features of the case in the former condition should be sufficient to prevent all difficulty in the diagnosis. See PERICARDIUM, Diseases of.

*TREATMENT.*—The treatment of fatty accumulation on the heart is so intimately associated with the subject of the formation of fat in general, that this point can be discussed with more advantage in its wider relations (see OBESITY). Whilst the treatment directed to this point is being carried out, we can do little more for the heart itself than aim at giving strength to the portion of its texture still available for duty—by tonics,

<sup>1</sup> Dr. Henry Kennedy, of Dublin, in a lately published special monograph on *Fatty Heart*, states that the points upon which the diagnosis of fatty growth on the heart mainly turn are the following: 'First, a large full pulse, beating at the natural standard of frequency; secondly, evidence derived from percussion of the heart's dullness being more extended than natural; thirdly, the possible presence of a soft systolic murmur over the aortic orifice, occupying the first sound of the heart only, and leaving the second normal; and, lastly, the condition of the individual as to his being fat or otherwise' (p. 39). The present writer hesitates to agree with Dr. Kennedy as to the condition of the pulse generally, and certainly this description will not apply in the cases of those persons of small frame, with small arteries, who often become obese and present symptoms of fat-heart after the middle period of life.

steel, quinine, phosphorus, &c.; secondly, by lightening as far as possible the work which the heart has to do; and thirdly, by attending to the excreting organs, so as to prevent congestion there, and consequently embarrassment to the weak heart.

RICHARD QUAIN.

**HEART, Fibroid Disease of.**—SYNON.: Chronic Myocarditis.

**DEFINITION.**—A morbid condition in which the muscular fibres of a portion of the walls of the heart are replaced by fibroid tissue.

**ÆTIOLGY.**—Fibroid disease of the heart is met with most frequently in middle-aged male subjects. The disease is supposed to be occasionally nothing more than an extension between the muscular bundles of a chronic process that has commenced with endocarditis or pericarditis. The cause of this, which is generally rheumatism, is then regarded as the cause of the fibroid growth; but in reality it is more probable that in such cases the serous inflammation is the result, not the cause, of the fibroid change. Fibrosis is sometimes the consequence of acute interstitial myocarditis. In a considerable number of cases of fibroid disease, and in most of the cases of so-called 'fibrinous deposit,' the change is probably syphilitic in its nature. In other instances it is associated with degenerative changes in the vessels, and even infarction, or with disease of the kidneys. Increase of fibrous tissue in the myocardium may also be the result of prolonged moderate congestion of the coronary veins (*see* HEART, Congestion of). Very frequently no evident cause of the disease can be discovered.

**ANATOMICAL CHARACTERS.**—Our knowledge of the pathology of this disease is in a great measure due to the remarkable number of cases which have been described in the *Transactions of the Pathological Society*, since the first specimen was presented by Sir Richard Quain in the year 1850. Fibroid disease of the heart occurs most frequently in the walls of the ventricles. It is met with under several different forms. In rare instances, which might be described as cases of *connective tissue hypertrophy* of the heart, there is an uniform increase of fibrous tissue between the muscular fibres throughout the whole organ (*see* HEART, Connective-Tissue Hypertrophy of). In other instances, the disease appears as a local thickening of the connective tissue underneath an opacity of the endocardium or of the pericardium, whence septa run outwards or inwards between the muscular bundles. Most frequently, however, it presents the appearance of a *fibroid patch*, generally situated near the apex of the heart, replacing the muscular substance throughout its whole thickness, and over a greater or less extent of surface—possibly even a considerable portion of

one ventricle, and consisting of dense, firm, inelastic, greyish-white fibrous tissue. Smaller patches, nodules, scars and streaks may be found in the deeper parts of the myocardium. The apices of the papillary muscles, again, may become fibroid, especially in chronic valvular disease. Polypoid tumours, composed of fibrous tissue, have been met with on the endocardial surface of the heart, that is, projecting into one of the cavities; more especially into the left auricle. Possibly the detachment of such a polypus may be one mode of origin of the 'fibrinous balls' or 'concretions' occasionally found lying free in the auricular cavities. Fibroid and 'fibrinous' formations due to *syphilis* are described in the article HEART, Syphilitic Disease of.

Microscopically, fibroid disease of the myocardium presents a concomitant increase of the connective-tissue elements, and decrease by atrophy of the muscular fibres. Occasionally, in an early stage, as well as at the margins of the older patches, round and spindle-shaped cells and bundles of young fibrillæ have been observed. The latter increase in size and in number, press upon the intervening muscular fibres, and finally unite and form bands or patches of ordinary fibroid tissue. Meanwhile, the muscular fibres gradually become attenuated, granular, or fatty; and at last they disappear by absorption, or patches of them may be imprisoned within the fibroid growth.

The effects upon the heart of fibroid changes in its walls vary with their situation and extent. If a large portion of the wall of one cavity is fibrotic, irregular patchy dilatation of the chamber ensues. Localised fibrosis, especially if it commence beneath the endocardium, gives rise to aneurysm of the heart, by the yielding of the diseased area to the intra-ventricular pressure (*see* HEART, Aneurysm of). Deeper or more limited patches or lines of cirrhosis cause irregularity or puckering of the cardiac walls; and valvular insufficiency may result from this, or from fibrosis and functional disturbance of the papillary muscles. Lastly, fibroid disease occasionally involves the conus arteriosus in an annular form, giving rise to constriction and the formation of so-called 'cardiac stenosis.'

**SYMPTOMS.**—The symptoms of fibroid disease of the heart vary greatly in different instances, according to the extent, situation, and other conditions of the growth. When the fibrosis is very limited, few symptoms can be expected to be present. In the majority of cases in which a considerable portion of the cardiac wall has been found diseased, the symptoms have been described as those of 'ordinary heart-disease,' namely, dyspnoea on exertion; præcordial pain or distress; occasional palpitation; a small, weak, or irregular pulse; dropsy; and visceral

complications. As a rule, no endocardial murmur has been present; but fibrosis of the papillary muscles may sometimes give rise to the signs of incompetence of the auriculo-ventricular valves. The symptoms of cardiac aneurysm and of syphilitic disease of the heart, as well as those of connective-tissue hypertrophy, are separately described.

**COURSE AND TERMINATIONS.**—The course of fibroid disease of the heart is generally chronic, although urgent symptoms are sometimes observed a short time only before death. Attacks of pain, palpitation, and dyspnoea may occur and subside long previous to the last fatal illness. The development of cardiac aneurysm, and its possible terminations, will modify the course of the disease. Sudden death may occur, with or without previous cardiac symptoms, and must be regarded as a special mode of termination of fibroid disease of the heart. Otherwise the cases generally end by pulmonary complications, dropsy, and exhaustion.

**DIAGNOSIS.**—Fibroid disease of the myocardium has to be diagnosed from chronic valvular disease; from enlargement due to extracardiac causes, such as renal disease, gout, or emphysema; and from fatty degeneration. Under all circumstances, an accurate diagnosis is extremely difficult, if not impossible. The presence of a murmur does not exclude fibrosis, as the valves may become secondarily involved; and valvular disease is not always attended by a murmur. The other cardiac lesions mentioned must be excluded in the ordinary way.

**PROGNOSIS.**—When fibroid disease of the heart is attended with symptoms sufficient to establish a diagnosis, the prognosis is unfavourable as regards life, although it may not be immediately so.

**TREATMENT.**—This consists in relieving and supporting the heart by every possible means, especially by rest and cardiac stimulants, such as alcohol and ether. Iodide of potassium may be given with benefit in some cases, particularly if there be a history of syphilis. Digitalis will have to be administered with great circumspection.

J. MITCHELL BRUCE.

### HEART, Functional Disorders of.

**DEFINITION.**—A disturbance in the functions of the heart, with or without pain; having origin in causes other than inflammation or organic diseases of the heart itself.

This definition comprises various disorders in the dynamical functions and sensibility of the heart, from the slightest disturbance of only momentary duration, to urgent symptoms of considerable persistence. The frequency of the occurrence of functional disorders of the heart, and the similarity of many of the symptoms exhibited to those met with in organic diseases, as well as the

fact that these functional disorders may co-exist with organic disease, thus greatly exaggerating the apparent gravity of the latter, render it important to accurately determine how much of the disturbance may be due to the one or to the other of these causes. Here, however, functional disorder, uncomplicated with organic lesion, will only be referred to.

**ÆTIOLOGY.**—To estimate the *immediate* or *proximate* cause of functional disturbance of the heart, regard must be had to its structure, and how this is nourished and its motions regulated. For adequate and equable dynamical movement, the primary requirement is a healthy development of muscular structure. Weak muscular fibre, apart from degenerations by disease, becomes a predisposing cause of feeble and irregular action. But as the regular recurrence of the muscular contraction and expansion must, moreover, be ascribed to the agency of the cardiac ganglia, the vagus nerve, and the nerves and ganglia of the sympathetic system, all nourished and excited by the blood, any abnormal conditions of these have also their effect: the contractions of the heart being inhibited by impressions made on the terminations of the vagus, and accelerated by such as may be made upon those of the sympathetic. Through the medium of these impressions, afferent and efferent, the heart's action is remotely regulated by the cardiac centre in the medulla. Interference with the functions of these several nerves may so modify the action of the heart as to cause deficiencies of power of every variety and extent, giving rise to illustrations of abnormal contractility and irritability, which the heart exhibits in common with all other muscles. The heart further possesses the distinguishing feature of rhythmical action: and this action is also shown by experiment to be immediately dependent on the blood, whether venous or arterial; for without a supply of blood rhythmical action ceases. There are cogent grounds for the belief that this is not only thus due to the intrinsic ganglionic system of nerves; but, as errors of rhythm are certainly induced by such causes as improper diet, dyspepsia, the presence of worms, constipation, injuries or deformities of the chest, and diseases of the lungs, there can be no doubt that the heart is also liable to reflex irritation of the pneumogastric and sympathetic nerves. The healthy action of the heart, and the controlling energy of its nerves, greatly, if not entirely, depend on the supply of healthy blood; and any abnormal condition of the supply, whether in quantity or quality, shows itself by disturbance not only in the functions of the heart, but in the substitution of hæmic murmurs for the normal sounds. Morbid conditions of the general arterial and venous systems have also their influences; thus

the force and frequency of the pulse, and, more especially, the amount of the peripheral resistance, causing in their tubes dilatation or constriction through the vaso-motor or dilator nerves, and, indirectly, the compensation and relief afforded by the abstraction of fluid through activity in the Malpighian tufts of the kidneys, are to be duly considered. Moreover, the muscular substance of the heart itself is nourished by the blood circulating in the coronary arteries, and thus becomes susceptible to the quality and condition of the blood so distributed; hence, a blood too rich in fibrin or red globules, and thereby inducing plethora, frequently causes over-action of the heart and palpitation, whilst in anæmia a deficient amount of blood induces a weak and often excited and irregular action. The predisposing causes, in addition to those already named, may, therefore, be classed thus: (1) Those conditions acting through or upon the nervous system, such as the general exhaustion of the nervous system, all forms of reflex irritation, venereal excesses, vain longings, purposeless occupations and amusements, protracted mental exercise, abstinence from adequate repose, &c. To these must be added the special temperament and personal peculiarities of the individual, a congenital or superimposed want of vigour, general debility, deformities of the ribs and spinal column, a small weak heart, uterine irritation, hysteria, adynamic fevers, and the special sanitary influences under which the individual is placed. (2) Those conditions acting upon the general blood-supply of the body, and consequently affecting the special blood-supply of the heart, such as the turgid and plethoric states of gross feeders, depraved states caused by bad and deficient diet, and all forms of blood-disorder, as anæmia, gout, scurvy, &c. Amongst the immediately exciting causes may be named mental shock or distress; protracted and unusual physical exertion; various articles of diet, as tea, coffee, &c.; tobacco in excess; many medicines, as aconite and digitalis; as also prolonged abstinence, exposure to cold, and notably blows on the epigastrium.

**SYMPTOMS.**—An increased impulse, indicating a state of general plethora or surcharged circulation, is usually associated with a series of well-marked symptoms, when rapidity of action is superadded—such as flushing of the face; heat and pain of the head, with a sensation of a whizzing, or rushing upwards of the sounds of the heart; dimness of vision, with photophobia; and a tendency to syncope, and to clammy perspirations, with cold shivering. In some rare cases the voluntary muscles, more especially of the lower extremities, may refuse to act, so that the gait becomes tottering, or the patient grasps adjacent objects to steady himself, yet there is neither paralysis nor

vertigo. The respiration, though not generally embarrassed, may become irregular and oppressed, presenting the phenomena of a short inspiration with a prolonged expiration; but should the right heart be congested, independently of any frequency of cardiac impulse, the breathing may be accelerated and accompanied with dyspnœa, or even apnœa, and a short dry cough. Illustrations of this class of symptoms often occur in those of sanguine and nervous temperaments; and may be the result of violent and too protracted exercise, of emotional excitements, or of the over-indulgence in stimulants or food when associated with lives of idleness and inactivity. Should the increased impulse be associated with rhythmical disturbance, there is for the most part consciousness of the existence of such states, more especially on the first ingress of the attack, so that it becomes a source of much anxiety and even of terror, inducing the self-conviction of the existence of organic disease. The head-symptoms also become more marked, and associated with local pains and *tinnitus aurium*; whilst the breathing is marked by sighing, and often becomes lessened in frequency. The irregular form of nervous palpitating heart is often associated also with hæmic diseases, and with nervous affections, as chorea, disturbances due to masturbation, &c. When such diseases as scurvy or chlorosis exist, the attacks become more persistent; there is an increase of pectoral complication, even to dyspnœa; the headache is sometimes so bewildering that the mind becomes alarmed with vague apprehensions of danger, which give rise to general restlessness; the integuments over the region of the heart, as well as of the face, and even of the extremities, may become puffy and œdematous, especially in cases of extreme chlorosis, with enlargement of the thyroid gland and exophthalmos, where the morbid conditions inducing these may also possibly cause the irregular palpitation.

When functional disorder occurs with a diminished impulse, the general symptoms group themselves under anxiety and lowness of spirits, or actual dependancy, with mental and bodily incapacity for exertion; flatulent dyspepsia, with cold clammy extremities; anorexia, or, may be, deprived appetite; exhaustion, with tendency to faintness; and, should irregularity of cardiac action be very marked, there may be a sensation of præcordial pain. Males are more subject to this form of functional disorder than females, and it chiefly occurs in persons having a normally small and feeble heart, or where a state of general nervous debility is superimposed.

When the distinctive feature of functional disorder is rhythmical error, and this is appreciable to the patient, the special symptom is that of extreme anxiety, even to the fear of impending death; occasionally a single

intermission is so prolonged as to induce the impression that escape has only occurred by a miracle. These alarms often induce a palpitation not belonging to the rhythmical disorder. But if the rhythmical error be associated with a deficiency of systolic force, temporary paralysis of the heart's action or syncope may be induced, and in some extreme cases the functions may be weakened even to extinction. Such forms of disorder occur in those having normal but weak hearts; in the dyspeptic; in those subject to gout, especially if an attack is impending; and in those whose habits and occupations involve exhaustion of the nervous system. They are a characteristic of advanced age, but may be sympathetically induced in the young; and may be observed in the course of many diseases, such as tuberculosis, rheumatism, liver affections, or when malignant disease is making its ravages.

Functional disorders of the heart are not infrequently associated with pain. This may have its origin in the structural relations of the heart, though the heart itself be in a normal condition; or its seat may be confined to the præcordial regions. When occurring in the former, its primary source may be in gastric injury or irritations, or caused by other foreign disease transmitting its influences through the sympathetic system; or it may be due to mental distress, in which case the vagus, probably the depressor nerve, proceeding therefrom, and the ganglion of Remac are the immediate causes of spasm. Fainting from emotion is admitted to be due to reflex inhibition of the heart, the afferent impulses reaching the medulla through the vagus; and it may be concluded that this is also the case when pain is the result. In some rare cases, pain thus occurring may be the indication of urgent, or even of fatal, disorder; this is evidenced by the well-known effect of a severe blow in the epigastrium, or in cases of sudden mental shock. These are illustrations of purely functional angina pectoris; but for the most part this symptom has its origin in structural lesion of the heart itself. See ANGINA PECTORIS.

Præcordial pain is by no means an unusual symptom accompanying functional affections of the heart; it may aggravate the urgency of these disorders, yet appears to be little influenced by them. The pain does not march *pari passu* with the irregularity or strength of the impulse. The pain may be persistent, while the associated disorder is in abeyance; and in this respect it differs from *præcordial anxiety*, which is essentially only temporary, though often persistent in its duration, and acquires urgency from the symptoms with which it may be associated.

PHYSICAL SIGNS.—The physical signs referring to the cardiac action may be conveniently separated into the following groups, although in practice they will be found mingled or associated with each other: (1) *Increased*

or *diminished impulse*, connected or unconnected with increased rapidity or rhythmical irregularity; (2) *rhythmical disturbance*, with intermissions, the impulse being normal or diminished; (3) *increased or diminished frequency*, the rhythm and force being normal; (4) *haemic murmurs*, both of the left and right sides.

(1) The cases in which an *increased impulse* is the distinctive feature of the heart's disturbance present many varieties, chiefly referable to force and regularity; but to the simple forms of increased and accelerated impulse there is so very frequently added rhythmical disturbance, that this complication is perhaps the one most usually occurring. The rhythmical disturbance may occur both in the force and the rapidity of the systolic contractions, or it may result in a true intermittence, or occasionally the irregularities thus induced may be so great as to defy definite appreciation, save as a tumultuous whole. On palpation, the impulse, abrupt in stroke, presents the characteristic of a sudden bound, now strong, now failing, sometimes so rapid as to communicate the impression of a fremitus or agitation, then a pause or true intermittence followed by hurry or more evident irregularity; but amid all this irregularity the pulse preserves, in an unbroken surface-wave, the characteristics of efficient valves. On auscultation, the sounds are more difficult of appreciation than in simple palpitation; they are loud and clear, and sometimes so exaggerated and pronounced as to be audible both in the mammary and epigastric regions. But whether the exaggerations of sound and impulse be more or less, they will be found to act in unison with each other; the impulse and sounds increase together and diminish together. When there is an unusual amount of irregular functional excitement in systole, there may be occasionally heard, as a passing, but not persistent occurrence, a reduplication of the second sound, very rarely of the first; and usually the first portion of the divided or cleft sound is the most accentuated. This reduplication, though it may be met with in active inflammatory diseases, is chiefly the concomitant of functional disorders of the nervous heart. The pulse is generally sharp and jerking; it does not always beat in unison with the systole of the heart; if there be plethora, it has a force and fulness not otherwise observable; and if there exist congestion of the right ventricle, it becomes contracted and diminished in force.

In cases where the *impulse is diminished* in effort, the special characters are somewhat negative; the impulse and sounds being feeble, but otherwise normal, unless the systole be excited by mental shock or any undue bodily exertion, when irregularity and increase of impulse, with some slight sharpness of the sounds, take place. The first

sound may suggest, rather than have, a sharp ringing tone; while the second is prolonged.

(2) With respect to errors in *rhythmical action*, separately considered, it must here be noted that the chief and characteristic errors may be classified under the distinct heads of irregularity and intermittence. Irregularity may be in the force or in the frequency of one or more beats, and presents the many varieties which a want of normal uniformity may suggest; the minute and particular enumeration of these is rather satisfying to curiosity than instructive. It is sufficient to say that every variety of irregular frequency may occur; while, with certain beats, force may be increased or diminished. Sometimes there appears to be a kind of order in rhythmical disorders—that is, short series of varying irregularities may regularly succeed each other; there may be a fluttering or trembling, or that vibratory or vermicular motion to which the term ‘thrill’ has been given. True intermittence is not so frequent as irregularity; but when intermittence does occur, it is generally associated with irregularity. These disturbances may be only momentary or of long duration, slight or considerable; but, however this may be, their character is determined by the irregularity in the recurrence of the systole, and the consequent prolongation or retarding of the period of intermission in the systole.

The physical examination of this form of functional disorder shows no marked peculiarities, excepting those of systolic irregularity. To the ear is revealed irregularity in the recurrence and duration of the sounds, from the slightest appreciable pause to the most rapid and confused trembling, with very manifest alterations in tone and pitch. The sounds generally are intensified, the first sound being sometimes heightened to the extent of a sharp knock; whilst the second, save in intensity, is not materially altered. In extreme cases there may be so much ventricular irregularity as to induce in place of sounds an ill-defined fremitus; and so much force as to produce a metallic ringing, with a rubbing murmur on the systole. Thus the impulse of the heart, which in health is rarely appreciable, and its friction never, respectively become so to the touch and to the ear; and the abnormal sounds may exist to such an extent as to entirely obscure the normal first sound.

Occasionally there is met with an appreciable rhythmical disturbance in the pulse, which is not found to exist in the heart—*false intermittence*. The heart only indicates irregularity of power; and as there is occasional failure of force in the already weak systolic contractions, the impulse is not communicated to the artery at the wrist. These false intermissions accompanying irregularity most frequently occur when the heart is op-

pressed by flatus in the neighbouring viscera, or is excited by injurious articles of diet, as tea, or by the use of tobacco. The sensation communicated to the patient is that of a disagreeable flutter or ‘tumbling over’ of the heart, which tends to alarm, although habit may to a certain extent ameliorate the terror.

Rhythmical irregularity occasionally appears as a normal condition, having a life-long existence. Some cases are marked by an extension of the pause, with unsteadiness of the systolic impulse; others by its apparent extinction, so that there exists a rapidity of beats defying all analysis—tachycardia; but tachycardia also occurs either as an habitual state, or in paroxysms (*see TACHYCARDIA*). There is in these cases usually a small weak heart, with systolic impulse devoid of energy. Both these classes of cases present the remarkable feature of losing much of their distinctive irregularity when under the influence of a febrile attack; the slow pulse becomes quicker and more steady, the rapid one less frequent and more distinct; but the sensation to the individual is, nevertheless, not so comfortable as when the heart’s action is in its state of normal irregularity.

(3) Functional disorder occasionally assumes the form of either increased or of diminished *frequency*, while the force and rhythm remain normal. Each of these conditions may be congenital and proper to the individual, or may be the result of abnormal influences. The functionally fast beat is generally induced by other diseases, as fever, diabetes, tuberculosis, &c., and is indicative of injury to normal innervation. The slow and drawling beat is generally met with where the nerve-power is healthy, but the heart itself is weak or fatty; or there is a perverted innervation under the influence of digitalis, aconite, or injury to the ganglionic system—a blow in the epigastrium offering a familiar example.

(4) *Inorganic murmurs* are frequently heard in functional disorders of the heart, and more especially in those cases of hæmic disorder where the systolic impulse is increased, with rhythmical irregularity. These murmurs have the special characters usually attached to such sounds. They are systolic, basic, with conduction in the course of the great vessels. There is no apex-murmur; but at the apex, synchronously with the basic murmur, the first sound is clearly defined, with a metallic ringing second sound. The tone of these murmurs is musical, cooing, soft, of low pitch. The seat is in the aortic valves, and, as a rule, they are always accompanied with palpitation: this palpitation may be persistent, while the murmurs are not so. The murmurs in chlorosis and spanæmia, and, when they occur, in ichorrhæmia and leukæmia, have their seat for the most part in the pulmonary valves, and are not traceable in the course of the larger arteries; they

are also generally associated with the venous hum to be heard in the jugular veins. It is remarkable how large may be the amounts of blood drained from the system, and the frequency of the discharges, provided there be no diseased condition of the blood itself, without inducing the presence of a murmur. But under these circumstances, though there be no murmur, the first sound is usually flapping in character, and the second ringing in tone.

**COMPLICATIONS AND SEQUELÆ.**—The several functional disorders of the heart are often complicated with other diseases—many external to the heart, and some of the heart itself. The more prominent of the former are disorders of the nervous system and of the blood. Many of these have been already referred to. For the most part, those associated with a perverted innervation are examples of irritability, and exhibit rhythmical disorder, with pain. Hæmic diseases induce the simpler forms of palpitation: anæmia, gout, and dyspepsia induce palpitation with rhythmical disorder; spanæmia and chlorosis induce all these disorders, with murmurs superadded. The diseases of the heart with which symptoms of functional disorder, though only occasionally occurring, may be complicated, are mainly degenerations of the walls and valvular lesions. In all these cases the amount and urgency of the functional disturbance of the heart are no indication of the urgency of the disease with which it may be complicated.

Are there any distinct morbid states or other sequelæ traceable to functional disorder of the heart? To answer this question dogmatically may be difficult. Doubtless frequent and prolonged attacks of functional disorder are seen to occur without inducing any such. On the other hand, the long continuance of functional disorder is often marked by a depreciation of mental and bodily vigour. More specific organic changes are generally found to be due to some one or other of the diseases with which the functional disorder has been in its course associated.

**DIAGNOSIS.**—In order to make a correct diagnosis, the first consideration is to ascertain the entire absence of structural disease; or, should it be present, whether it be adequate to cause the full amount of the symptoms exhibited. Supposing this to have been done, if the agitation of the heart is not only excessive, sudden, and apparently increased in strength, even to violence, but presents the features of spasm rather than the calmness of rhythmical order; if the sounds are *pari passu* increased in sharpness and intensity, and diffused over a larger area than is proper to them; and if the pulse does not partake of the simulated force of the heart—the presence of an excited functional impulse may be assumed. The concurrence

of some symptoms usually associated with organic disease, such as dyspnoea or even apnoea and œdema, may, as has been shown, be due to the presence of chlorosis. So also where there is a deficient impulse, if the heart have its normal position and dimensions; if the sounds though weak be natural in tone and quality—it may be concluded that the cause is functional only, in the absence of any abnormal physical disease. The same may be said of rhythmical irregularities. When any of these symptoms are associated with hæmic murmurs, the character of the murmur, its seat, and its persistency must be considered in connexion with the absence or the presence of hæmic diseases. The symptoms of each of these several forms of functional disorder have been so fully described that there is no need to repeat them here. It must, however, be always borne in mind that the absence of the physical signs of disease is not always conclusive of there being no structural lesion, for there may be lesions, and important ones too, that do not yield evidence of their existence. The occurrence of the secondary changes, the immediate result of various congestions, is often an indication that the heart-symptoms are due to the presence of structural disease; still it is not always so, for congestions of the lungs and liver, and œdema, may be the consequence of spanæmia or of other morbid conditions of the blood. Hence, when these are present, the symptoms exhibited by a disordered heart may not be due to structural disease; and the same may be said of the effects of muscular exercise and of position, for either or both of these may distress if there be present any anæmic condition or an intercostal neuralgia. Nor, on the other hand, does the occasional subsidence of urgent symptoms, so frequently the case in functional disorder, absolutely affirm the conclusion that there is no structural lesion; for occasionally in the latter the normal rhythm and force of the heart may reassert themselves; but then in these cases there remain the other characteristics of the organic affection. Repeated examination and an accomplished experience will generally lead to a just diagnosis.

**PROGNOSIS.**—The prognosis of functional disorders of the heart, for the most part, is favourable. Where there are baneful constitutional tendencies, or complications with other diseases, the prognosis must not, however, be always so considered. In the leucoplegmatic temperament the symptoms may be severe and abiding, and generally distressing to the nervous system. If the disordered condition be so urgent as to distend the right heart, the liver may become loaded, and dropsy thence ensue. In this temperament mental shocks may induce palpitation, irregularity, and syncope, whence may ensue not only persistent disorders, having reference to these symptoms, and eventually disease,

but even immediate death. When functional disorder is the concomitant of scurvy or anæmia, the prognosis is not always favourable. Still in the young and middle-aged there is good chance of ultimate recovery; for if these diseases be subdued the functional disorder subsides. When occurring in the aged, or in those having a constitutional tendency to hypochondriasis, or when associated with structural diseases, or excited by inflammations of the endocardium, a less favourable prognosis must be given. Functional rhythmical irregularity, for the most part, does not indicate danger, but it may do so if associated with some obscure structural lesion. Nevertheless, cases of simple functional disorder, so severe as apparently to indicate an immediately fatal termination, prove, for the most part, manageable, and result in a restoration to health. The freedom of the heart from all agitation and other indications of disease, before and after an attack, is due to its being a normal and uninjured organ; and though liable during an attack to the morbid influences of spasms and congestive loading, it still may be a healthy organ. This holds whether the periods of disorder be short or prolonged, occurring rarely or frequently recurring. They are distressing but not dangerous.

**TREATMENT.**—The treatment of functional disorders of the heart should have reference primarily to the mitigation of the symptoms; and, secondly, to the prevention of their recurrence by avoidance or cure of the exciting causes.

*Treatment during the paroxysms.*—Functional disorder with increased impulse accompanied by accentuation of the second sound is to be met mainly by depletory measures. If the symptoms present an aspect of urgency, venesection is called for; but otherwise the promotion generally of the secretory and excretory functions, especially through the portal and renal systems, and the enforcement of a regulated, unstimulating dietary, are the measures to be resorted to. The medicines thus indicated are the alkaline diuretics and the saline aperients, and either colchicum or mercurials in occasional alterative doses. When rapidity of action is superadded, there are two classes of remedies to be resorted to—either sedatives, or the excitants of the inhibitory nervous influences of the vagus. Of the former, aconite, bromide of potassium, diluted hydrocyanic acid, and lobelia are types; of the latter, digitalis and strophanthus are the most reliable; but convallamarin, adonidin, and scillain are members of the same group. When there is diminished impulse or malnutrition of muscle, the causation, as in spanæmia or scurvy, must be met, and the remedies to be adopted are either the sedatives with stimulant action, such as bromide of ammonium or camphor, or the nervine stimulants, such as sumbul,

valerian, or castor. Functional aortic murmurs, generally assumed to be indicative of spanæmia, are to be met by blood tonics, of which iron is the type; but sometimes they would appear to be strictly neurotic in origin, as in some forms of chlorosis, when the nervine tonics, represented by arsenic, zinc, or phosphorus, are preferable. The cause of intermittence is often to be found in the abuse of tea (the tasting habit), and of tobacco; otherwise it is generally to be viewed as a nerve failure, to be remedied by tonics, especially the simple bitters combined with ammonia and stimulants. When irregularity, on the other hand, whether with increased or diminished impulse, is more than probably due to an irritable state of the sympathetic or intrinsic cardiac ganglia, arising in some instances (as in gout) from irritative causes within the blood, sedatives, and especially the alkaline sedatives, are called for. If it be induced by a temporary paresis of the inhibitory centres, whether of the vagus or the ganglion of Ludwig, stimulants are needed. The abuse of digitalis and the like produces irregularity, firstly of the pulse, then of the heart. This becomes, therefore, the warning note in the administration of digitalis; belladonna, on the contrary, excites the action of the heart through the motor ganglia, and can partly annul the inhibitory nervous power when this is not influenced from the medullary centre.

*Treatment between the paroxysms.*—The paroxysm being allayed, it is then well to examine carefully into the state of the health, so as to ascertain if there be any of those disordered conditions which may probably have been its exciting cause. Dyspepsia is to be relieved, the liver is to be set right, the uterine functions are to be restored to regularity, loaded bowels relieved, plethora subdued; spanæmia and chlorosis strengthened into health; exhaustion compensated for, and debility counteracted; and the overworked must seek renovation in travel and cheerful recreation. In persons prone to these disorders many precautions should be observed. The young and the plethoric must avoid extremes of diet and exercise; the food should be moderate in quantity and unstimulating in quality; and exercise should be unfatiguing, and chiefly taken in the open air. Hot and ill-ventilated rooms, and the postponement of sleep by late hours, should be especially avoided. A careful mental discipline should be observed; and this must be sought in a healthy exercise of the brain, and restraining, by a well-ordered intellectual culture, the tendency to vain imaginings and emotional passions. The constitutionally nervous and irritable in mind must sedulously avoid exciting situations, as well as exhaustion by overwork. The sluggish and hypochondriacal must resist the temptations to inaction, seeking to overcome these tendencies by

exercise, healthy mental occupation, and cheerful companionship; by cold bathing, more especially by means of the shower-bath; and, if the bowels be confined, by the judicious use of aperients. As all the varieties of functional disorder of the heart are peculiarly under the influence of a morbid will, it becomes of the first importance that the medical attendant should generally encourage and cheer; and as soon as careful investigation has satisfied the requirements of a just prognosis, further investigations by the stethoscope should be avoided. Repeated investigations tend to prolong the disordered action, and perhaps so to impress the imagination as to forbid recovery.

THOMAS SHAPTER.

### HEART, Hæmorrhage into the Walls of.—SYNON.: Cardiac Apoplexy.

DEFINITION.—Extravasation of blood into the substance of the heart.

ÆTIOLOGY AND PATHOLOGY.—Blood is extravasated into the substance of the heart in various pathological conditions, but as these are described under their respective headings, it will not be necessary in this place to do more than refer to them.

*Rupture of the heart* is the most frequent origin of hæmorrhage into the walls of the organ. The blood in these cases may be derived from the cavity of the ventricle, and forced between the muscular fibres at each contraction. More rarely, a partial rupture of the wall may occur, unconnected with the cavities, and hæmorrhage take place into the seat of the lesion from one of the coronary vessels or their branches, constituting what has been called 'cardiac apoplexy.' In both classes fatty degeneration is generally the cause of the rupture.

The formation of false consecutive *aneurysm of the heart* may be attended with hæmorrhage into the walls; an abscess, blood-cyst, hydatid-cyst, or gumma having burst or made its way into one of the cavities.

The *coronary arteries* may be the source of the hæmorrhage: for instance, in rupture of a coronary aneurysm; in cases of cancerous ulceration of their walls; and in embolism or thrombosis of their lumen, leading to infarction.

*Echymosis of the heart* is a form of hæmorrhage belonging to a different category. It is generally met with in association with parenchymatous degeneration of the heart, for example in the acute specific fevers; with that form of fatty degeneration which is produced by certain poisons, such as phosphorus and arsenic; and with other pathological states in which echymoses occur in the viscera generally, as in purpura and scurvy, and especially in cardiac and pulmonary disease.

In cases of non-fatal hæmorrhage into the walls of the heart, the blood undergoes the

changes usual in extravasations, and gives rise to the collections of pigment-particles which are sometimes found between and upon the muscular fibres; to blood-cysts; or to collections of puriform matter.

Hæmorrhage into the myocardium, whatever its pathology may be, possesses no special clinical relations.

J. MITCHELL BRUCE.

**HEART, Hydatid Disease of.**—A considerable number of cases have been recorded, in which hydatids, in the wider sense of the term, have been found in the human heart. According to the late Dr. Cobbold, 3·5 per cent. of all cases of hydatids in man occur in this situation.

ANATOMICAL CHARACTERS.—Hydatid cysts of the heart are either simple or multiple, the latter being the more common of the two forms. They are situated in the myocardium of either side of the heart; but tend naturally by enlargement to project either into the pericardial sac, or into one of the cardiac cavities, in the form of a prominent cystic tumour. In this condition they have generally been found *post mortem*; but it is probable that in other cases the parasite may undergo degenerative changes in the heart, as in other organs, without its existence being suspected during life or discovered after death. In still other instances the hydatids rupture or are dislodged from their seat in the cardiac wall—either inwards or outwards or in both directions at once. In the first event, the parasite or its contents or fragments becomes impacted in the cardiac cavities or orifices, or give rise to embolism of the great vessels or of a distant branch. Rupture of a cyst into the pericardial sac causes pericarditis; and rupture both internally and externally has given rise to hæmato-pericardium.

Hydatids of the heart are frequently associated with the same disease in other viscera. The appearance and structure of the parasite do not require to be described here. See ENTOZOA.

SYMPTOMS.—In several cases of this disease, the subjects have died suddenly during exertion; or, as in a case recorded by Dr. Wilks, after a hearty meal. These persons were not known to have suffered previously from symptoms referable to the heart. In other instances the ordinary phenomena of chronic cardiac disease were present, including endocardial murmurs; but it is not certain that these were always due to the presence of the hydatids in the heart. A sudden fatal termination will be the result of internal rupture and embolism, or of hæmato-pericardium, as described above.

DIAGNOSIS.—Hydatid disease of the heart does not appear to have ever been suspected during life. Cardiac symptoms and signs, or sudden death, occurring in an individual

known to be suffering from hydatids of other viscera, would suggest that the heart was possibly also affected.

TREATMENT.—The disease cannot be said to have any special interest therapeutically.

J. MITCHELL BRUCE.

**HEART, Hypertrophy of.**—SYNON.: Fr. *Hypertrophie du Cœur*; Ger. *Hypertrophie des Herzens*.

DEFINITION.—An excessive growth of one or more of the tissues of which the heart is composed, the result of which is an abnormal increase in the weight and bulk of the organ.

The walls of the heart are composed of muscular tissue, connective tissue, and fat; and an increase may occur in any one of these elements. Here we have to deal with hypertrophy of the muscular tissue. Increase of the fat and of the connective tissue will be found described under HEART, Fatty Growth on; and HEART, Connective-Tissue Hypertrophy of, respectively.

ANATOMICAL CHARACTERS.—The most important anatomical change in cardiac hypertrophy is an increase of the proper muscular tissue of the heart. There is no growth of new tissue different from the normal heart-muscle: there is an increase in the number and it may be in the size of the muscular fibres. There may be hypertrophy of only one part of the cardiac walls, abnormal thinning being found in other parts. The organ may be greatly enlarged from general dilatation, without any notable thickening of the walls; but the capacity of the chambers should always be carefully noted in estimating the degree of hypertrophy, as there may be a greatly augmented *extent* of wall, although its actual thickness seems normal. Simple hypertrophy is very frequently the first condition, preceding hypertrophy with dilatation. See HEART, Dilatation of.

The increase of weight and bulk may be due to (1) augmentation of the size of the muscular fibrillæ, which may become more than double that of the normal; (2) the formation of new muscular fibres; (3) increased development of blood-vessels; (4) augmentation of connective tissue; and (5) fatty accumulation. In the course of time the hypertrophied tissue may unduly press upon the blood-vessels and contribute to induce degeneration, whilst the continuance or increase of the primary cause of obstruction may lead to dilatation of the cavity (auricle or ventricle), the work of which is obstructed.

GENERAL CONSIDERATIONS.—The weight of the heart of the infant at birth becomes more than doubled at the age of two years. Between seven and fourteen years of age it is about eight times the weight at birth; from fourteen to twenty the increase is such that the weight is again nearly doubled. There is, speaking generally, progressive augmentation even to old age. Wilhelm Müller, how-

ever, has shown that the progressive increase of weight with age is due rather to the augmentation in the weight of the great vessels within the pericardium than to that of the heart-substance itself. There has been a general consensus amongst observers that the heart of the male is, as a rule, heavier than the heart of the female. W. Müller has shown, however, that this is only because the weight of the heart is proportionate to that of the body. Up to the body-weight of 44 lbs. the heart is relatively heavy as compared with the body, whilst from 44 to 176 lbs. its proportional weight diminishes. Witness the extremes as following: Proportion of weight of heart to that of body, ages 1 to 10, male .0059; ages 80 to 90, female .0028.

The average weight of the female being less than that of the male, the heart of the former is of course less. It will be readily seen that, as the weight of the heart varies with that of the individual, one cannot express with exactitude any figure as the normal weight. We may roughly say that the heart of the adult should weigh about 10½ oz. One that weighs 12 oz. is hypertrophied. The weight of a hypertrophied heart may vary between this and the enormous figure of 60 oz., which has been recorded; not uncommonly it reaches 27 to 30 oz. In taking the thickness of the muscular walls of the ventricles as a standard, we may regard a left ventricle of an adult as hypertrophied when the breadth of the thickest portion surpasses six-tenths of an inch, and a right ventricle as hypertrophied which measures over a quarter of an inch. In hypertrophy the thickness of the left ventricular wall and of the septum has been found as great as 30 and even 40 millimetres (1¼ inch and 1½ inch). The columnæ carneæ may be greatly enlarged in hypertrophy, but the hypertrophy chiefly tells upon the walls of the ventricle.

The walls of the auricle may be considered as hypertrophied when the thickness exceeds 4 millimetres (about one-eighth of an inch).

VARIETIES OF HYPERTROPHY.—Bertin in 1811 described three forms, which most succeeding writers have referred to, namely: (1) *simple hypertrophy*, in which the parietes of the compartments are thickened, the cavities retaining their natural dimensions; (2) *hypertrophy with dilatation (eccentric hypertrophy)*, in which the cavities are increased in capacity, while the parietes are either of natural or of augmented thickness; (3) the so-called *concentric hypertrophy*, or *hypertrophy with diminished cavities*, in which new material was supposed to be added, chiefly in the interior of the ventricular walls. Cruveilhier and Budd pointed out that the condition called concentric hypertrophy is the result, not of hypertrophy, but of a powerful contraction of the organ suddenly arrested, as it were, by death. Budd found that the hearts of persons who had

died a violent death presented this so-called concentric hypertrophy; but that such hearts became relaxed, and showed the normal size of cavities and thickness of walls after maceration. Rokitansky and Bamberger acknowledge the rarity of concentric hypertrophy, but think it does sometimes occur. There can be little doubt that *concentric hypertrophy* of the ventricles has no real existence. The appearances which have given origin to the term are due, in the great majority of instances, to the occurrence of *rigor mortis* in the muscle of the ventricle, which has caused firm contraction and thus diminution, to almost obliteration, of the cavity. If a heart presenting such signs be soaked for some hours in warm water a gentle dilatation with the finger will restore the ventricle to its normal dimensions. The left ventricle may simulate concentric hypertrophy in some cases of mitral stenosis when the disease has developed in early childhood. The ventricle has not received its normal blood-contents, and development has proceeded disproportionately in the right chambers and the left auricle. The cavity of the left ventricle has thus been left relatively small till the time of death. In like manner, in congenital stenosis of the pulmonary artery the *right* ventricle, hypertrophied in intra-uterine life, may so continue, and its cavity never become dilated to the normal extent. The ventricles are much more frequently hypertrophied than the auricles, and the left ventricle more frequently than the right; but the right auricle more frequently than the left auricle—which last shows the change very seldom.

**I. Hypertrophy of the left ventricle.**  
**ANATOMICAL CHARACTERS.**—The whole heart appears to be elongated, and the apex-portion of the left ventricle especially prolonged. The walls of the left ventricle and the inter-ventricular septum are thicker and firmer than in the normal: the septum may bulge into the right ventricle, diminishing its capacity and even producing actual obstruction of its conus (Coats). In transverse section the thickening and bulging of the septum are very manifest, the right ventricular cavity presenting the appearance of a crescentic appendage to the left chamber.

**ETIOLOGY AND PATHOLOGY.**—Sir Richard Quain (*Lumleian Lectures*, 1872) has classified the *exciting* causes of hypertrophy under three heads—*nervous*, *mechanical*, and *nutritive*.

(1) Amongst *nervous* causes are prolonged mental excitement or strain, and those emotional conditions which produce frequent palpitation. The immoderate use of strong coffee, tea, or spirituous liquors might come under this head. It is not in all cases that a heart which beats for long periods at an abnormal rate of rapidity becomes hypertrophied, inasmuch as the general nutritive

processes may be feeble or imperfect in some persons, active or vigorous in others. In cases of rapid heart (tachycardia) the writer found evidences of enlargement in three only out of seventy-five cases; in some the heart seemed even smaller than the normal. In Graves's disease, when there has been abnormal rapidity of heart, persistent or paroxysmal, for several years, the cardiac area has been sometimes found not larger than under conditions of health, and in many cases the *post-mortem* examination has demonstrated no morbid change. The just conclusion, therefore, seems to be that for the production of hypertrophy of the left ventricle in cases of nervous excitation there must be an additional factor to rapidity—either a thickening of the arteries, or a vasomotor irritation causing contraction of their muscular walls, whereby the intra-arterial tension is increased, or a quantity or quality of blood affording better nutrition. If the blood-pressure be at or below the normal, the rapidly beating heart does not tend to hypertrophy.

(2) Amongst *mechanical* or *physical* causes are all those obstructive conditions to be specially examined presently. Violent and long-continued athletic or other exercises, which notably accelerate the contractions of the heart, or produce excessive blood-pressure, may be mentioned here. It has been pointed out that great muscular exertion with the arms is specially prone to cause hypertrophy, as in the case of hammer-men, &c. Prolonged working in a bent or constrained position is also mentioned as a cause.

In some callings and conditions in which muscular effort is severe, hypertrophy of the left ventricle is prone to occur—*e.g.* Cornish miners (Peacock), dwellers in hilly regions, especially those accustomed to carry heavy loads (Münzinger, Allbutt), stonemasons, slaughterers (Leyden). In many cases it is probable that the two factors just mentioned—nervous excitation and muscular overstrain—combine to produce the hypertrophy. So may be best explained the signs and symptoms in the cases of 'irritable heart' of soldiers during the American Civil war (Da Costa), those in our own Foot Guards who carried heavy accoutrements (Myers), and those in the Franco-German war (Fraentzel) (*see* EXERCISE). The excitement of a soldier's life co-operates as a cause with the muscular effort, which may be excessive and either prolonged or violent and fitful. It is a significant observation of Hirsch that there has been a notable increase in the proportion of heart-diseases in a community at times of great political and social excitement. This was observed in France during the great Revolution and after the political agitation of 1830, in Rome after 1848, in Sicily in 1860, and in the Argentine States after the political confusion and civil war.

(3) *Diseases of the valvular apparatus*—aortic and mitral—induce hypertrophy of the left ventricle by causing an obstruction to the outflow of blood or an abnormal blood-pressure within the cavity from regurgitation.

(a) *Stenosis of the aortic orifice* is a common cause of hypertrophy of the left ventricle. The opening is not only narrowed, but is also rendered more rigid, and thus increased force is necessary to propel the blood. Along with the valvular lesion there may also be, especially in advanced life, a loss of elasticity and a roughening of the inner coat of the aorta from degenerative changes—conditions which increase the mechanical strain upon the left ventricle.

(b) *Aortic regurgitation* often induces so great enlargement of the left ventricle, from hypertrophy and dilatation, that the heart in such cases merits the name *cor bovinum*. The back-flow of blood increases the intra-ventricular pressure, tends to dilate the cavity, and calls forth augmented efforts of ventricular contraction. In some cases of injury and rupture of the aortic valves it has been possible approximately to estimate the rate of hypertrophy. In two cases, related by Dr. Stone, of injury to these valves by blows upon the chest, assuming that the heart was healthy previous to the injury, there must have been an increase of nearly an ounce a week during the four or five weeks that elapsed before death. (c) *Mitral regurgitation*. Insufficiency of the valve at the left auriculo-ventricular orifice of necessity causes the left ventricle to be subject to abnormal pressure, with the result that it becomes hypertrophied as well as dilated.

(4) *Aneurysm of the aorta*.—There has been much difference of opinion on the question whether aneurysm of the thoracic aorta is a cause of hypertrophy of the left ventricle. The divergence is capable of explanation. When the situation of the aneurysm is at the commencement of the aorta, where it involves the semilunar valves, hypertrophy is the rule. When the aneurysm is so situated as not to involve the valves, hypertrophy is a rare exception. This is proved to demonstration by the following figures, deduced from the records of the London Hospital, the cases being collected by Dr. F. J. Smith, medical registrar. Of forty-one cases of aneurysm of the thoracic aorta in which the valves were *not* implicated, enlargement of the left ventricle was evidenced in only nine instances, and in three of these the hypertrophy was recorded as only slight. In forty-one cases in which the valves *were* involved, hypertrophy and dilatation were manifested in thirty-five.

(5) *Renal disease*.—Hypertrophy of the left ventricle may occur in all forms of Bright's disease, the lardaceous form excepted. Of these *chronic Bright's disease* (contracted kidney) is a very important cause, the hyper-

trophy being often of the purest type, without dilatation. This change is the result of the great increase of blood-tension produced by the resistance offered to the blood in the small arteries through the whole body, as well as in the kidneys.

(6) *Pregnancy*.—It has been shown by many observers that the left ventricle tends to become hypertrophied and the arterial tension augmented during pregnancy. The hypertrophy may continue for a considerable portion of the period of suckling, and then the heart, like the uterus, undergoes a process of involution. According to Duroziez, the ventricle tends, after many pregnancies, to remain hypertrophied.

(7) *Excess of food*.—With regard to the *alimentary* causes of hypertrophy of the heart, the state of the local nutrition and the nutritive quality of the blood have both to be taken into account. Rich nitrogenous food, and the use of ferruginous medicines, will favour hypertrophic changes. With the increase of the muscular structure there is proportional enlargement of the coronary arteries, so that the hypertrophied organ has an increased blood-supply.

PHYSICAL SIGNS OF HYPERTROPHY OF THE LEFT VENTRICLE.—In simple hypertrophy (*e.g.* when occurring in athletic persons, or in relation with chronic renal disease in the earlier periods) the wall of the chest on *inspection* may be observed in the neighbourhood of the apex to be raised with each ventricular systole. Pulsation, also, of the carotid arteries in the neck may be observed, such pulsation being rather prolonged, and not sudden (as in aortic regurgitation), unless there be concurrent fever. On *palpation* the fingers are lifted by the apex, the impulse of which is prolonged and heaving. The duration of the heaving impulse is in proportion to the degree of hypertrophy. The impulse is often strong enough to visibly move the bedclothes, and even to raise the head of the auscultator by the impact against the stethoscope. Of course a larger portion than usual of the heart's surface impinges against the chest-wall. The outline of the apex-portion of the heart, as obtained by *percussion*, is acutely triangular, its acute angle pointing downwards and to the left. This becomes broader and more rounded as dilatation preponderates over hypertrophy. In simple hypertrophy the heaving apex may be in the sixth or seventh, rarely in the eighth, left intercostal space; but in dilatation with hypertrophy the area downwards and towards the left axilla is greatly increased. On *auscultation* the first sound at the apex is found to be dull and prolonged. It is the sound of muscular contraction that the ear in the greatest degree appreciates, the element of valve-tension being muffled by the thick wall of the ventricle. Over the second right costal cartilage the second sound

is loud, and may have a ringing character; it may sometimes be the only one of the two normal sounds audible. In this situation it is much louder than over the second left interspace (the commencement of the pulmonary artery) unless the ventricular hypertrophy is accompanied by mitral insufficiency. If with evidence of powerful left ventricle and firm arteries the aortic second sound is not loud, but dull and ill-pronounced, there is strong probability that the aortic cusps are swollen or thickened from disease. In later stages of hypertrophy, when the muscular tone of the left ventricle is impaired, the sounds of the heart may have a triple rhythm, resembling the cantering of a horse—the *bruit de galop*, carefully studied by Potain. This is especially observed in the hypertrophy associated with renal disease, and in the hypertrophy with dilatation accompanying gastro-hepatic derangement.

The *radial pulse* is felt to be prolonged; the artery is firm and resisting, and its coats may be thickened. In the hypertrophy resulting from muscular overstrain the pulse is large and full, the first wave is prolonged, and there is no diastole. In chronic renal disease, as the arterial coats become thickened the amplitude of the pulse is manifested in less degree, the vessel being felt hard, resisting, and full between the beats. In some cases the fullest force exerted by the finger may fail to entirely obliterate the pulse, and on the distal side of the point compressed the wall of the artery may be felt cylindrical and firm. In the hypertrophy associated with aortic stenosis the pulse is somewhat narrow and gradual both in its rise and fall. In that manifested with aortic incompetency it is sudden and powerful in its impact against the finger, but rapidly receding, so that the artery seems empty between the beats—it is the collapsing or water-hammer pulse.

**SYMPTOMS OF CARDIAC HYPERTROPHY.**—It must not be forgotten that simple hypertrophy may exist without producing symptoms attracting the attention of the patient, and that there is a natural tendency to some degree of cardiac hypertrophy with the advance of age. *Dyspnœa.*—In moderate hypertrophy without complication, there is usually easy and natural breathing when the patient's body and mind are at rest. But mental excitement or bodily effort at once induces more or less of temporary dyspnœa. In some cases the due expansion of the lungs may be mechanically impeded by the increased volume of the heart. In eccentric hypertrophy with dilatation, more especially when the right cavities are thus affected, pulmonary congestion and œdema are usually present, and then marked dyspnœa is a prominent and distressing symptom. *Cough.*—In simple hypertrophy there may be an occasional dry, irritating cough, and in young

plethoric women a wheezing cough may be complained of. In right-side enlargements, when pulmonary obstruction and dropsical effusions supervene, cough is, in most cases, a very frequent and painful addition to the other sources of discomfort to the patient. *Hæmoptysis and other hæmorrhages.*—Hæmoptysis from capillary engorgement is common, being generally active and sudden. Niemeyer points out that, in left-heart hypertrophy, there is often active distension, and sometimes rupture, of the branches of the bronchial arteries. In left hypertrophy, too, the cerebral arteries are specially liable to give way. In right-side enlargement with pulmonary obstruction, the blockage may influence the vessels of the liver and the portal system generally, so as to produce hæmatemesis or melæna. Epistaxis may also be due to cardiac enlargement. *Palpitation* is a common symptom in all organic diseases of the heart, and is often very marked in cardiac enlargements. The least excitement, bodily or mental, may induce a greater or less degree of this symptom. Especially in eccentric hypertrophy with dilatation, most distressing paroxysms of palpitation are apt to occur from time to time. Besides bodily and mental excitement, other conditions, such as indigestion, flatulence, or an overloaded stomach, readily call forth this symptom. When there is much dilatation, the palpitation may be irregular and intermittent, and is then more particularly a very alarming symptom.

The *renal and urinary* derangements are very important. In the hypertrophy of the left ventricle, associated with chronic Bright's disease, there will be found the copious excretion of a pale urine of low specific gravity. Albumen may be found in small quantity, and may sometimes be absent. In the hypertrophy associated with gastro-hepatic disturbance, the urine will be scanty, of high specific gravity, often thick with urates, containing much urea, and dark with the colouring-matter of the urine, of the bile, or of both.

Certain *cerebral symptoms* ought to be mentioned in connexion with the other more direct signs of cardiac hypertrophy. A feeling of fulness, or perhaps of throbbing, may be felt in the head after great muscular exertion or mental excitement. In pronounced cases there may be headache, ringing in the ears, vertigo, *muscæ volitantes*, and disturbing dreams. The bright and shining, or perhaps bloodshot, condition of the eyes is an indication of the hyperæmia of the cerebral vessels.

**II. Hypertrophy of the right ventricle.**—The normal weight of the right ventricle is probably less than half that of the left (average in men: left ventricle 173 grammes, right 72; in women: left ventricle 150, right ventricle 62. Du Castel).

The capacity of the right ventricle is generally greater than that of the left—according to recent researches in the proportions of about seven to six. The walls are from a quarter to one-third the thickness of those of the left.

In the great majority of cases the right ventricle becomes hypertrophied only in association with a morbid change in the left, and when such hypertrophy occurs it is nearly always accompanied by dilatation. There are exceptions to this rule, as will be presently seen.

The right ventricular wall may be thickened to the extent of one inch, instead of the normal two and a-half lines; its greatest thickness is at its base. The columnæ carneæ of the right ventricle are even more liable to hypertrophy than the wall. In dilatation with hypertrophy the columnæ carneæ become stretched and attenuated. The substance of the hypertrophied left ventricle can generally be torn with ease, whilst that of the hypertrophied right ventricle is usually tough and leathery.

**ÆTIOLOGY AND PATHOLOGY.**—The right ventricle may be observed in a state of simple hypertrophy in—

(1) *Congenital anomalies of the heart.*—Chief of these is stenosis of the pulmonary artery, which in the majority of cases results from fetal endocarditis. The obstruction may be below the valves, the conus of the right ventricle being narrowed. In each case the right ventricle becomes hypertrophied in consequence of the obstruction in front, and there is no dilatation of the cavity.

(2) In *emphysema, fibrosis, and consolidated or compressed conditions of the lungs*, the impediment to the pulmonary circulation induces hypertrophy with dilatation of the right ventricle.

(3) In the subjects of *curvature of the spine* the capacity of the thorax is nearly always diminished; the lungs are emphysematous, and often suffer other pathological changes. The two branches of the pulmonary artery may be unequal, owing to the differences of development of the two lungs. The right ventricle is dilated as well as hypertrophied.

(4) *Endocarditis affecting the tricuspid valve.*—It is rarely that endocarditis attacks the valvular apparatus of the right side of the heart without that of the left side participating in still greater degree, except in intra-uterine life. Byrom Bramwell has shown the great probability that when endocarditis affects the valves of the left side it frequently involves also the right, though the inflammatory changes in the latter may be evanescent. Such cause may co-operate to impair the muscle of the right heart and hasten dilatation. In some cases (and these not very infrequently) the result of endocarditis is stenosis of the tricuspid orifice, in

which case the right ventricle tends to hypertrophy rather than to dilatation, whilst the right auricle becomes greatly dilated.

(5) The most common cause of hypertrophy with dilatation of the right ventricle is *valvular disease of the left side of the heart*. Whether there be mitral stenosis or regurgitation, the consequences are hypertrophy of the right ventricle to antagonise the opposing forces, and dilatation on account of the persistent overstrain.

**SIGNS AND SYMPTOMS OF HYPERTROPHY OF THE RIGHT VENTRICLE.**—In enlargements of this ventricle, *inspection* may reveal a rounded smoothness of the epigastrium, with perhaps some bulging of the ensiform and lower left costal cartilages. The apex-beat may be seen to be very diffused, extending towards the tip of the ensiform cartilage. Facial lividity is frequently seen; and jugular pulsation may be observed when there is tricuspid regurgitation. *Palpation* over the lower part of the sternum detects an impulse, which feels as if immediately under the hand, and usually wants the heaving character of the impulse of a hypertrophied left ventricle. Epigastric pulsation is often very pronounced. The liver-pulsation in such cases may result either from venous regurgitation or from right systolic action exerted through the diaphragm. On *percussion*, the inferior line of dullness is found to extend lower down and farther towards the right than normal, sometimes reaching an inch or more beyond the right sternal edge. On *auscultation*, the second sound over the pulmonary valves is found to be louder than over the aortic. The accentuation of the pulmonic second sound, however, is not so marked as in cases of well-compensated stenosis or insufficiency at the mitral orifice. The aortic second sound is weak, and though palpation may detect a forcible impulse at the apex of the heart, the radial pulse may be felt small and contracted. In some cases a triple sound—the *bruit de galop*—may be heard over the right ventricle.

The *radial pulse* in hypertrophy of the right ventricle is usually small and resistant. There is a difficulty in the passage of blood through the pulmonary circuit, and the supply to the left chambers is reduced in amount; moreover, the blood is imperfectly oxygenated and, containing an undue proportion of carbon dioxide, it stimulates the vaso-motor influences and augments the intra-arterial tension.

**III. Hypertrophy of both ventricles.**—**COMPLICATIONS AND SEQUELÆ.**—Simple hypertrophy of the heart may go on quietly for a long time, just balancing the obstructive influence, and giving rise to no other form of disease. But when dilatation co-exists with hypertrophy, palpitation, dyspnoea, venous congestion, and serous effusions are the ordinary results. Diseased conditions of the arteries may occur simultaneously, or

may be induced by the long-continued additional strain put upon them by a hypertrophied heart. Cerebral hæmorrhage often occurs in connexion with a hypertrophied left ventricle, as in Bright's disease; although there are always perhaps other factors, besides the mere excessive propulsive power of the heart, in the production of apoplexy. Pulmonary and general congestion and œdema are the usual attendants of mitral lesions with right-side enlargements. Pulmonary hæmorrhagic infarction (the so-called 'pulmonary apoplexy') generally results from engorgement of the branches of the pulmonary vessels, and takes place in connexion with right-heart enlargement. Sanguineous exudation in the tract of the bronchial mucous membrane may occur in left-heart hypertrophy. Persons suffering from cardiac hypertrophy are apt to be gravely affected by acute febrile diseases, because the resultant acceleration of the heart's action increases the embarrassment of the organ. Of course, hypertrophied cardiac walls are subject to the fatty degenerative changes described elsewhere.

**IV. Hypertrophy of the auricles.**—Hypertrophy of the *right* auricle is scarcely ever met with except in association with dilatation of the cavity. Simple hypertrophy is only possible in the case of tricuspid stenosis occurring in infant life. Hypertrophy and dilatation are marked in cases of mitral disease, both regurgitant and obstructive. The hypertrophy is in greater degree in the obstructive lesion. In both, but especially the latter, the auricle may be sufficiently powerful to cause a back-flow into the venous channels by its systole, and thus a venous pulsation, presystolic in time, in the veins of the neck. Hypertrophy of the *left* auricle occurs in greatest degree in mitral stenosis. One of the writers has found in such case, in a boy of nine, a left auricle varying in thickness from one-eighth to one-quarter of an inch—*i.e.* the thickness in some parts being nearly that of the right ventricle of an adult. In some cases—in children—the hypertrophied auricle may be observed to cause a distinct presystolic pulsation in the third left intercostal space at the outer portion of the cardiac dulness. As the case progresses, dilatation occurs, and the muscular wall becomes thin. An increase of dulness to the left over the situation of the left auricle is often manifested on percussion in cases of mitral stenosis.

**V. Hypertrophy of the whole heart.** This is manifested in the highest degree in cases of combined aortic and mitral disease; either when a rheumatic lesion has induced mitral and aortic regurgitation coincidentally, or when the initial lesion has been aortic regurgitation, and mitral inadequacy has resulted from the abnormal intra-ventricular pressure. In mitral disease, however, when

the aortic valves are intact, the hypertrophy and dilatation of both ventricles may be very considerable. Differences of opinion have been expressed as to the influence of *pericardial adhesions* in determining hypertrophy of the heart. It can scarcely be doubted that pericarditis is a potent factor of hypertrophy during the period of childhood. A heart presenting signs of almost universal pericardial adhesion will be often found to evidence a far greater degree of hypertrophy than could be ascribed to any coexisting valvular imperfection. When the period of heart-growth has ceased, in fully adult life, the consequences may be different, and no hypertrophy, but, on the contrary, wasting, may ensue.

**PATHOLOGY.**—True muscular hypertrophy of the heart is compensatory, or conservative. The heart obeys the law that muscle tends to increase in force and in development when an obstacle is imposed to its normal working (*see* HYPERTROPHY; and HEART, Valves and Orifices of, Diseases of). We have just seen what the obstacles are in hypertrophy of the different parts of the heart. Some obstacles are direct, such as stenoses; and hence by this change the heart is enabled to overcome deficient valvular action or obstruction to the blood-current, the increased peripheral resistance of Bright's disease and of emphysema. Other lesions are indirect or relative obstacles, particularly valvular incompetence, which permits over-filling of the cavities, and demands a corresponding increase of vigour in the systolic discharge, which leads to hypertrophy.

**DIAGNOSIS.**—An extended area of dulness, displacement of the apex-beat downwards and to the left, and a slow, heaving systolic action, with augmented force of impulse, are the chief diagnostic physical signs of cardiac hypertrophy. In *young and thin people* the last of these signs may seem to be present, but the accompanying conditions readily exclude hypertrophy, especially the non-extension of the cardiac dulness. An *emphysematous left lung* may mask hypertrophy when present; and *lung-consolidation* might, though only for a moment, suggest it when absent. In *pericardial effusion* the triangular shape of the area of dulness, with the apex of the triangle upwards, is a distinctive feature; there would, moreover, be the history of an acute disease, with lancinating pain, dyspœnia or suffocative sensations, and other symptoms not found in mere enlargement of the heart. *Pleuritic effusion* or *aneurysm* would be still more readily discriminated. The differential diagnosis between left-heart and right-heart enlargements has been sufficiently discussed in speaking of the symptoms and signs. *Dilatation*, as distinguished from hypertrophy, is characterised by the feebleness and diffuse-

ness of the apex-beat, which may even be quite imperceptible; by the great irregularity and intermittency of the heart's action; and by the general signs and symptoms of a feeble circulation.

**PROGNOSIS.**—Simple, uncomplicated hypertrophy, as in the young and in athletes, is not incompatible with long life, if the cause be removed in time. According to the extent and degree of complication, whether in the form of valvular lesions or co-existent pulmonary disease, the prognosis will be unfavourable. When the cardiac change is itself producing secondary lesions, as degeneration of the arterial coats, when dilatation is advancing, and when there is Bright's disease, the prognosis becomes very unfavourable.

**TREATMENT.**—Hypertrophy being in itself a conservative change, protective from worse results, the primary object is to remove, if possible, the cause of the hypertrophy. To aim merely at reducing the hypertrophy, irrespectively of its cause, as by lowering the nutrition, would gravely favour the worse evil of dilatation. General therapeutic principles, and the morbid conditions accompanying the hypertrophy, must therefore be carefully attended to. All mental and bodily exertion which excites the circulation must be scrupulously avoided. Alcoholic stimulants should be taken sparingly, and no more wine allowed than such as may seem to benefit digestion. The diet should be carefully selected, nitrogenous food being generally necessary. The digestive organs must be sedulously looked after, not only because good nutrition is very important, but also because flatulence and dyspepsia directly embarrass the heart's action. Mild saline and aloeitic aperients with alteratives should be given. Diuretics will be necessary if there is a tendency to dropsy, and in all cases great attention must be paid to the removal of congestion when it affects important organs, and the restoration of their functions when affected, more especially of the liver and the kidneys. When there is great excess of cardiac action, direct cardiac sedatives, as aconite, diluted hydrocyanic acid, conium, and perhaps the local application of ice, may be called for. When there is dilatation or feebleness of texture with the hypertrophy, iron and digitalis are the chief remedial drugs.

J. R. WARDELL. A. E. SANSOM.

**HEART, Inflammation of.**—Inflammation of the heart may affect either the lining membrane, or the substance or walls of the organ; and the subject will be best discussed under the separate heads of *endocarditis* and *myocarditis*. Inflammation of the investing membrane of the heart is described in the article **PERICARDIUM**, Diseases of.

**I. Endocarditis.**—**SYNON.**: Fr. *Endocardite*; Ger. *Endocarditis*.

**DEFINITION.**—Inflammation of the lining membrane of the heart.

Three forms of endocarditis are commonly described: (1) Simple endocarditis; (2) Ulcerative endocarditis; and (3) Chronic endocarditis. Only the two first forms will be discussed here; chronic endocarditis is referred to under the head of **HEART, Valves and Orifices of, Diseases of**.

**1. Simple Endocarditis.**—**ÆTIOLOGY.** Endocarditis generally occurs in association with acute rheumatism; less frequently with other acute specific febrile diseases, such as scarlet fever, measles, tonsillitis, erythema, pyæmia, and septicæmia—including puerperal fever; much more rarely with typhoid fever and variola. It may be observed in the course of pregnancy, and after parturition; in acute and chronic Bright's disease; and in syphilis. Injuries of the heart, such as rupture of the valves, may also lead to endocarditis; and local endocarditis is frequently the result of the unnatural contact of one part of it with another during the cardiac revolution, as, for example, by growths from the walls or valves, or by unnatural blood-currents. It is also common in chorea. Possibly it may be idiopathic.

Age is an important predisposing factor in the ætiology of acute endocarditis, the occurrence of which as a complication of acute rheumatism is certainly most frequent in young subjects, and declines as age advances. Women also are more subject to rheumatic endocarditis than men.

The localisation of the endocardial inflammation appears to be determined chiefly by pressure and tension, rather than by any peculiarity of the membrane itself, or of the blood in contact with it. Thus the left ventricle is almost the sole seat of the disease (at all events of readily recognisable degree) in the adult, and the right ventricle in the fœtus; whilst endocarditis is rarely seen beyond the boundaries of the valves, that is, the parts most subjected to strain. In the same way, chronic endocarditis is usually due to increased pressure within the heart, as in chronic Bright's disease, and in conditions that entail prolonged severe strain upon the valves during exertion. A similar cause is at work in pregnancy.

**ANATOMICAL CHARACTERS.**—Inflammation of the endocardium affects chiefly the valves and the chordæ tendineæ, and especially the lines of contact or the surfaces of the valves exposed to the force of the blood-current. The endocardium of these parts at first appears slightly swollen, velvety, soft, and of various shades of red; whilst the lines or points of contact of the valves present warty enlargements of a similar character, which are known as 'vegetations.' As the process

advances, the inflamed areas become more opaque and firm; and a fibrinous deposit is entangled with their surface. When the endocarditis has gone thus far, resolution is probably rare; and the most common result is what is known as 'chronic valvular disease,' that is, that the affected parts are left opaque, puckered, and thickened by growth of connective tissue, whilst the vegetations develop into firm fibroid or even cartilaginous-like bodies. As a consequence of these changes, the valves may become much altered in size and shape, and the ostia contracted and irregular, so that the mutual adaptation of the parts is greatly disturbed. Other results of inflammation are not uncommon in the progress of endocarditis, such as adhesions between the neighbouring structures, and ossification or calcification of the altered tissues. Laceration of the valves and rupture of the chordæ tendinæ during the stage of diminished resistance, ulceration, suppuration, and the formation of aneurysm are rarer events.

The microscopical appearances of inflammation of the endocardium correspond with the naked-eye characters. In the early stages, the proper tissue of the endocardium is swollen by hyperæmia, œdema, and the appearance between its fibres of a number of leucocytes; the latter rapidly multiply to form the bulk of the vegetations; and the surface presents various thicknesses of deposited fibrin, which in its turn may become organised. The further development of connective tissue gives rise to the opacity, thickening, and puckering of the valves, and to the formation of permanent vegetations.

The effects of these changes upon the functions of the valves and their appendages are described in the article HEART, Valves and Orifices of, Diseases of. Particles of fibrin, and even of the vegetations, are occasionally detached from the endocardium, and give rise to embolism.

**SYMPTOMS.**—The symptoms of endocarditis are inseparable from the symptoms of the disease with which it is associated, and the diagnosis of it is made almost entirely from the presence of physical signs. Thus fever probably precedes the advent of endocarditis in every case; and it cannot be said that the simple uncomplicated disease in any respect affects either the pyrexia or any other element of the same. Local symptoms are almost equally rare, unless the endocarditis leads to serious lesion of the cardiac valves. As long as these remain sound, and the disease is acute and does not involve deeper structures, pain in the heart, præcordial distress, syncope, shortness of breath, and other symptoms of heart-disease cannot be said to occur at all frequently in endocarditis. The cardiac contractions are necessarily increased in frequency; and palpitation and dyspnœa may occur on movement. It is otherwise when the inflammation has lasted so long

as to render the valves incompetent, or to obstruct the orifices; or when the myocardium is attacked, and dilatation ensues. The symptoms just enumerated then make their appearance. In children, rheumatic endocarditis is peculiarly latent, and also dangerously progressive or recurrent. See RHEUMATISM, ACUTE.

**Physical Signs.**—The physical signs of acute endocarditis are—increased extent and frequency, with variable strength, of the visible and palpable impulse; moderate increase in the area of præcordial dulness; and various alterations in the cardiac sounds. At the beginning of endocarditis, the first sound at the left apex is frequently heard prolonged and hollow, or muffled; and, as the process advances, this alteration of character may gradually pass into a murmur, which is at first indistinct, but afterwards well-formed. If the aortic valves are affected, the second sound may similarly lose its characters, become dull, and finally be converted into, or be complicated with, a murmur. The most frequent murmur in acute endocarditis is mitral systolic; aortic murmurs are decidedly less common; and mitral præ systolic murmur is very rare. Various murmurs, inorganic or organic, may also appear and disappear during the course of the disease.

**COMPLICATIONS.**—Endocarditis is itself always a complication of the diseases previously mentioned. Myocarditis and pericarditis may be correctly regarded as complications of endocarditis, when the inflammation begins in the lining membrane of the heart. According to some authorities, clots may form in the heart in endocarditis, and give rise to very urgent symptoms (*see* HEART, Thrombosis of). Embolism may arise from detachment of fragments of coagula or vegetations. Congestion or inflammation of the lungs frequently occurs in association with endocarditis, and so may albuminuria.

**COURSE, TERMINATIONS, AND SEQUELÆ.**—The course of simple endocarditis is very uncertain, and varies with the course of the original disease with which it is associated, as well as with the complications. If acute rheumatism be quickly checked, inflammation of the endocardium will be also arrested; but the two diseases may recur together. In a considerable number of cases, however, endocarditis passes on to chronic valvular disease. The late Dr. Sibson found that seventeen out of seventy cases of endocarditis with mitral systolic murmur ended in established valvular disease, and less than a half of the cases with diastolic basic murmur.

Simple endocarditis very rarely proves fatal, but, being by far the most common cause of valvular disease of the heart, it leads indirectly to much suffering, and in such cases as a rule ultimately to death.

**DIAGNOSIS.**—The diagnosis of endocarditis depends upon the discovery of the develop-

ment of an endocardial bruit of organic origin during the course of one of the diseases already named. From functional murmurs the bruits of valvular inflammation may be diagnosed under different circumstances—first, by their locality, which is most frequently the mitral area; secondly, by their time, diastolic or præ systolic murmurs being always organic; and, thirdly, by their association with pericardial friction. The special characters of inorganic murmurs are described elsewhere. Chronic valvular disease may be diagnosed from acute endocarditis by the presence of cardiac enlargement and many well-known signs; of marked cardiac symptoms—especially pain and dyspnœa; of certain lesions—mitral stenosis, for instance, being very rarely an acute lesion; and of visceral complications. Much more difficult of diagnosis is acute endocarditis occurring in the course of chronic valvular disease. Change of the character of the murmur, if this have been previously known, may lead to the suspicion of fresh inflammation but cannot positively establish the diagnosis of its existence. Acute endocarditis is most frequently overlooked when the primary disease is 'latent'—particularly rheumatism in children.

**PROGNOSIS.**—The immediate prognosis of acute endocarditis is generally favourable, and may be safely estimated by the absence of local symptoms. The remote prognosis, on the other hand, as regards both life and health, is bad, inasmuch as endocarditis so frequently ends in chronic valvular disease. This result is very often difficult or impossible to forecast. A feeble, soft, and smooth murmur, or a feeble and grave murmur, is much more likely to disappear than a loud, extensive, well-defined bruit. The probability of the disappearance of diastolic basic murmurs is very small; it may be best estimated by the absence of the effects produced by aortic incompetence upon the heart and vessels. Murmurs referable to endocarditis disappear more often in young subjects than in adults.

**TREATMENT.**—The treatment of acute endocarditis has to be discussed under three heads—namely, first, preventive; secondly, immediate; and, thirdly, subsequent treatment.

(a) *Preventive treatment.*—When a patient is suffering from any disease which may become complicated with endocarditis, and especially if he be suffering from acute rheumatism, every means must be adopted to prevent, as far as possible, the occurrence of inflammation of the heart. Thus, in acute rheumatism it is all-important to check at once the intensity of the disease by recourse to one or other of the salicyl compounds or other means; for experience shows that endocarditis, when it does occur in acute rheumatism, generally makes its appearance within the first week. Again, the duration of the pri-

mary disease must be curtailed if possible, inasmuch as endocarditis, although it generally appears early, may possibly occur at any period of the disease. Thus the medicinal preventive treatment of endocarditis in these cases resolves itself into the medicinal treatment of acute rheumatism. Another point of equal importance in the prevention of endocarditis is diminution of the cardiac activity. We have seen that the pressure within the heart is an important factor in the causation of endocarditis; and this pressure must be reduced by diminishing the work to be done by the heart, without lowering the cardiac power. Rest must therefore be enforced in the recumbent posture—an end which is usually already secured by the presence of acute rheumatism of the joints. Excitement of every kind has to be strictly interdicted. The personal comfort of the patient must be zealously attended to, and pain relieved, so that restlessness and irritability may be avoided; and for this purpose carefully selected anodynes may be necessary. Stimulants must be cautiously ordered; the bowels regularly and fully moved; and the various secretions kept as active as possible.

(b) *Treatment during an attack.*—When endocarditis has actually made its appearance, the various means just insisted upon must be enforced as rigorously as before, so as to diminish the intensity of the inflammation, and to limit the extent of surface involved. Rest is still of the first importance, mental as well as bodily. The medicinal treatment of the original disease—especially of acute rheumatism—must be persevered in. Local applications to the præcordia, such as cataplasms, or leeching, in cases of sthenic inflammation, are often of great service. The administration of stimulants will require the greatest care; excitement of the heart, on the one hand, being avoided, and, on the other hand, digitalis, ammonia, or alcohol being employed if symptoms of cardiac distress supervene. Equal caution is demanded in the use of anodynes which may be indicated to relieve distress connected with the joints; and local applications, such as cotton-wool, poultices, aconite, and belladonna, should be employed in preference to opium, chloral, and other cardiac depressants.

(c) *Treatment after an attack.*—When the primary disease, such as rheumatism, has subsided, and the restoration of the various functions indicates that convalescence has commenced, the physician must not forget the state of the endocardium which has recently been inflamed, and which is still in a condition of great physical weakness, and the seat of new cell-growth. Instead of urging the patient to sit up and walk about under these circumstances, as must have been frequently done under the 'rival methods' of treating acute rheumatism, we should recommend a very gradual return to

exercise, and the most jealous avoidance of actual exertion. There can be no question that, at this stage, rest for several weeks is of more importance than medicinal treatment. At the same time various tonic and other remedies should be employed.

**2. Ulcerative Endocarditis.**—**SYNON.:** Malignant Endocarditis; Diphtheritic Endocarditis; Fr. *Endocardite Ulcéreuse*; Ger. *Infectiose, Maligne Endocarditis*.

**DEFINITION.**—An acute, subacute, or chronic infective disease, characterised by ulceration of the endocardium or by vegetative growth upon it.

**ÆTIOLOGY.**—Ulcerative endocarditis occurs most frequently between the ages of twenty and forty, but is not uncommon in children. More women suffer than men, in consequence of its frequent origin in puerperal disease. Previous valvular disease distinctly predisposes to it.

An exciting cause sometimes cannot be discovered. In most instances, however, ulcerative endocarditis is but part of a general septic or pyæmic process. The common sources of infection are septic wounds, puerperal thrombosis, pylephlebitis, otitis media with thrombosis of the associated sinuses, acute osteomyelitis, gonorrhœa, injuries and ulceration of the urethra and prostate—in connexion, for instance, with stricture and catheterism; also acute pneumonia and pulmonary gangrene, acute rheumatism, and other acute specific diseases. The primary lesion may be so slight as to escape observation.

**ANATOMICAL CHARACTERS.**—Malignant endocarditis, as the name is commonly employed, comprehends a variety of morbid conditions in connexion with the heart. Two of these are fairly distinctive, the *ulcerative* and the *vegetative*, and they may be conveniently selected for description as being the most diverse types between which there lie a number of intermediate forms.

In the *ulcerative* form, the endocardium presents small areas where the proper tissue has disappeared, leaving behind it shallow ragged erosions. This change occurs most frequently in connexion with the mitral valves and opening, including the auricular aspect of the curtains and the line of attachment of the valves—particularly the two commissures of the segments, and the anterior aspect of the anterior segment; also the aortic cusps; parts of the septum and general endocardial surface; and the papillary muscles and chordæ tendinæ. The endocardium of the right ventricle is relatively more frequently involved in malignant than in simple endocarditis.

The earliest stage of the morbid process may sometimes be observed as small yellowish opacities of the endocardium. But as a rule the surface is found already eroded, presenting a small, irregular, oval, circular or linear ulcer; its ragged grey base covered

with a loose friable thrombus; its edges fringed with vegetations, swollen, indurated, and undermined. Such an ulcer increases but slowly in area, but it readily advances into the depth of the tissue. A variety of secondary changes may then occur—acute valvular aneurysm, complete perforation of the valves, abundant vegetative or fungating growths, and suppuration, which ends in acute cardiac aneurysm or in complete perforation of the parietes.

The *vegetative* form of malignant endocarditis is readily recognised *post mortem* by a prominent and possibly extensive growth of polypoidal vegetations, in connexion usually with the auriculo-ventricular opening, the neighbouring surface of the left auricle, and the auricular aspect of the mitral segments. Less frequently the aortic valves are involved. In either situation the vegetations form a large mass of soft growth, almost choking the mitral or aortic opening, whilst portions of it must have floated and waved in the blood-current, and by their contact or impact during the cardiac movements have injured the neighbouring or opposed surfaces of the endocardium, there setting up fresh disease of the same kind. The surfaces of these vegetations are sometimes calcified.

In both forms of malignant endocarditis it is obvious that during life portions of the diseased endocardium and thrombi, as well as their fluid products, whether on a small or on a large scale, must have been readily detached or washed off, and carried as emboli into other parts of the circulation.

In addition to the lesions just described, the heart may present a variety of morbid changes. It is in at least 75 per cent. of cases (Sansom) the seat of chronic valvular disease and associated enlargement; sometimes of congenital malformation. The myocardium may be swollen and in a state of granular degeneration like other visceral structures; or it presents a number of small embolic, hæmorrhagic, inflamed, or fatty degenerated areas. Pericarditis is sometimes associated.

*Embolism* is one of the essential anatomical characters of ulcerative endocarditis. It occurs most often in the spleen, kidneys, and heart, but may be almost universal. In the vegetative form of the disease the emboli may be so large as to be arrested even in the abdominal aorta; the tibial, brachial, and other vessels of the limbs are often occluded, as well as the splenic, renal, mesenteric, cerebral, and a variety of arteries in other parts of the body. In the purely ulcerative type of the disease the emboli are much smaller, and their effects are only recognised *post mortem* by infarcts in various conditions of change. Sometimes these secondary foci proceed to abscess—suppurative meningitis is fairly common. The skin and mucous

membranes, the serous membranes, the vessel walls, and the retina are also favourite seats of minute embolism and hæmorrhage. The lungs are specially affected in some cases: pulmonary embolism and infarction accompany ulcerative endocarditis of the right chambers; in other instances acute pneumonia appears to be the primary lesion, in which the endocarditis of the left chambers of the heart has originated. The local effects of infective embolism occur very extensively in ulcerative endocarditis, namely, hæmorrhage, softening, and acute aneurysm ending in fatal rupture. See EMBOLISM.

In a considerable proportion of cases of ulcerative endocarditis micro-organisms are found both in the valvular lesion and in the emboli and infarcts. No less than ten different kinds of organisms have been described in this connexion, the chief of these being *Staphylococcus pyogenes*, *aureus* and *albus*; *Streptococcus pyogenes*; *Diplococcus pneumoniae*; and *Bacillus tuberculosis*.

Micrococci have also been found in the circulating blood. See MICRO-ORGANISMS.

**SYMPTOMS.**—In its clinical characters malignant endocarditis is either (a) an *acute*, severe, and fatal malady with markedly typhoid features; or (b) a disease of *subacute*, possibly *chronic*, course, pyæmic in character, occasionally ending in recovery. These two forms call for separate description.

(a) **Acute Typhoid Malignant Endocarditis.**—In this variety of the disease, the patient is seized with a rigor when apparently in good health, or during convalescence from an acute illness—particularly rheumatism; in other instances the invasion is more gradual. Pyrexia and the other phenomena of severe fever are quickly developed, and the patient complains of malaise, debility, and cardiac distress; but for the first two days the medical attendant probably fails to make a complete diagnosis. By the end of this time, the patient's condition having become steadily worse, the cardiac sounds are found to be impure; they shortly become murmurish; and probably on the next day an endocardial murmur is developed, systolic or diastolic, still changeful in its characters. The general symptoms now begin to assume a typhoid type. The patient lies helplessly on his back; the temperature rises to 104°; the tongue, at first furred, becomes dry and brown; sordes form; the respiration becomes hurried, possibly with thoracic pains and hæmorrhagic sputa; and somnolence sets in, broken by low muttering delirium, jactitations and subsultus. The spleen is enlarged and tender, suggesting infarction. The bowels may be relaxed. The skin is visited by heavy sweats, and may become covered with a peculiar petechial eruption. The urine, passed naturally or incontinently, or withdrawn with the catheter, is found to contain blood, albu-

men, and various micro-organisms. The ophthalmoscope reveals retinal and other intraocular hæmorrhages. Bleeding takes place from the bowel. Less frequent symptoms are parotitis, arthritis, pericarditis, and jaundice. Meningitis, associated with headache and delirium, and ending in coma, is more common, and gives a new or different aspect to the case.

Malignant endocarditis of this severe type may be said to always terminate in death after a duration of less than fourteen days; it has been known to end fatally in two days. The course which the disease pursues is one of steadily increasing gravity, broken only by equally ominous complications in the form of meningitis, hæmorrhages, or sudden paralysis referable to cerebral hæmorrhage. Very rarely the disease passes into the subacute or chronic form.

The pyrexia belongs to the continued type, the maximum rise being 104° or 105° F., with a daily remission of about one degree. In some instances it may not exceed 101°, or even keep close to normal. With the fever may be associated repeated rigors, followed by increased prostration and stupor. The physical signs connected with the heart have already been referred to. The endocardial murmur is usually systolic and mitral. It develops under observation, changes in loudness and quality day by day, and may be reinforced by a second murmur over another part of the præcordia. If traced to the right side of the heart, it is of special significance. Occasionally the murmur is absent, suggesting a parietal lesion. The radial pulse presents all the characters of the typhoid state with increasing prostration, rather than those of any particular form of valvular disease. The so-called skin 'eruption' consists of fine reddish spots, with paler centre representing the seat of a minute embolus. The signs of pleurisy, pulmonary infarction or gangrene, or even of pyo-pneumothorax, are sometimes to be determined.

(b) **Subacute and Chronic Malignant Endocarditis.**—This variety of the disease bears but little superficial resemblance in its clinical characters to the severe form just described. Its general features are those of subacute or chronic cardiac disease accompanied with pyrexia and multiple embolism. Heart-disease is the central fact, on which are superadded the phenomena of general infection. There is no prostration until the last; no typhoid aspect. The patient is anæmic, sallow, poorly, and he wastes steadily; but he sits up in bed, reads, engages in conversation intelligently and cheerfully, and may even resent strict treatment. The physical signs connected with the heart consist mainly of a well-developed endocardial murmur, possibly referable to a previous valvular lesion, but changing its characters from time to time under observation.

It is usually systolic and mitral; occasionally aortic systolic or diastolic, or mitral presystolic; and it may be suddenly altered or complicated by the development of a fresh lesion in the heart, such as rupture or perforation. The pulse varies greatly.

The type of pyrexia in fully developed cases of malignant endocarditis of subacute course is markedly remittent, or even intermittent. Three, four, or even six degrees F. of a rise and fall (between 98° and 102° to 104°) may be recorded daily for weeks on end. In other instances the disturbance of temperature takes the form of irregular bouts of pyrexia complicating the downward course of a severe case of valvular disease. Some patients suffer from repeated rigors, unexpected and irregular, followed by profuse sweats and prostration.

The other phenomena of general infection are mainly significant of embolism: pain and the proper physical signs of infarction of the spleen and lungs, blood in the urine, loss of pulsation in the arteries of the limbs, the development of acute aneurysm with subsequent rupture, and even gangrene of the extremities. This element of the disease gives it a peculiarly uncertain and protean character, its course being, for instance, suddenly interrupted by the development of severe hæmorrhage into the peritoneal cavity, or possibly into the brain, with paralysis and coma ending in rapid death.

The duration of this disease is almost indefinite. It extends from six or eight to twelve or sixteen weeks; in one case of Dr. T. Henry Green's it lasted eighteen months. The progress is very striking. The patient steadily but almost imperceptibly loses strength, flesh, and colour. With very few exceptions, the disease terminates in death, and such patients as have recovered were left with permanent heart-disease.

**PATHOLOGY.**—In the less severe type of ulcerative endocarditis (as in the more familiar ulcers of the integuments) portions of the ill-nourished, degenerated, or lowly organised fibroid tissue of chronic valvular lesions break down in serious impairment of the general health, in acute disease, and in other unfavourable constitutional states (*see* ULCER AND ULCERATION). Subjected to constant strain and impact, the ulcerous surfaces extend rather than heal, establish on and around them an increasing growth of vegetations, and these become the source of multiple embolisms. In cases of this kind no morbid micro-organisms are present; the disease runs a comparatively slow course; and recovery may occur. In the acute typhoidal type of the disease it appears that true contagia, derived from some obvious source (the 'primary lesion'), or entering the blood through some unknown channel ('idiopathic'), settle on the endocardium whether previously diseased or injured—possibly even

when intact. The organisms flourish on this soil, shed their products into the circulation, and plant embolic colonies of their own kind as multiple foci of disease throughout the body. This process is necessarily associated with a septicæmia of a more or less severe kind, according to the nature of the organisms and emboli. Some authorities deny that the organisms are the essential cause of the morbid process, and it must be acknowledged that the full and complete proof of this connexion is not yet established.

**DIAGNOSIS.**—Now that malignant endocarditis has been so carefully investigated, its recognition ought not to be attended with much difficulty. The acute form has to be diagnosed from typhoid fever, acute tuberculosis, and other infective diseases belonging to its own type. The diagnosis usually turns on its association with some primary disease, and on the result of a physical examination of the heart and the correct interpretation of any murmur that may be discovered. When no murmur is developed, ulcerative endocarditis may escape recognition. Repeated rigors, the type of pyrexia, the character of the spots, and the occurrence of retinal hæmorrhages will further serve to exclude typhoid fever and tuberculosis. When delirium and other cerebral symptoms are urgent, ulcerative endocarditis has been mistaken for 'cerebral rheumatism' and simple meningitis.

The subacute and chronic forms are especially liable to be confounded with a number of diseases which present a similar type of pyrexia, particularly the many pathological conditions which underlie obscure persistent pyrexia, such as latent tuberculosis; lymphoma; chronic suppuration in connexion with the liver, prostate, bowel, kidney, internal ear, lymphatic glands, and connective tissues; and also syphilis. The existence and changeable character of the murmur, the sudden embolisms, and the absence of other local signs, all indicate the cardiac seat of the disease. From malaria it can be distinguished by the history, by the absence of periodicity in the rigors and fever, as well as by the cardiac murmur.

Some of the gravest complications of this form may, for a moment, be misleading, such as violent headache ending in coma, referable to the development and rupture of embolic intracranial aneurysm. Experience teaches us to be prepared for every possible variety of symptom in a disease of which a striking feature is visceral embolism.

**PROGNOSIS.**—The prognosis of acute malignant endocarditis is practically hopeless. In the subacute form the prospect is but little better. However confident and cheerful the patient may be at first, the disease will surely, if slowly, exhaust the strength, and may at any period rapidly prove fatal by some complication. If the temperature fall and become

less remittent, if the rigors cease, and particularly if weight and strength increase, hope may begin to rise that the case will prove one of the very few exceptions and ultimately recover. But the practitioner must not forget the precarious nature of such improvement, and the incompleteness of the recovery at the best.

**TREATMENT.**—The failure of every means of cure hitherto employed in acute malignant endocarditis serves to impress on us the importance of preventive treatment. Dr. Sansom urges the necessity of protecting the subjects of chronic valvular lesions against zymotic diseases, and also of attending in them with special care to the treatment of suppuration. When ulceration has commenced, the case must be managed on the general principles applicable to all acute specific fevers, the best possible nursing, abundance of food, and a free supply of stimulants being provided. Fresh air is essential; and if the disease be traced to some error of sanitation, it may be advisable to take the risk involved in changing the room, or even the house, in which the patient is lying.

A great variety of drugs have been tried in both forms of this disease, unhappily with little benefit, including mercurials, quinine and other antipyretics, and the essential oils of eucalyptus, turpentine, and their allies. Two methods are reported to have been attended with success in a few instances of the chronic form: the sulphocarbolates of sodium introduced by Dr. Sansom in 30-grain doses four times a day, and quinine in 15-grain doses three times a day with a free allowance of alcohol (in the form most agreeable to the patient), which is recommended by Professor Fraentzel of Berlin.

**II. Myocarditis.**—**SYNON.**: Carditis; Fr. *Myocardite*; Ger. *Myocarditis*.

**DEFINITION.**—Inflammation of the walls of the heart.

This disease may be either acute or chronic; but the latter form, which is attended with the formation of fibroid tissue in the myocardium, is described under the head of HEART, Fibroid Disease of. Pyæmic inflammation of the substance of the heart also constitutes so special a form of disease that it is treated separately (*see* HEART, Pyæmic Abscess of). Acute myocarditis alone, therefore, has to be considered in the present article.

**ÆTIOLOGY.**—A certain amount of myocarditis is sometimes associated with acute endocarditis and pericarditis, and depends upon the same causes; the most frequent being acute rheumatism. In a small proportion of cases, rheumatic myocarditis appears to occur independently of inflammation of the lining or of the covering membrane. In the great majority of recorded cases of localised myocarditis ending in abscess, the cause of

the disease was altogether obscure. It has been observed most frequently in males, and before the twenty-fifth year of life. Exposure to cold, severe exertion, and local injury are mentioned amongst exciting causes, but with questionable correctness.

**ANATOMICAL CHARACTERS.**—Acute inflammation of the myocardium generally involves the connective tissue as well as the muscular fibres; but in a few instances the latter alone have been found affected, constituting so-called *parenchymatous myocarditis*.

The ordinary form of the disease is characterised by the appearance of leucocytes between the muscular fibres of the heart. In one class of cases, the inflammation is moderate in intensity but *diffused* in extent, affecting one or more layers of muscle underlying the endocardium or the pericardium, which is also inflamed; in another class of cases, the inflammation is more active, and proceeds to the formation of abscess, whilst it is, as a rule, comparatively *localised*.

In the first or diffused form, the myocardium, as it is seen through its inflamed covering, appears of a mottled opaque buffy colour, and is somewhat swollen and softened. The microscopical characters consist chiefly in the appearance of leucocytes and inflammatory effusion in the intermuscular connective tissue; swelling, opacity, nuclear proliferation, and rupture of the muscular fibres, followed by fatty degeneration and atrophy of the same; and the ordinary inflammatory changes of the vessels of the part. Beyond this stage, unless the case prove fatal, the diffused form of myocarditis passes into a chronic condition; and it ends either in fibroid disease with a moderate amount of atrophy, by development of the inflammatory products and compression of the affected fibres; in fatty degeneration; in calcification; or in cardiac aneurysm.

Suppuration of the heart, on the other hand, generally takes the form of swollen yellowish-white patches or abscesses, surrounded by dirty-red or ecchymosed tissue, boggy or pulpy to the finger, and containing on section a small quantity of variously coloured puriform matter, consisting of pus and muscular débris. In the same cases a great part of the walls of the heart may be in a condition of parenchymatous degeneration; and in some recorded instances the whole of the cardiac tissue is described as infiltrated with pus. Abscesses resulting from acute localised myocarditis are generally very small, varying from the size of a pea to that of a nut. They may either burst externally into the pericardial sac, or internally into one of the cavities or through one of the valves, leading to pyæmia, and to the formation of an acute cardiac aneurysm; or the pus may make its way both externally and internally, and lead to fatal hæmorrhage into

the pericardium. In other cases the pus undergoes the usual changes, and becomes inspissated or cheesy; or calcification takes place.

In both forms of interstitial myocarditis the left ventricle is most frequently the seat of inflammation.

**SYMPTOMS.**—The principal symptoms of acute rheumatic myocarditis are restlessness and urgent dyspnoea; severe pain and distress referred to the præcordia; and palpitation, which gradually passes into irregularity and greatly increased frequency, and finally into complete cardiac failure. The pulse corresponds. The countenance is anxious, and either pale or cyanosed. The mind is fearful and distressed at first; and delirium frequently supervenes before death, especially in young subjects. Vomiting is not uncommon.

The *physical signs* are generally associated with those of endo- and peri-carditis. When uncomplicated, they may be described as consisting of violent cardiac impulse at first, which rapidly loses in strength and regularity while it increases in frequency; a somewhat increased area of cardiac dullness; and short sharp sounds, afterwards becoming duller and more feeble.

When these symptoms and signs make their appearance, they generally run their course rapidly, and end in death. In a small number of cases they as rapidly disappear.

The symptoms of localised suppurative myocarditis leading to abscess are not unlike those just recorded. There are the same distressing symptoms locally, with restlessness and anxiety, passing on to delirium and ending in collapse. Rigors have been observed in some cases; and a peculiar pustular eruption on the skin in other cases. The physical signs also are not special; excepting that a murmur may be suddenly developed by rupture or perforation of part of the wall or of a valve.

The majority of cases of abscess of the heart prove fatal by asthenia; but the other terminations of abscess mentioned above will be attended by their respective symptoms, and the possibility of sudden death is especially to be noted.

Diffuse *parenchymatous* myocarditis is clinically known only as a cause of sudden death.

**COMPLICATIONS.**—The complications of acute myocarditis have already been sufficiently indicated, such as, first, ætiologically, pericarditis, endocarditis, acute rheumatism, and other causes of these forms of inflammation; and secondly, pathologically, rupture of the cardiac walls or valves, acute cardiac aneurysm, hæmato-pericardium, embolism, and septicæmia.

**COURSE AND TERMINATIONS.**—The course of acute interstitial myocarditis, as already stated, is generally rapid, extending from a

few hours to eight days in different cases. Death occurs, in the great majority of cases, from the effects of cardiac failure, if the inflammation be extensive or proceed to suppuration. The formation of acute aneurysm by internal rupture, the production of pericarditis by external rupture, and other complications will variously modify the progress and termination of cardiac abscess. Simultaneous rupture both externally and internally causes sudden death.

**DIAGNOSIS.**—The diagnosis of acute myocarditis is extremely difficult. Occurring in connexion with acute rheumatism, it has to be distinguished from endo- and peri-carditis. The absence of murmur and of the characteristic signs of pericarditis, along with symptoms of cardiac failure and severe local phenomena, such as pain, distress, dyspnoea, and finally collapse, should generally serve to establish the diagnosis of inflammation of the walls of the heart. It cannot be said that cardiac abscess has ever yet been diagnosed; but the careful consideration of all the points in the case, including the sudden development of a murmur indicative of rupture of a portion of the wall, or of a valve, may hereafter ensure greater success. In the event of the development of the last-named sign, and of septicæmia or embolism, cardiac suppuration would have to be carefully diagnosed from ulcerative endocarditis. In children, the delirium of acute myocarditis has to be distinguished from acute meningeal inflammation, an object which may be effected by the careful observation of the signs and symptoms connected with the heart.

**PROGNOSIS.**—The prognosis of myocarditis, when it is either so extensive or so intense as to give rise to unequivocal symptoms, is extremely unfavourable.

**TREATMENT.**—The two principal indications of treatment in acute inflammation of the substance of the heart are to rest and sustain that organ, and to relieve the pain and distress. Local anodynes, especially in the form of the preparations of belladonna and poultices; and stimulating 'counter-irritants,' such as mustard cataplasms, will conduce to fulfil the second indication. Such relief is the first essential, if rest is to be secured. The patient must be spared the very smallest exertion. Food must be given in small quantities, and be easily digestible and highly nutritious; the bowels must be kept open; and the flow of urine should be as free as possible. Alcoholic stimulants will be urgently called for, but must be employed with great discrimination; and palpitation, as much as weakness of the impulse, is to be regarded as an indication of the necessity for these. At the same time, digitalis, ammonia, and other cardiac stimulants should be given cautiously, so as to sustain the cardiac action.

J. MITCHELL BRUCE

**HEART, Malformations of.**—SYNON.: Fr. *Affections Congénitales du Cœur*; Ger. *Missbildungen des Herzens*.

CLASSIFICATION AND DESCRIPTION.—The cardiac anomalies of development may be classed as follows:—

I. Those dependent on arrest of the process of development at an *early period of fetal life*, so that the organ retains its most rudimentary form, the auricular and ventricular cavities being still single or presenting only slight indications of division, and the primitive arterial trunk being retained, or the aorta and pulmonary artery being very imperfectly evolved.

II. Those in which the defective conformation occurs at a *more advanced period*, when the auricular and ventricular partitions are already partly formed, and the aorta and pulmonary artery more or less completely developed. Such are the cases in which, with imperfect separation of the ventricles and auricles, the arterial or auriculo-ventricular passages are constricted or obliterated, and the origins of the aorta and pulmonary artery are misplaced.

III. Cases in which the development of the organ has progressed regularly till the *later periods of fetal life*, so that the auricular and ventricular septa are complete, and the primary vessels have their natural connexions, but in which there are defects which prevent the heart undergoing the changes which should ensue after birth. Such are the premature closure of the foramen ovale, the non-development of the ductus arteriosus, and the occurrence of slighter sources of obstruction at the arterial or auriculo-ventricular passages or in the course of the aorta.

IV. Cases in which there is some irregularity in the formation of the valves, or in the connexions with the vessels, or in the vessels themselves, which, though not immediate sources of obstruction, *may become so during the progress of life*, so as to lay the foundations of subsequent disease.

I. Cases of the first class are of very infrequent occurrence, and are the more rare according to the extent of the imperfection. The first case of simply biloculate heart was placed on record by Mr. Wilson, in the *Philosophical Transactions*, in 1788, and the specimen is preserved in Dr. Baillie's museum, in the possession of the Royal College of Physicians. The anomaly was found in the body of a child, which survived for seven days. From defect in the diaphragm, the heart lay in a sac on the upper surface of the liver; and the organ was found to consist of an undivided auricle and ventricle, and a single artery, evidently the primitive arterial trunk, which first gave off a vessel that furnished the branches to the lungs, and the vessels to the head and upper extremities. The coronary arteries arose by a common

trunk from the aorta before its final division. Since the publication of this case, others have been placed on record, illustrating the gradual advancement from the simple to the more complicated forms—the ventricle becoming more completely divided, the septum of the ventricles being more fully developed, and there being two vessels given off from the ventricle, though in some cases one of these may be abortive, or if there be only a single vessel, that being shown by the origin of the coronary arteries from its commencement to be really the aorta.

II. Of the second class, the examples which have been described are much more numerous. In cases of this kind the auricles and ventricles are fully formed, though the septa which divide them are incomplete, and there is usually more or less displacement of the origins of the primary vessels, so that the aorta more especially may come to arise partly or almost entirely from the right ventricle; or the points of origin of the vessels may be transposed, the aorta arising from the right ventricle, and the pulmonary artery from the left. Cases of the former description, in which the septum of the ventricles is incomplete, and the aorta misplaced to the right, are almost always found to coexist with some obstruction to the passage of the blood from the right ventricle, either (1) from smallness of the pulmonary artery; (2) from imperfection of the valves; (3) from constriction at the outlet of the right ventricle, or at the end of the conus arteriosus or infundibular portion of the ventricle; or (4) from constriction at the commencement of the conus or point of union between that portion of the ventricle and the sinus. A case of the second description was published by Sandifort, in 1777, and one occurred to Dr. Hunter in 1761, but was not published till 1763. The fourth form of obstruction, or that occasioned by constriction between the sinus and the infundibular portion of the right ventricle, has only recently been explained, though cases of the kind have for some years been placed on record. It is indeed probable that the existence of a very decided partition in this situation led to the idea entertained by some of the older pathologists, that the heart occasionally had three ventricular cavities. The abnormal septum is partly formed by hypertrophy of the muscular structure, and partly by the endocardium becoming thickened; and in some cases very decided obstruction is so caused. The defect is generally developed at an early period of fetal life. The septum of the ventricles is therefore incomplete, the defect being at the posterior part, so that the aorta comes to arise from the sinus of the right ventricle, while the pulmonary artery takes its origin from the infundibular portion, which seems to constitute a distinct cavity. The heart thus, as pointed out by Mr. Grainger, bears an

almost exact resemblance to the condition of the organ in the turtle. In the turtle there are two aortic ventricles and one pulmonic ventricle; the right aortic and the pulmonic ventricle being the analogues of the sinus and infundibular portion of the right ventricle, and being in connexion, while the left aortic ventricle is distinct.

Much more rarely there has been found an entire obliteration of the orifice or trunk of the pulmonary artery, the first case of this description of anomaly having also been published by Dr. Hunter at the same time as the former case. Much more rarely, also, the defect in the ventricle has been found in connexion with obstruction or obliteration of the right auriculo-ventricular, the left auriculo-ventricular, or the aortic aperture. Where the septum of the ventricles is incomplete, the defect generally exists at the base, at the part which has been termed the *undefended space*—the space which intervenes between the contiguous sides of the left and posterior semilunar segments, where on the left side the muscle is naturally deficient; and in this way a connexion may exist between the left ventricle and right auricle or ventricle, either immediately above or below the right auriculo-ventricular opening. More rarely the septum between the left ventricle and the conus arteriosus of the right ventricle is defective; and still more rarely an aperture exists at a lower part of the septum. The portion of the septum dividing the left ventricle from the sinus of the right is termed by Rokitsansky the posterior—that between the left ventricle and conus arteriosus, the anterior septum. With the defects now mentioned, the auricular septum may also be incomplete, or the foramen ovale may be still open, or the ductus arteriosus pervious. Indeed, when the pulmonary artery is much contracted or impervious, one or other of the former conditions necessarily exists, and the ductus arteriosus becomes the means by which the blood is conveyed to the lungs, though occasionally there are compensatory branches derived from the aorta or one of the large vessels also distributed to the lungs.

The transposition of the aorta and pulmonary artery also occurs at an early period of fetal life. The first case of the kind recorded was related by Dr. Baillie in 1797: the specimen is figured in his plates, and still exists in the museum in the possession of the Royal College of Physicians. In this anomaly the septum of the ventricles is generally defective, and the two fetal passages open, and the organ may indeed be very defective in conformation. The heart also is often misplaced in the chest. Another form of defect is that in which the descending aorta is given off from the pulmonary artery through the ductus arteriosus. This condition is apparently the result of imperfect de-

velopment of the isthmus aortæ between the origin of the left subclavian artery and the point of entrance of the duct, so that an adequate supply of blood cannot be conveyed from the ascending into the descending aorta. Generally the condition coexists with defect of the septum of the ventricles, as in two cases formerly in the possession of Sir Astley Cooper, and now contained in the museum of St. Thomas's Hospital, described by Dr. Farre in 1814. In some instances of this kind the portion of aorta between the left subclavian artery and the duct is imperforate, and yet in others, as in a case related by Steidelle and referred to by Hein in 1816, there is no connexion between the two portions of the aorta, the ascending part giving off the vessels to the head and upper extremities, the descending portion being wholly derived from the pulmonary artery. This form of defect is closely allied to the cases which are occasionally seen in after-life, in which there is constriction or obliteration of the isthmus aortæ beyond the left subclavian artery, the circulation being maintained through collateral channels.

III. The third class of cases consists in the premature closure of the foramen ovale; or the non-development or disappearance, at an early period of fetal life, of the ductus arteriosus; or in diseased conditions of the valves, which prevent the heart undergoing the changes which should ensue after birth.

The first condition is of very rare occurrence. The first instance recorded was related by Vieussens in 1715. In these cases, the blood during fetal life being all transmitted through the right cavities and the pulmonary artery and duct, those portions of the heart are unduly developed, while the left side of the organ becomes atrophied. In the second class of cases the heart is defectively developed, and the right ventricle gives origin to the aorta, and often also vessels are distributed from the aorta to the lungs, while the ordinary pulmonary artery may be very small in size, or may be entirely absent. In a case of this kind, described by Dr. Ramsbotham, the pulmonary artery is said not to exist; but by examination of the specimen preserved in the London Hospital Museum, the writer has ascertained that this is not correct. The artery exists as a very small vessel, but the scanty supply of blood to the lungs which it furnished was complemented by small vessels from the aorta. In cases which are not of uncommon occurrence, and may be classified with the malformations now spoken of, but which are closely allied to the next class, there exists some source of obstruction to the transmission of the blood from or into the right ventricle, which determines the imperfect closure of the foramen ovale, or the patency of the ductus arteriosus. The obstruction in these cases generally depends on disease of the pulmonic valves, or obstruction

at the end or beginning of the conus arteriosus, or at the right auriculo-ventricular aperture.

IV. The fourth class of malformations consists of defects, of a slighter description, of the valves, or narrowing of the orifices or of the isthmus aortæ.

The semilunar valves may be excessive or defective in number. The former condition probably does not materially interfere with the functions of the heart—the latter often does so, and especially when, as is very frequently the case, the valves become the seat of disease in after-life. If there be only two valves, one of them imperfectly representing two distinct segments, there is great liability to incompetence. If there be only one valve, representing three imperfect segments, obstruction is almost necessarily occasioned.

It is probable that the cases in which the tricuspid valve is found represented by a kind of membranous diaphragm, stretched across the orifice and perforated in the centre, and some of the so-called cases of button-hole mitral, are also of congenital origin.

It is not considered necessary in this article to refer to many examples of these different forms of malformation, or to allude to other of the less important deviations from the natural conformation of the heart. The subject will be found more fully treated of in the works of Dr. Farre,<sup>1</sup> of Gintrac,<sup>2</sup> of Freidberg,<sup>3</sup> in the papers of Dr. Chevers,<sup>4</sup> and in the writer's own work.<sup>5</sup> The more recently published cases also are abstracted in the treatise of Taruffi,<sup>6</sup> and numerous examples of different forms of malformation are given by Rokitsansky.<sup>7</sup>

MODE OF FORMATION.—It is probable that all the different forms of irregularity in the development of the heart are due to arrest or development, occurring at different periods of evolution, so that the heart retains the forms proper to it at such stages. The cause to which this arrest is to be ascribed can rarely, however, be traced in cases where the defect is great, such as those of biloculate heart, or where the vessels are transposed with or without very marked imperfection in the organ itself. These defects must be ascribed to the imperfect evolution of the

double set of cavities, and of the pulmonary artery and aorta from the primitive trunk and branchial arches. In the less marked defects, however, the irregularity can often be traced to a source of obstruction to the transmission of the blood through one or other of the apertures or vessels. Such obstruction is much the most common in connexion with the right ventricle and pulmonary artery. In cases of this kind the septum of the ventricles is deficient, so that the aorta arises wholly or in part from the right ventricle. Dr. Hunter, in the paper before referred to, when describing a case of obstruction of the pulmonic orifice with defect in the septum of the ventricles, suggested that the imperfection in the septum was probably caused by the pulmonic obstruction. Meckel, however, adopted the view that the primary defect was in the septum of the ventricles, and that the pulmonary artery became more or less abortive from being thrown out of the course of the circulation by the ready outlet afforded for the blood from the right ventricle into the aorta. The former view seems, however, to afford the more satisfactory explanation, and is in accordance with the almost constant occurrence of disease of the valves in these cases. According to the view of Meckel, the pulmonary artery should simply be small, as when the ductus arteriosus is absent, like the case of Dr. Ramsbotham's before referred to, but such a condition is very rarely found. The excess in the number of the semilunar valves might seem to afford an example of redundant development, but this condition also probably depends on arrest of development; though, as we do not clearly understand the mode in which these valves are developed, it is impossible to express a very decided opinion as to the cause of the apparent excess.

SYMPTOMS AND DIAGNOSIS.—There cannot generally be much difficulty in recognising a case of malformation of the heart during life. Not only in cases of a very marked kind is there generally a complete history of the condition of the subject during its short life, but the symptoms are also very characteristic. The child is very markedly cyanotic; the cheeks, lips, hands, and feet are excessively livid; the fingers and toes are clubbed; the nails are incurved; and the patient is liable on any excitement, or on exposure to cold, to attacks of dyspnoea, often followed by convulsions. There are also often present difficulty of breathing, cough, and expectoration of blood; with cardiac palpitation, and pulsation of the vessels of the neck. If also there be any obstruction at or near the pulmonary orifice, there will be a harsh systolic murmur heard in the course of the pulmonary artery; and if there be a defect in the septum of the ventricles, the murmur will be heard probably also in the course of the aorta. Often there are unhealthy ulcerations about

<sup>1</sup> *On Malformations of the Human Heart.* London, 1814.

<sup>2</sup> *Observations et Recherches sur la Cyanose, ou Maladie Bleue.* Paris, 1824.

<sup>3</sup> *Die angeborenen Krankheiten des Herzens, etc.* Leipzig, 1844.

<sup>4</sup> *Collection of facts illustrative of Morbid Conditions of the Pulmonary Artery.* London, 1851, and *Medical Gazette*, 1845 to 1851.

<sup>5</sup> *Malformations of the Human Heart.* Second edition, 1866.

<sup>6</sup> *Sulle Malattie congenite, etc., del Cuore.* Bologna, 1875.

<sup>7</sup> *Die Defecte der Scheidewände des Herzens.* Wien, 1875.

the fingers, toes, and anus or vulva. If the case do not attract notice till later in life, there will probably be less marked signs of obstruction to the circulation, and possibly they may be entirely absent, and there may be no history of the patient's previous condition. In cases of this kind the probability will be that if there is a murmur at the pulmonary artery there is some defect at or near the orifice of that vessel, with or without an aperture in the septum of the ventricles or an open state of the ductus arteriosus. The former condition is so rare as the result of disease in after-life, that if the signs point to pulmonic valvular disease, its congenital origin may safely be surmised. The open foramen ovale and ductus arteriosus could probably not be diagnosed with any certainty, though cases have occurred in which peculiar murmurs noticed during life were believed to be so produced. It might be supposed that when so small a proportion of the blood is subjected to the influence of the air as in some of these cases, the temperature of the patient would not reach the natural standard; but the most careful observation of the temperature of children labouring under congenital cardiac cyanosis has generally failed to detect any marked difference in this respect between them and other children of about the same age.

**Cyanosis.**—There are few subjects which have excited more discussion than the causes of cyanosis or *morbis cæruleus*. Morgagni, in 1761, when describing the case of a girl who had obstruction at the orifice of the pulmonary artery with an unclosed foramen ovale, expressed the opinion that the general congestion was probably the cause of the remarkable lividity which had been noticed during life; and Dr. Hunter, in 1783, in describing a case of pulmonic obstruction with imperfection in the septum of the ventricles, ascribed the lividity to the intermixture of the venous and arterial currents of blood. These two views have since received support from various writers. The view of Morgagni has been maintained by Louis, and that of Hunter by Gintrac. It has been very fully shown that there is no just and constant relation between the intensity of the cyanosis and the amount of intermixture, and indeed that very marked cyanosis may exist without any intermixture; while on the other hand in all cases of marked cyanosis there are present causes capable of producing great venous congestion. The writer is, therefore, of opinion that the evidence is very greatly in favour of the correctness of the views of Morgagni and Louis, that the cyanosis results from stasis of the blood, though probably other causes conduce to the intensity and peculiarity of discoloration. Thus, probably, the defect must be congenital, or, at least, of very long duration, so that the smaller vessels may become greatly dilated; the integu-

ments must be thin, so as to allow the colour of the blood more readily to be seen; and lastly—probably also from the very small portion of the blood which can be subjected to the influence of the air—the whole mass of the blood is of an unusually dark colour, and so the intensity of the lividity is increased.

**DURATION AND TERMINATIONS.**—The duration of life in the subjects of the different forms of malformation varies greatly, according to the degree of the defect in the heart. In cases in which the organ presents a very rudimentary condition, life can only be prolonged for a few hours or days; while in the slighter forms of defect the patient may survive to puberty or to manhood or womanhood, or even to more advanced age. Thus, in cases of contraction of the pulmonary artery, without other defect in the organ, cases are on record in which patients lived to 44 and 63; when, with the pulmonic disease, the foramen ovale was unclosed, the subjects have reached 40 and 57. Where the septum of the ventricles was deficient, a few patients are stated to have lived to between 20 and 30. Where the ductus arteriosus was still open, patients lived to 13½ and 19 years; but of course these ages are the extremes, and by far the largest proportion of the subjects die much younger. When the pulmonic orifice or artery is impervious, but few patients survive for more than two years, but cases are on record in which 9 and 12 years of age were attained; the age being greater according to the facility afforded for the transmission of the blood from the right side of the heart, as when the septum of the ventricles was imperfect, than when the ventricles were completely separated by a fully developed septum.

Transposition of the aorta and pulmonary artery is a defect incompatible in any of its forms with the prolongation of life for any lengthened period. Four cases are, however, on record in which the patient survived to between 2 and 3 years of age—the imperfection of the septum of the ventricles tending in these cases also to the prolongation of life.

The most common causes of death in cases of malformation of the heart are affections of the brain and lungs, hæmoptysis, &c.; and, if the patient survive for a sufficient period, tuberculous affections. Notwithstanding the very great obstruction to the circulation, dropsical symptoms do not generally arise to any marked degree.

**TREATMENT.**—It is scarcely necessary to speak of the treatment of these cases. It must consist in protection against cold; in the maintenance of bodily and mental quiet; and in the use of a nutritious and easily digestible diet. Their special liability to tuberculosis must not be forgotten.

T. B. PEACOCK.

**HEART, Morbid Growths in.**—The various forms of morbid growth that have been met with in the heart may be thus enumerated in the order of their frequency: (1) Malignant disease; (2) Lymphomatous or lymphadenomatous growths; (3) Non-malignant tumours; and (4) Cysts. Fibroid growths, syphilitic gummata, and tubercle, as well as hydatids affecting the heart, are discussed separately under their respective heads. Calcareous, cartilaginous, and osseous changes of the myocardium are noticed in the article HEART, Degenerations of.

**1. Malignant Disease of the Heart.**—Cancer, although the most common of the new formations found in the heart, is still very rare in this situation, and is a subject chiefly of pathological interest.

**ETIOLOGY.**—Malignant disease of the heart is, with very few exceptions, always secondary; and the primary growth may have its seat in any part whatever of the body. Occasionally the heart becomes involved by continuity, the lungs and mediastinum being the seat of the primary disease. Cases have occurred at all periods of life, from infancy to old age; but at least one-half of the subjects have been in the middle period of life. The disease has been most frequently found in males.

**ANATOMICAL CHARACTERS.**—Carcinoma, epithelioma, and sarcoma, including colloid cancer and melanosis, have all been found in the heart in different instances. Any part of the organ may be affected, and the right side appears to be more frequently invaded than the left; but the disease is generally multiple. The morbid growth usually presents itself upon either of the surfaces of the heart, rather than in the substance of the myocardium. In these situations there may appear one or more masses of malignant disease, which as a rule are easily distinguished from the cardiac tissue around, and which possess the ordinary characters of such formations, according to their respective kinds, encephaloid being the most common, and epithelioma by far the most rare. Any difficulty in the recognition of the disease is removed by section and microscopic examination. The extent of cardiac wall involved by the growth is sometimes great. When the masses of malignant disease project externally, they are frequently associated with pericarditis, either local or general. Prominent nodules in the interior of the heart may cause local endocarditis; and in other instances the valves and their appendages may be so involved that incompetence results. In very rare cases malignant disease of the heart proceeds to ulceration.

**SYMPTOMS.**—Of thirty-six cases of malignant disease of the heart, the histories of which were collected by Sir Richard Quain

(*Lumleian Lectures*), in thirty no symptoms were present, or they were not recorded. In one of the remaining six cases, the subject of the disease—a man of thirty-seven, in whose heart a single large mass of encephaloid cancer was found *post mortem*—had been subject to attacks of excruciating pain in the præcordial region, dyspnœa, palpitation, and vomiting; and death occurred suddenly. Pain in the chest and oppression, not referable to other causes, are recorded in two other cases; in the fourth case there were anginal seizures. In cancer of the heart spreading from the mediastinum or lungs, dyspnœa, cough, and pain are necessarily frequent symptoms.

With respect to the physical signs of malignant disease of the heart, tenderness on percussion over the præcordia (in association with local pain), pericardial friction and endocardial murmurs due to involvement of the valvular apparatus in the new-growth, appear to be the only phenomena that have been specially observed.

**COURSE.**—The disease naturally ends in death; and in more than one instance this termination was sudden, and perhaps directly due to the affection of the heart.

**DIAGNOSIS.**—This condition has probably never been diagnosed during life. The appearance of true cardiac pain, or of any of the physical signs just mentioned, in the course of a case of cancer, would, however, be strong evidence that the heart was secondarily involved.

**TREATMENT.**—The treatment of malignant disease of the heart is necessarily limited to the relief of any symptoms that may be present, and does not differ from the treatment of cardiac distress from other causes.

**2. Lymphoma of the Heart.**—Lymphomatous or lymphadenomatous growths have been met with in the heart in several cases in which the disease was general, but this affection of the organ cannot be said to have any clinical importance.

**3. Non-Malignant Tumours of the Heart.**—These growths are also of purely pathological interest, and are amongst the very rarest of morbid appearances in connexion with the heart. *Myomata* have been recorded as instances of this class of diseases. *Lipomata* lying under the endocardium are referred to in the article HEART, Fatty Growth on.

**4. Cysts of the Heart.**—The occurrence of true cysts in the myocardium (hydatids, abscesses, hæmatomata, and softening gummata being excluded) is doubtful.

J. MITCHELL BRUCE.

**HEART, Palpitation of.**—**SYNON.**: Fr. *Palpitation du Cœur*; Ger. *Herz klopfen*.

**DEFINITION.**—Abnormal movement of the heart, without appreciable structural lesion; the movement, frequent or tumultuous, varying in tone and force.

**ÆTIOLOGY.**—The *immediate* or proximate cause of palpitation is an over-stimulation of the excitability of the muscular structure of the heart, induced by functional errors of the cardiac ganglia and of the vagus, more especially the depressor nerve proceeding from its trunk, or of those other nerves which, proceeding from the ganglia of the great sympathetic, supply the heart; it being borne in mind that the cardiac centre in the medulla is subject to impressions co-existent with the whole nervous system, and that these are transmitted by the vagus or sympathetic to the heart. It is therefore a true neurosis. The disordered action of these nerves may be induced either directly by dynamic or adynamic causes, or by reflex action; but in either case the phenomena as regards the heart are the same—namely, the morbid activity or irregularity in a normal function, and which must be here considered as independent of any accompanying organic lesion. In dynamic and reflex palpitation the general blood pressure thus resulting is influenced, partly by peripheral resistance, but mainly by the tumultuous increase in the heart's action. The lessened duration of the diastole adversely determines, first, the period of rest; secondly, the recruiting of the nerve-power of the heart, by lessening the nourishment afforded to its general structure through the coronaries; and, thirdly, the stimulus afforded, by a deficiency in the due amount of blood supplied to the ventricles. As the variability of this duration is measured by the recurrence of the systole (defined and uniform as regards time), the relative frequency, force, and tone of the pulse-stroke become important elements in the appreciation of these forms of functional disturbance. In the adynamic form the palpitation is mainly influenced by an increase in the peripheral resistance effected by morbid action of the vaso-motor system, and evidenced by a marked deficiency of tone and force in the beat.

The *predisposing* and *exciting* causes of palpitation of the heart are various. The chief *predisposing* causes are to be found in the nervous and excitable temperaments; general debility; inanition; physical depression; exhaustion, whether bodily or mental; early age; hysteria; venereal excesses; and in deterioration of the blood, as occurs in gout, scurvy, chlorosis, or spanæmia. Amongst the *exciting* causes may be classed violent exercise; mental shock, emotion, and all forms of sudden excitement of the nervous system; exposure to too low a temperature; dissipation; injurious articles of diet; and dyspepsia.

**SYMPTOMS.**—In the dynamic and reflex forms of simple palpitation there may occur (1) a single action; or (2) a series of actions, which may become prolonged, and of such a character as to be esteemed chronic. The single abnormal beat not infrequently occurs

during a first sleep, and the patient is wakened by a consciousness of it. Sleep may then return, and the attack subside without other inconvenience; or it may be associated with a feeling of weight, fulness, anxiety, sinking, or even pain of the præcordia. More frequently, however, the attacks are prolonged and recurrent, returning with an accelerated and uncertain frequency. To the patient the act of palpitation causes various and widely different sensations. There may be a mere occasional flutter, or a slightly increased action continuing for a time; or there may be increased action attended with great rapidity, and such violence, that the heart appears forcibly to strike the chest-walls, diffusing its influence over the whole sternal region, and even at times agitating the whole body (a phenomenon probably due to an associated general nervous agitation); or the heart, again, may seem to the sufferer to rise, as it were, into the throat. With these several symptoms there may be the accompanying disturbances of choking—the *globus hystericus*; vertigo; tinnitus aurium; impaired vision, with a feeling of distension of the eyeballs; a copious secretion of pale limpid urine; a clammy coldness of the extremities; fear of death; partial unconsciousness; or actual syncope. These attacks may be preceded by a somewhat prolonged state of cerebral disturbance, as evidenced by heat of brow and vertex, headache, and an inaptitude to think or regulate the thoughts; and as there is generally a self-consciousness of the abnormal action of the heart, the anxiety on this account serves to impress the mind with so much fear and inquietude as to tend to increase and prolong the disorder that has induced them. In the adynamic form there is usually increased frequency with failure of systolic power, so that the pulse becomes deficient in tone, force, and rhythm, and may be even reduced to a mere flutter. The patient is painfully sensible of the disturbance, which induces a feeling of restless anxiety; relief is sought, but not easily obtained, either by moving about or in repose. When this adynamic form of palpitation has its causation primarily from undue depression of temperature, in the peripheral circulation and subsequently in the venous system and right heart, these symptoms become more urgent—may be to the extinction of life; the vaso-dilators of the arteries are paralysed; the systemic current fails, and hence the cerebral vessels and the nutrient vessels of the heart itself are unfilled, the immediate result being syncope and death. This is probably the frequent course of events in drowning. It is certainly so in those cases of death from the injudicious use of the cold bath, taken when physical exhaustion or other circumstances interfere with the necessary reaction from the shock.

**PHYSICAL SIGNS.**—The physical examina-

tion of the heart in the dynamic and reflex forms of simple palpitation, shows the apex-beat to be normal in position, but diffused and much exaggerated in force. The area of dullness, as a rule, is not enlarged upwards, but it may be temporarily enlarged, under certain circumstances, towards the right side. The sounds, always exaggerated, at times become very much so, and usually with a sharp metallic ring. Occasionally a kind of remitting humming sound is superadded, and may be heard even by the patient; but this is never constant. Sometimes the sounds are heard over a great extent of surface; but this extent is no measure of their intensity, for they may not be loud, but abnormally clear and distinct only. Occasionally there is a pericardial rubbing accompanying the mitral apex-shock, simulating the friction-sound of pericarditis; but there is never true friction-sound. The basic second sound, more frequently than the first, presents the metallic ring. Sometimes it becomes lower-pitched and less clicking than in an ordinary attack of palpitation; and may even, as also the first sound, so lose sharpness and abruptness as to assume somewhat of the character of a soft murmur. The aorta, carotids, and large arteries also throb, and have an excited impulse. The smaller arteries are not sensibly affected. The pulse at the wrist is thus often an uncertain indicator of the amount of action exhibited by the heart. Sometimes it has the character of being sharp and jerking, without force; or, should the right side of the heart become loaded with blood, it may be small and soft, and weak. This is markedly the case in the adynamic form; the impulse may be weak even to extinction, and the sounds so feeble as scarcely to be appreciable, while there are indications of a loaded state of the right heart. The symptoms may be summed up as indicative of depressed irritability, even to suppression, with venous congestion. On the subsidence of an attack, the ventricular impulse may return to its natural force and frequency, and the sounds be unaccompanied by any exaggerations. Nevertheless, though the attack may have subsided, there may be slight irregularity of the heart's action perceptible for some short time afterwards.

**DIAGNOSIS.**—Though the diagnosis of palpitation of the heart in some cases may present difficulty, yet, in the absence of evidence of structural lesion, an increased impulse presenting the above distinctive characters may be assumed to be functional in its origin, and not dependent on any organic disease of the heart itself. We have, in fact, to do with an exaggeration, sometimes highly marked, of the natural nervous susceptibilities of the heart; and this nervous increase of impulse, even when only slight, is usually more appreciated by the patient, more painful, and more a source of anxiety

than is that attending organic disease, especially in its earliest stages.

**TREATMENT.**—Palpitation, in reference to treatment, may be briefly viewed as dynamic, reflex, and adynamic. Reflex palpitation, as irritative in its centripetal origin, is, for the most part, dynamic in character; and therefore its occurrence with the dynamic forms is to be temporarily met by corresponding remedial agents. Those sedatives which combine sedation with diffusible stimulation are in practice very generally selected, because a rapidly acting heart is not necessarily distributing the blood evenly through the vessels; in fact, the pulse may be no guide to the action of the heart, this being influenced by other causes. These remedies include ether, camphor dissolved in spirit of nitrous ether, ethereal tincture of lobelia, and bromide of ammonium, sedatives combined with ammonia, and carminative volatile oils. The rational treatment, however, even during the attack, is rather to strengthen those measures which before and after are to be used to counteract the cause, and to lessen vascular tension, as well as to quell inordinate cardiac action. Aconite, diluted hydrocyanic acid, or the green hellebore, are therefore especially indicated; or the inhibitory action of digitalis and its allies may be called for. In adynamic palpitation the sedative action is not required, but stimulation, either through the nerves or upon the heart itself, is mainly to be relied on. Thus, sumbul, castor, valerian, or alcohol, and ammonia, or compound tincture of lavender, and allied medicines may temporarily relieve the attack. Of all stimulants to an enfeebled and impeded circulation and adynamic inordinately acting heart, nitroglycerine or trinitrin is the most reliable and most powerful; and the allied remedies are to be found in amyl nitrite and the alkaline nitrites. It is important, however, to observe that the action must be maintained in a properly regulated dose, for this should not involve any depressing influence on the nervous system, as cannot generally be avoided when amyl nitrite is given by inhalation; and the dose of trinitrin cannot be measured by a fixed standard of one minim of a 1 per cent. alcoholic solution, for in some cases half the standard dose will be sufficient; in other cases double the amount will be required to produce the requisite degree of stimulant action. T. SHAPTER.

**HEART, Pyæmic Abscess of.**—DEFINITION.—Abscess of the heart occurring in pyæmia.

**ÆTIOLOGY.**—Abscess of the heart has been most frequently observed in cases of pyæmia following acute necrosis of bone or diffuse periostitis; less frequently after phlebitis, chronic or acute arthritis, urethral stricture, chronic abscess, and cancerous ulceration.

In eleven out of fourteen cases, the histories of which were collected by Sir Richard Quain (*Lumleian Lectures*), the age of the patients was seventeen years or under; and twelve out of the same fourteen cases were males. In other words, pyæmic abscess of the heart has been most frequently found in cases following injury to a bone or to a joint in boys. In older subjects it has been associated with pyæmia secondary to one or other of the diseases just mentioned. In a few cases no primary disease was discovered.

**ANATOMICAL CHARACTERS.**—Pyæmic disease of the walls of the heart most commonly affects the left ventricle—towards the base and in the papillary muscles. Pericarditis co-exists in the great majority of cases, and very frequently endocardial inflammation also. The pyæmic foci are generally multiple, and vary in size from a pea to a pin's head. They appear at first as small, slightly elevated, yellowish or buff-coloured, softened patches, projecting either on the external or on the internal surface of the heart, and covered with inflammatory deposit. On section, these patches either present an appearance of diffused yellowish softening, or contain one or more collections of dark dirty puriform matter, with ragged, ill-defined boundaries, as if formed by destruction of the discoloured tissue around.

Microscopically examined, the yellowish patches prove to be portions of the myocardium which are infiltrated with pus and granular matter; the muscular tissue itself being in a condition of granular or fatty degeneration. The puriform material represents an advanced stage of the same change, consisting of granular matter and other muscular debris, blood, various micro-organisms, and frequently pus-corpuscles. The several stages of the pyæmic process have been found side by side in some cases; and emboli have been discovered in the branches of the coronary arteries, where they may have served as the foci of the abscesses. The walls of the heart are sometimes in a condition of softening throughout. Pyæmic abscess of the heart occasionally bursts; and the contents make their way either into the left ventricle—producing cardiac aneurysm, and perhaps giving rise to further embolism and pyæmic disease—or into the pericardial sac.

**SYMPTOMS.**—Whatever the symptoms of pyæmic abscess of the heart may be, they have in recorded cases been completely obscured by the general symptoms of pyæmia, and by the local symptoms and signs of pericarditis. Thus, the patients are described as presenting a febrile, typhoid, or pyæmic appearance, an anxious look, dyspnoea, and præcordial pains; and pericardial friction has generally been discovered over the heart. Delirium probably occurs more frequently than in ordinary cases of pyæmia, but may be referable to the accompanying

pericarditis. The *physical signs* found in these cases are chiefly those of acute pericarditis. Sometimes an endocardial bellows-murmur may be heard, due either to valvular lesion, or to the formation of an acute aneurysm of the cardiac wall.

**COURSE AND TERMINATIONS.**—The cases of pyæmia in which the heart has been found *post mortem* to be involved, have generally proved rapidly fatal, the patients dying from exhaustion. Rupture of the abscess in either direction may tend to accelerate the fatal termination; but complete rupture of the wall in both directions, with sudden death from hæmato-pericardium, as in non-pyæmic abscess of the heart, does not appear to be on record.

**DIAGNOSIS.**—In every case of pyæmia the physical condition of the heart should be regularly investigated; and there should no longer be any risk of acute inflammation of the heart or pericarditis being mistaken for meningitis or simple delirium. Pyæmia with multiple arthritis and involvement of the heart is more difficult of diagnosis from ordinary acute rheumatism with cardiac inflammation; and mistakes in such cases have not infrequently occurred. The history of the case, including the evidence of a definite injury, however slight, is of the greatest value; but a careful consideration of all the facts of the case alone can prevent mistakes. The only difficulty that remains in the diagnosis of pyæmic abscess of the heart is the determination of its existence in the presence of pericarditis, which is rarely absent. For this purpose the facts of the ætiology of the case are more important than the symptoms; and especially the occurrence of an injury to the periosteum of a youthful subject as the original cause of the pyæmia. As a matter of fact, the symptoms, either general or local, appear never to have suggested the diagnosis of pyæmic abscess of the heart.

**PROGNOSIS.**—If a diagnosis of abscess of the heart can be made in pyæmia, the only possible prognosis that can be given is one of speedy death.

**TREATMENT.**—The treatment of pyæmia affecting the heart cannot be said to differ in any important respect from that of ordinary cases of the disease (*see* PYÆMIA). The accompanying pericarditis will call for local treatment.

J. MITCHELL BRUCE.

**HEART, Rupture of.**—The heart is liable to rupture from external injuries, and from causes acting from within. The latter are called spontaneous ruptures, and these only will be considered here. Spontaneous ruptures may affect either the walls or the valves. The latter form of lesion will be found discussed under the head of HEART, Valves and Orifices of, Diseases of.

**ÆTIOLOGY.**—Rupture of the walls may be said never to occur spontaneously when the heart is healthy. The following have been enumerated by different writers as the diseased conditions of the heart's walls that *predispose* to rupture:—A thin or atrophied condition, simple softening, a 'gelatiniform' condition of the walls, apoplectic or hæmorrhagic effusion into the walls, abscess, ulceration, and fatty degeneration. The writer finds from a table of 100 cases of rupture, the histories of which he has collected from different sources, that the heart had undergone fatty degeneration in 77; in 6 the walls were described simply as being softened; in 1 there was rupture of an aneurysmal dilatation; in 1 there was bursting of an abscess; in 12 the heart is said to have been healthy in texture, or not to have been examined; but in most of these latter cases mention is made of the previous existence of endocarditis, or of changes in the coronary arteries, fully justifying the impression that there was disease of the texture of the heart.

The influence of *age* in relation to rupture of the heart can be distinctly traced. For example, of the 100 cases just referred to, 63 were above the age of sixty years. Arranged in decades, the cases stand thus:—2 were between ten and twenty; 1 between twenty and thirty; 3 between thirty and forty; 6 between forty and fifty; 13 between fifty and sixty; 33 between sixty and seventy; 24 between seventy and eighty; 6 were over eighty; and in 2 the age is not stated. With respect to *sex*, of 98 out of 100 cases in which it is mentioned, 54 were males and 44 females.

The *exciting* cause of rupture of the heart is usually some mental excitement or physical effort; but the accident may occur when the subject of it is at rest, or pursuing the ordinary avocations of life.

**ANATOMICAL CHARACTERS.**—*Seat.* In 76 cases out of the 100 to which we have already alluded, the left ventricle was the seat of the rupture; and in 43 of these cases the lesion was in the anterior wall. The right ventricle was found ruptured thirteen times, nine instances occurring in its anterior wall. The right auricle was ruptured seven times; the left auricle twice; and a rupture was found in the septum four times. These results correspond remarkably with those of other writers on the subject. Elléaume (*Mon. des Hôpit.*, 1858) in 55 cases found the rupture 43 times in the left ventricle, seven times in the right ventricle, three times in the right auricle, and twice in the left auricle.

On examining a heart in which rupture has occurred, the torn part is found to present different characters in different cases. The lesion may be *complete*, causing perforation of the walls; or it may be *incomplete*. In *complete* rupture the opening is sometimes barely sufficient to admit a probe, whilst in other instances it may be two or three inches

in length. The rent is sometimes longer externally, and sometimes it is longer internally. There may be but one, or there may be more than one, rupture; and in the latter case the ruptures may or may not communicate with one another. In *incomplete* rupture the injury may be confined to the internal surface, or to the external surface, or it may occur in the substance of the walls. The edges of the rent are ragged, irregular, and sometimes ecchymosed. The irregularity of the edges is due to the manner in which the muscular fibres are torn, whether across or split longitudinally. This description refers more correctly to rupture in a heart that is the subject of fatty degeneration. The appearances are different when the rupture is secondary to an abscess, or to ulceration, or to certain other causes presently to be described. In such cases the lesion has been described as a rent, tear, ulceration, or perforation. The condition of the heart in the majority of cases of rupture has been already referred to under the head of *Ætiology*. Ecchymoses are sometimes found in the vicinity of the lesion. The pericardium generally contains an effusion of blood, which often surrounds the heart with coagulum, leaving the sac filled with serum, to the amount, it may be, of thirty ounces, as in an instance which came under the writer's notice.

**MECHANISM.**—Rupture of the heart is doubtless nearly always the result of a strain or of pressure acting upon the muscular walls. The walls of the healthy heart are sufficiently strong to resist any ordinary force to which they are exposed. But when they are softened by degeneration, or are very thin, as is sometimes the case in the auricles or the right ventricle, they may give way before the pressure to which they are exposed during muscular efforts or strains, or even in the ordinary action of the organ. Thus, when a part of the wall of the heart is weakened by softening or other cause, this spot may be, as it were, torn across by the contraction of the healthy fibres among which it is situated. Or again, when the walls of the heart are thick, it may be that the outer surface, being strained over the contents of a distended ventricle, as would be the outer surface of an overbent hoop, gives way, tears, and the opening gradually extends from without inwards. These facts enable us to understand why rupture is more frequent in the left than in the right ventricle. A further explanation is to be found in the fact that the left ventricle is more frequently than the right the seat of fatty degeneration, from causes elsewhere alluded to (*see* HEART, Fatty Degeneration of). There is yet another way in which softening leads to rupture. A softened spot occurs in the substance of the heart, and into it hæmorrhage takes place, constituting what is termed *apoplexy of the*

*heart.* At times this hæmorrhagic softening may yield either externally or internally, and give rise to rupture. Lastly, the writer has seen more than once a small spot of softening with loss of substance occurring on the internal surface of the ventricular wall, most frequently in the left; this softening and breaking down of tissue gradually insinuates itself amongst the muscular fibres, until finally perforation of the outer wall of the heart occurs.

**SYMPTOMS.**—The symptoms of rupture of the heart may be described as those which are premonitory; and those which occur at the time of the accident. The former are such as indicate a diseased condition of the organ—namely, breathlessness on exertion, palpitation, more or less irregularity of pulse, and faintness. In some instances recorded these symptoms were so slight as hardly to attract attention; in others so severe as to cause intense suffering. In the majority of the cases noted in the table referred to, no mention is made of any preceding symptoms, death being sudden. In several cases it is distinctly stated that no symptoms preceded the fatal attack.

The occurrence of the lesion itself, when the patient has lived long enough to describe his sensations, has always been marked by intense cardiac suffering, more or less distress in breathing, restlessness, rapid and irregular pulse, faintness, pallor, coldness of the skin, sometimes vomiting, and by various nervous symptoms. When life is prolonged beyond a few minutes, there may be more or less intermission in the progress of these symptoms; but the whole attack is marked by anguish more or less severe. The duration of the attack itself from the first fatal seizure varies remarkably. In 71 out of the 100 cases alluded to, death was sudden, occurring within one or two minutes. One patient, however, lived eight days, 1 six days, 1 three days; 5 lived over forty-eight hours, 3 lived under twenty-four hours, and 19 under twelve hours.

The special symptoms indicative of a fatal seizure are, in addition to those already mentioned, severe præcordial pain, dyspnœa, vomiting, cyanosis, pallor, loss of consciousness, and convulsions. These symptoms, or some of them, were noted in 44 out of the 100 cases; and in 24 of these the patient lived for more than five minutes after seizure, and in some of the cases for more than twelve hours. These cases, doubtless, are instances in which the muscular fibres are torn apart layer by layer successively. In the other 20 cases the patient was seized with severe pain, and then expired; or with dyspnœa and some of the other symptoms mentioned above, and lived but a few seconds.

The *physical signs* of complete rupture having occurred, so far as can be ascertained, are merely a greater or less amount of dul-

ness in the region of the heart; the impulse diminished; the sounds muffled, distant, or imperfectly developed; and the pulse weak and intermittent.

**COURSE AND TERMINATIONS.**—The difference in the progress of the fatal malady depends much upon the seat of the rupture, on the size of the opening, and on the rapidity with which the extension of the laceration takes place. In the cases in which the septum is torn, there is no external hæmorrhage, and life is prolonged until the patient dies from disturbance in the functions of such an important organ as the heart. (*See a case reported by Dr. Peacock, Path. Trans. vol. v.*) The progress of the symptoms is also influenced by the direction and course of the rupture. If the torn fibres overlap from the inside or from the outside, the injury penetrates slowly through the cardiac wall, and the fatal progress is also slow. (*See cases recorded by the writer in the Path. Trans. vol. iii., and also in vol. xii.; and a case by Dr. Peacock in vol. xvii. of the same Trans.*)

**PROGNOSIS.**—As far as is known, rupture of the heart is always fatal. Still it is possible that such an accident, owing to the small size of the opening, its incomplete character, and its occlusion by a coagulum, may not prove fatal. Numerous instances are recorded of severe wounds of the heart, the subjects of which have survived. Ollivier has collected 29 such cases, only two of which proved fatal within forty-eight hours, the others living from four to eight days. Cases are recorded in which persons have survived many years severe wounds of this important organ. These cases, however, differ from those of rupture in this particular, that they occur in the healthy organ, whilst spontaneous rupture occurs in the heart when it is seriously diseased. *See HEART, Wounds of.*

**TREATMENT.**—In the way of treatment of rupture of the heart little can be done. The patient's sufferings may perhaps be relieved by the hypodermic injection of morphia, or by the use of sedatives or antispasmodics. Perfect rest should, if possible, be maintained.

RICHARD QUAIN.

**HEART, Softening of.**—This term was formerly applied to several conditions of the heart in which the consistence of the cardiac tissue was diminished, whilst the process to which it was due was obscure or anomalous. It is probable that under the name of softening of the heart there were especially included instances of acute myocarditis, parenchymatous degeneration, and fatty degeneration. In the present more advanced state of cardiac pathology, it seems desirable that the expression 'softening,' while retained to express a familiar physical condition, should cease to be employed as a classifying term—that is, to designate any specific morbid state.

J. MITCHELL BRUCE.

**HEART, Syphilitic Disease of.**—Syphilitic disease of the heart is by no means a rare condition, having been found in a large number of instances in which the specific nature of the lesion was determined with certainty; whilst, in another series of cases, similar anatomical appearances were present, although the existence of syphilis was not ascertained. Syphilitic disease of the heart is therefore of much pathological interest; but it cannot be said that a great deal is known as yet with respect to its clinical history.

**ETIOLOGY.**—There appears to be nothing of importance known as to the causes of the location of syphilis in the heart. The congenital as well as the acquired form of the disease has been met with.

**ANATOMICAL CHARACTERS.**—This morbid condition presents two leading appearances *post mortem*. The first is the well-marked syphilitic *gumma*, which closely resembles the same form of growth as it is met with, for example, in the liver and testicles. Gummata of the heart appear as pale yellow patches in the cardiac wall, or as yellowish nodules which are found on section. They present a variety of appearances, according to their age. When young they are firm or even scirrhoid, elastic, and homogeneous; break on section; and are very slightly succulent: when older, they become soft and cheesy, like a mass of 'yellow tubercle.' In either form the masses are not isolated, but pass continuously into the myocardium, either directly, or through the medium of soft vascular connective tissue, so that they were formerly described in this country as 'infiltrations' or 'fibrinous deposits.' The superjacent endocardium or pericardium is vascularised and dull in the early stage of the nodules; opaque and thickened in the more advanced stage. The masses or nodules occur in various numbers in different instances, but are generally multiple. They may be found in any part of the heart. Gummata most frequently become caseous in the centre, as described; and they may then soften more completely and discharge inwards, leading to acute cardiac aneurysm and ulcer of the wall; but more frequently the cheesy products are in a great measure absorbed, leaving a puckered fibroid patch behind.

The second form of syphilitic disease of the heart is the *fibroid patch*. This is sometimes well-defined and localised, and in such instances it represents the stage of full development of an area of ordinary syphilitic interstitial inflammation. In other specimens, the fibroid patches appear as irregular masses of indurated fibroid tissue, occupying part of the wall of the heart, and sending septa into the depth of the myocardium, whilst the endocardium and pericardium that correspond to them are opaque, thickened, and puckered. The syphilitic

nature of such patches may be determined by the presence of specific lesions in other viscera.

A form of the disease intermediate between the two forms just described is one in which the outer zone of the gumma has undergone development into fibroid tissue, and the caseous centre remains as a 'fibrinous' mass.

The microscopical characters of syphilitic growths do not require to be described here. In the heart, the primary seat of the disease is the intermuscular tissue; the muscular fibres lying imbedded in the gummatous products or in the fibroid growth being either healthy in appearance, or fattily degenerated and broken up.

Syphilitic endarteritis (*obliterans*) may also occur in the vessels of the myocardium, and give rise to infarction of the walls of the heart.

Amongst the occasional effects of syphilitic disease of the heart are chronic aneurysm of the walls; distortion of the ostia and of the valves and their appendages; and, more frequently, adhesion of the pericardium. Some of the other viscera present, as a rule, evidence of syphilitic disease.

**SYMPTOMS.**—The subjects of syphilis of the heart may, from a clinical point of view, be divided into three classes. The first class of patients suffer from some one or other of the ordinary symptoms of chronic cardiac disease, such as dyspnoea, cardiac distress, palpitation, pulmonary complications, and general dropsy; whilst the physical signs are those of cardiac enlargement, and perhaps of valvular incompetence. Præcordial uneasiness, syncopal attacks, and remarkable infrequency of the pulse, have been prominent features in several recorded cases.

The second class of subjects of this disease die suddenly, after few if any complaints referable to the heart.

The third class die of syphilitic marasmus, and may or may not present some evidence—by physical signs or otherwise—that the heart is not sound.

In many of the cases, other symptoms of visceral syphilis have been prominent—for example, phenomena connected with the brain and nervous system.

**DIAGNOSIS.**—Well-defined symptoms or physical signs connected with the heart, such as those just mentioned, occurring in a syphilitic subject, would furnish considerable grounds for the diagnosis of specific cardiac disease, in the absence of other more probable causes—such as a history of endocarditis, strain, or Bright's disease.

**PROGNOSIS.**—If such a diagnosis were positively made, the prognosis would be more favourable than it is perhaps in any other form of chronic heart-disease, inasmuch as the condition might be successfully removed by treatment.

TREATMENT.—Anti-syphilitic remedies, especially iodide of potassium, should be freely tried, along with the other remedies indicated on general principles.

J. MITCHELL BRUCE.

**HEART, Thrombosis of.**—SYNON.: Heart-clotting; Fr. *Thrombose Cardiaque*; Ger. *Gerinnungen im Herzen*; *Herzpolypen*.

DEFINITION.—Coagulation of the blood within the cavities of the heart during life.

ÆTIOLOGY.—Thrombosis of the heart is most frequently due to local arrest of the movements of the blood, comparatively or absolutely, within its cavities. Such arrest is itself generally referable to weakness of the cardiac contractions, whether associated with dilatation secondary to valvular, renal, or pulmonary disease, or due to some primary affection of the muscular walls. The peculiar saccular condition of the extremities of the auricular appendages, and the trabecular arrangement of the columnæ carneæ of the ventricles, as well as the distance of the same parts from the main blood-currents, determine the favourite localisation of the thrombosis. Roughening of the endocardium is another cause of thrombosis, but one which is to be considered less common than the causes already mentioned, unless the fibrinous coagula of endocarditis, or vegetations, be regarded as thrombi, which, in the strict sense of the term, they partly are. Possibly certain conditions of the blood may contribute to the occurrence of cardiac thrombosis. Finally, thrombi once formed tend to promote the further progress of the condition.

ANATOMICAL CHARACTERS.—Coagula found within the heart are of two kinds, which have been termed *active* and *passive*, according as they are formed during life, or at or after death, respectively; and the characters of the former, with which alone we are here concerned, cannot be understood until those of the latter have been briefly described.

*Passive* coagula are found in the heart in most necropsies, occupying the track of the principal blood-currents. Frequently they appear as black or red blood-clots, occupying the auricles principally, and moulded in their cavities. In other cases they take the form of masses of firm whitish fibrin, cleaving with some tenacity to the endocardium, but not truly adherent; matted with the chordæ tendinæ and columnæ carneæ; and projecting some distance into the pulmonary artery. Or, thirdly, passive coagula may be a combination of the two previous forms, the upper part (according to the position of the body) being decolorised or fibrinous, and the deeper part resembling more an ordinary blood-clot. In certain cases these passive clots are peculiar. In

phthisis and other diseases proving fatal by very slow exhaustion, they are remarkably firm and fibrinous, and closely matted amongst the chordæ tendinæ—appearances which seem to indicate that coagulation was slowly proceeding for some time before the heart had finally ceased to beat. In anæmia they are jelly-like and translucent. In leukæmia they are soft and creamy in appearance, and yield, when broken up, a puriform fluid. In the acute exanthemata these passive clots are soft and friable; and in many cases of these and of other forms of acute disease and of sudden death, no coagula are found in the heart, which contains only fluid blood.

*Active* coagula—the result of thrombosis of the heart—are, on the contrary, situated in the saccular appendages of the auricles, at the apex of the ventricles, and in the recesses behind and between the columnæ carneæ—in other words, as far as possible from the track of active blood-currents. In these situations they may be seen projecting in the form of fleshy knobs or globes, with their free surface smooth and rounded. Their deep surface is adherent to the endocardium, from which, however, it can generally be separated without much difficulty, leaving behind it a discoloured mark. If the thrombus be incised, it will be found to be laminated in structure, somewhat after the fashion of an onion, the colour of the section being greyish-brown or yellowish, with irregular patches of red and black. In most instances the centre is less firm than the periphery; and usually it is of a fluid consistence, in the form of a foul, sanious, puriform substance.

If the process of thrombosis have been proceeding for some time, these formations may extend in all directions, embrace the columnæ carneæ, coalesce in front of them, and finally may fill up a considerable portion of one, or even of more than one, cavity. The thrombi are generally friable; but sometimes they gain in firmness by the deposit of lime-salts within them; and at other times it is possible that they become detached and form into the ‘fibrinous balls’ which have been found lying free in the cavities of the auricles. Cardiac thrombi may, in part at least, be reabsorbed. They frequently give way during life; and portions of them, as well as of their puriform contents, are conveyed into the circulation, causing embolism and pyæmia.

**Cardiac Embolism.**—It may be added that embolism of the heart has frequently been found—thrombi or simple clots, sometimes of remarkable size, having been carried from the veins, and arrested in the heart or in the mouth of the pulmonary artery.

SYMPTOMS.—The clinical phenomena associated with true cardiac thrombosis may be best described as those of the last stage of

chronic disease of the heart. Præcordial distress and restlessness; irregularity and feebleness of the pulse; œdema and coldness of the extremities; pulmonary congestion, infarction, and œdema; dulness of expression, and sopor, broken by low weak delirium; with other symptoms, as well as with the signs of cardiac failure and imperfect emptying of the cavities in systole—all these phenomena are associated with the process of active coagulation within the heart. It would not, however, be correct to describe these phenomena as symptoms directly referable to the thrombosis. All that can be said is, that in such a case thrombosis is probably going on and increasing the embarrassment and the gravity of the condition. An unusual degree of cyanosis appears in some instances. The symptoms of arterial embolism may suddenly make their appearance from detachment of particles of the clots; and, if the puriform contents find their way into the circulation, septicæmia may result.

*Cardiac embolism.*—The dislodgment *en masse* of a large venous thrombosis, and the impaction of the same, or of a 'fibrinous ball,' in one of the ostia of the heart have frequently caused rapid death. See PULMONARY VESSELS, Diseases of.

*Passive coagulation.*—It should be added that the appearance of the 'passive' form of coagulation within the heart, which has been already referred to as a *post-mortem* process, or one occurring *in articulo mortis*, has been differently interpreted by some authorities, who regard passive coagula as formed *ante mortem*, and as giving rise to severe symptoms by the embarrassment which they produce in the circulation. The symptoms caused by this condition are said to be—great præcordial pain and distress; tumultuous action of the heart, passing on to irregularity, flickering, and finally arrest, whilst the pulse is very feeble; urgent dyspnoea; cyanosis; hæmoptysis; coldness of the extremities; deepening stupor; and coma ending in death—in short, the congeries of symptoms which would be referred by most authorities to failure of the muscular walls of the heart, the coagulation being regarded as only another result of the same condition.

**DIAGNOSIS.**—In the presence of the very serious and complex conditions with which cardiac thrombosis is usually associated, the question of its existence can hardly be said to occur to the physician as a point of great importance. An unusual degree of cyanosis, especially if it be progressive, favours the recognition of this state; and in the absence of valvular disease, the occurrence of embolism or pyæmia would tend to confirm it.

**TREATMENT.**—The treatment of cardiac thrombosis consists in the treatment of its cause; and nothing is demanded or can be done for the former which is not indicated

for the relief of the latter. Those authorities who see in 'passive' coagula the evidence of rapid *ante-mortem* thrombosis, recommend the use of stimulants, and even of certain drugs which are supposed to have a solvent effect on fibrinous formations, especially ammonia.

The treatment of *embolism* of the pulmonary artery is described separately.

J. MITCHELL BRUCE.

**HEART, Tuberculosis of.**—Independently of the pericardium, the heart itself is believed to be rarely the seat of tubercular disease. Grey miliary tubercles have been found in the connective tissue of the wall of the heart, in some cases of acute general tuberculosis. Tubercle bacilli occasionally occur on endocardial ulcers. In other instances the 'tubercle' has been of the yellow or cheesy kind, in the form of small nodules lying at various depths in the muscular tissue beneath the pericardium; the latter also being frequently affected, as well as the lungs, intestines, and other organs.

There appears to be no evidence that tuberculosis of the myocardium gives rise to definite symptoms, or that it can be recognised during life.

J. MITCHELL BRUCE.

**HEART, Valves and Orifices of, Diseases of.**—**CLASSIFICATION.**—The diseases of the valves and orifices of the heart which produce mechanical disorders of the circulation, by establishing abnormal relations between those parts, are of two kinds—*obstructive* and *regurgitant*. Valvular disease, on the one hand, is said to be *obstructive* when narrowing of an orifice presents an obstacle to the passage of the blood-current—a condition better named *stenosis*. On the other hand, when the blood regurgitates or flows back through an orifice, in consequence of imperfect closure of the valves, due either to valvular changes or to widening of the orifice, the condition is called *regurgitation* or *insufficiency*.

*Aneurysm* of the valves of the heart is discussed separately. See HEART, Valves of, Aneurysm of.

**ÆTIOLOGY.**—Each of the orifices may be affected with one or both forms of disease, but the frequency with which the several orifices are attacked varies. The results of organic disease are chiefly met with in the left side of the heart, and are due to local inflammation—endocarditis and its consequences; or to chronic degenerative changes, such as atheroma. In adult life the valves of the left side are more frequently affected than those of the right, because they have to bear a much greater pressure; but in fetal life, when the pressure is reversed, the right valves suffer more. Endocarditis is commonly of rheumatic origin, and attacks the

mitral more frequently than the aortic valves; the former having to sustain the full force of the ventricular systole, while the latter only bear the force of the aortic recoil. In addition to rheumatic fever, the chief diseases which tend to develop endocarditis are pyæmia, puerperal fever, the exanthemata, diphtheria, chorea, typhoid, tuberculosis, chronic renal disease, and syphilis. The aortic valves and orifice are more commonly affected than the mitral by chronic endarteritis extending from the aorta, the chief causes of which are gout, old age, syphilis, and the abuse of alcohol. These facts explain why mitral affections (commonly rheumatic) occur mostly in early life, and aortic affections in later life. Valvular lesions are more common in men than in women, from the strain of the heart incidental to more laborious occupations. Strain helps to swell the greater proportion of disease of the aortic valves, which are liable to rupture from effort; but similar accidents may occur to the mitral valve and its tendinous cords. Valvular defects are also due in some cases to congenital malformations.

**ANATOMICAL CHARACTERS.**—The pathological changes in the valves and orifices of the heart, which cause valvular diseases, are mostly the results of acute or chronic endocarditis. In the acute form, the valvular lesion is caused by the growth of vegetations which interfere with the action of the valve-segments; or by softening and ulceration of the valve-structure, which lead to valvular aneurysm and perforation, or to loss of substance and consequent insufficiency. The more chronic form of inflammation produces thickening of the valves from overgrowth of the connective tissue, with subsequent calcareous degeneration, and retraction from shrinking of the hyperplastic connective tissue; or adhesion of the valve-segments causing stenosis.

*Aortic stenosis* is generally the result of thickening and calcareous degeneration of the valves, or of deformity of the valves from vegetative growths which obstruct the free passage of the blood from the ventricle. Sometimes it is due to adhesion of the valves preventing their elevation, and causing them to form a diaphragm with a narrow central aperture. More rarely it is caused by thickening and contraction of the fibrous ring of the aortic orifice, gradually involving the valves; or by endocardial thickening producing contraction immediately beneath the aortic ring.

*Mitral stenosis* results most frequently from a leather-like thickening and rigidity of the valves, which contract all round the mitral orifice, so as to narrow the outlet, and form a diaphragm between the auricle and the ventricle. This diaphragm is in some cases funnel-shaped, while in others the mitral orifice is transformed into a button-

hole aperture, or narrowed to the size of a goose-quill. The tendinous cords of the valve are shortened, and their muscles thickened. In some cases the valves are smooth and thin; in others they are thickened, studded with vegetations, rough and calcareous. This latter state may cause stenosis, without any funnel-formation, as may also fibrinous clots or polyply obstructing the orifice. In many cases of mitral stenosis the valves are also insufficient. Mitral stenosis is mostly observed in young females, as a result of mild rheumatic attacks or chorea. It is sometimes called congenital in cases in which it can be referred to no acute illness.

*Aortic insufficiency* sometimes depends on dilatation of the aortic orifice, due to softening of the aortic coats, with little or no change in the valves, which are incapable of closing the enlarged orifice. This may be called *relative* insufficiency. Vegetations, thickening, retraction, calcareous degeneration, adhesions, perforations, loss of substance, and rupture of the valve-segments by effort are all causes of aortic insufficiency. In rupture of the valves, a full description of which was first given by Sir Richard Quain (*Edin. Monthly Journ.* 1846), the valve-segment is torn from its angle of attachment, and its free edge retroverted towards the ventricle. This accident happens more frequently in cases where the valves were previously diseased by chronic inflammatory changes connected with the strain of very laborious occupations, and in such cases further laceration may subsequently occur.

*Mitral insufficiency* is due to thickening, retraction, or deformity from vegetations of the valve-curtains; adhesion of the curtains to each other or to the ventricular wall; and calcareous degeneration. In some instances, one of the valves is perforated or torn; and sometimes the tendinous cords are shortened and thickened, or ruptured as the result of degeneration, preventing the normal action of the valve-curtains. In rarer cases associated with dilated ventricle, the papillary muscles are so weakened by degeneration that they can no longer aid in the closure of the orifice. Dilatation of the left auriculo-ventricular orifice is also a cause of *relative* mitral insufficiency. This form is unconnected with structural valve changes, and is due to defective muscular contraction, such as occurs in anæmia, typhoid fever, and the failing systole of the later stages of aortic insufficiency.

*Valvular defects on the right side of the heart* are due to similar changes. They arise chiefly during fetal life, when the right cavities have to bear greater pressure. In adult life these defects are generally associated with diseases of the lungs, which cause increased tension in the right cavities, and lead to their dilatation.

*Combined valvular lesions* are not infre-

quent. The most common are stenosis and insufficiency of the aortic valves, and the same morbid changes of the mitral valves. In the last stages of both forms of aortic valve-disease, the mitral valve becomes insufficient, either from chronic endocarditis, or from dilatation of the ventricle and of the auriculo-ventricular orifice. Mitral stenosis is not infrequently associated with aortic insufficiency, but is more commonly connected with some degree of narrowing at the aortic orifice. Tricuspid insufficiency is usually met with in the last stages of diseases of the left heart; and tricuspid stenosis is met with in connexion with a similar narrowing of the mitral orifice.

**SYMPTOMS.**—Valvular diseases of the heart produce a series of morbid phenomena, which are connected together by a necessary sequence. Each and every form of valvular defect impairs the perfection of the heart as a pumping machine, and disturbs the normal relations between the contents of the arteries and of the veins. Wherever the valve-mischief is, and whatever its nature, it robs the arterial circulation and enriches the venous. In front of the lesion there is less blood; behind it there is more. In aortic valvular diseases, the first effects are increase of the blood-pressure in the left ventricle, and lessened blood-pressure in the aorta; next, from the difficulty which the left auricle has in emptying all its contents into an over-full ventricle, there is produced increased pressure in the left auricle and pulmonary veins. Mitral valve-lesions cause similar results: first, increased pressure in the left auricle, less pressure in the left ventricle, and consequently lessened pressure in the aorta; with a gradual increase of pressure extending from the left auricle to the pulmonary veins. Aortic affections thus act first on the arterial, and secondly on the pulmonary circulation; while mitral lesions affect the pulmonary vessels more immediately. The final results of the two forms are, however, identical, and may be stated in the form of a law—namely, that all valvular diseases of the heart tend to lessen the quantity of blood in the arterial system, and to produce increased fulness and stasis in the veins. From the action of this law, various associated visceral disorders follow as consequences. These disorders, however, vary greatly in the period of their occurrence, and in the intensity of their manifestations. This variation is due to the more or less perfect way in which the original valvular defect has been compensated for, by changes in the power of the cardiac muscle and in the capacity of the cardiac cavities. These changes often suffice to maintain fairly the normal balance between the arterial and venous contents, thus *compensating* for the valve lesion; and the process by which this is effected demands careful consideration.

*Compensation.*—Compensation is effected

differently, according to the form of disease. It may be stated, generally, that it consists in hypertrophy of the cavity immediately behind the defect. Now hypertrophy means increased contractile power, and this means better filling of the arteries, and consequently increased arterial tension. Thus it makes up for the valvular incompetency, which tends to lessen arterial tension. When the increased power of the ventricle exactly balances the effects of the valvular mischief, the compensation is complete.

In *aortic stenosis*, hypertrophy of the left ventricle is the mode in which compensation is effected; the obstacle to the blood-current is overcome by the increased muscular power.

In *aortic insufficiency* there is some dilatation of the ventricle as the primary result of the lesion. This is counterbalanced by greater hypertrophy; and as long as the dilatation does not progress, the insufficiency is compensated for. A sufficient excess of blood is thrown into the aorta at each systole to allow for the regurgitation during each diastole; and thus the balance is maintained, though not always equally.

In *mitral lesions* the left auricle is dilated as the primary consequence of the condition of the valves; hypertrophy follows, but is insufficient to prevent increased fulness of the pulmonary veins. This impedes the circulation in the lungs; and increased tension in the pulmonary artery soon begets the necessary hypertrophy of the right ventricle. It is by means of this increased power of the right ventricle that the blood is driven through the lungs in spite of the defect in the left heart, and pulmonary stasis is prevented; and the blood entering the left auricle under greater *vis à tergo*, the compensation of the valvular defect is effected. The compensation, from the nature of the means on which it depends, is manifestly less perfect than in aortic lesions.

On the *right side of the heart* similar modes of compensation are observed.

The basis of the salutary changes just described is increased cardiac nutrition; and, consequently, a free coronary circulation is a necessity. Conditions which interfere with this prevent compensation, and so diminish the duration of life. Wherever the compensation begins to fail, dilatation of the cavities and vessels behind the lesion commences. This may, however, be checked, and the power of the heart restored for a time. Sooner or later, however, changes in the nutrition of the cardiac muscle, in the vessels, and in the general nutrition, bring on failure of compensation. The cardinal symptom in such cases is weakened contractile power of the heart, or *astholie* (Beau). In this state, the cavity chiefly affected has no longer power to expel its contents fully into the vessels, and consequently becomes

gradually and increasingly distended. Failing compensation in aortic valve-disease manifests itself by dilatation of the left ventricle, and the development of secondary mitral insufficiency. Similar retro-dilatation marks the failure in mitral cases, only here it is the right ventricle which dilates, and tricuspid insufficiency and general venous stasis are added to the pre-existing pulmonary engorgement.

The earliest *symptoms of failing compensation* are attacks of palpitation from very slight exertion or excitement, or during sleep. Irregularity of the pulse soon follows, if it have not previously existed. This is especially the case in mitral disease. The irregularity is due not so much to true cardiac intermission as to abortive contractions, which do not reach the wrist: or to contractions unequal in force, or in the quantity of blood expelled. The pulse is small, unequal, irregular, and compressible. In aortic valvular disease true intermissions occur, and are of grave import. With failing cardiac power there usually supervene cardiac oppression, anginous attacks from distension of the cavities of the heart, and faintness and giddiness from cerebral anæmia.

*Visceral complications.*—The most important of the associated disorders of chronic valvular disease, depending on defective contraction of the heart, are the visceral congestions.

In the *lungs*, the habitual engorgement of mitral diseases produces a hyper-secretion of mucus and a state of chronic catarrh. The blood-vessels also undergo changes from the excessive intravascular pressure, and become dilated, varicose, and atheromatous; whence œdema and hæmorrhage arise. In mitral stenosis especially, grave and frequent attacks of hæmorrhage, with laceration of the pulmonary substance, are liable to occur; these attacks are called pulmonary apoplexy, and the lung-condition splenisation. The lungs, from long-continued congestion and œdema, undergo brown induration. The varicose condition of the vessels in the alveoli interferes with oxidation, both by favouring stasis and by mechanically narrowing the alveoli, and so aids in the deterioration of blood, which the other visceral congestions favour.

In the *liver*, the general venous stasis is felt by the obstruction to the passage of blood from the hepatic veins into the inferior cava. Passive congestion ensues, and 'nutmeg liver' results. This term 'nutmeg liver' refers to the rough changes in the viscus, the dark congested centre of each lobule being surrounded by a paler area. In course of time the compression of the central cells of each lobule by the distended veins leads to atrophy, and the growth of a hyaline fibroid tissue, which causes the toughness; the liver, from being large, shrinks in

very chronic cases to half its size; and the condition may, like true cirrhosis, lead to ascites. The passive congestion in the liver, as in the lung, causes catarrh of the tubes, and may thus be productive of jaundice. Amongst other symptoms associated with the hepatic congestion may be mentioned hæmorrhoids and epistaxis.

The *spleen* is a very easily distended organ, and suffers like the liver, but frequently before it; and this may partly account for the pain in the early stages, which is complained of beneath the left ribs. In long-standing cases the spleen becomes tougher, and the capsule opaque and thickened; while hæmorrhagic infarcts are common.

From the hepatic congestion there naturally follows distension of all the other radicles of the portal vein; hence the congestion and chronic catarrh of the *stomach* and *intestines*, which impede digestion and assimilation, and so reinforce the other causes producing the cachexia of chronic valvular disease.

In all cases of valvular disease, when the mechanical effects extend to the general circulation, the function of the *kidneys* is more or less disordered. The first stage of general circulatory trouble is lessened arterial tension; this makes itself felt in lessened pressure in the Malpighian tufts, and is manifested by scanty, dense, high-coloured urine. When the more advanced circulatory trouble—namely, general venous stasis—is developed, a further change takes place in the urine. The arterial anæmia keeps it still scanty and dense, but the venous stasis in the kidney leads to the transudation of serum—a dropsy of the kidney, as it were; and consequently albumen appears in the urine. Long-continued venous congestion ends in structural changes, which, as elsewhere, consist in connective-tissue hyperplasia and degenerative (rarely fatty) changes in the tubules. These renal changes sometimes add uræmia to the patient's ailments.

In the *brain* decided alterations are not found, except when a detached vegetation produces embolism and its special phenomena. The brain-substance is, however, generally œdematous, and the membranes are thickened. Delirium is an occasional symptom in heart-disease, and when present to any great degree is of evil import. The blood-vessels of the general circulation are frequently affected with atheroma in hypertrophy of the left ventricle, and it is these degenerative changes, as well as embolic aneurysm, which favour the occurrence of apoplexy.

*General dropsy.*—The mechanical impediments to the circulation which produce these several visceral congestions, also manifest themselves in the general dropsy which is common in the last stages of heart-disease. The dropsy begins as a puffiness of the ankles, especially the left, at bedtime. The general

venous stasis, thus first indicated, advances slowly and surely, if not checked, to general anasarca, and even to dropsy of the serous cavities. The increased venous tension, and the hydremia of blood deterioration, are the causes of this serous transudation, which shows itself first in the feet, the most dependent portions of the body, where the pressure of the blood-column is naturally greatest. The horizontal posture, by distributing the pressure, is sufficient at first to disperse the œdema of the ankles. General anasarca is much more frequent in mitral, especially the regurgitant form, than in aortic lesions.

In some cases a solid form of œdema is observed. This occurs mostly in the last stages of valvular affections, and is due to thrombosis of venous trunks, in which, the circulation being much impeded, coagulation easily takes place. The termination of the external jugulars is a common site for such thrombosis; and the left innominate vein, from its transverse position, and from its emptying almost at right angles to the current in the superior vena cava, is, in the writer's experience, more commonly obstructed than the right. Solid œdema is consequently seen more frequently on the left side of the head and neck and in the left arm, than on the right side.

*Defective compensation.*—The phenomena just described are associated with valvular diseases of the heart, as the consequences of partial or defective compensation. These conditions are more or less developed according to the individual case, and consequently give rise to symptoms in varying degrees. These symptoms will now be described.

Palpitation is intimately related to the state of cardiac nutrition and innervation, and has no special connexion with any form of valvular disease. Cardiac pain, varying in intensity from mere uneasiness to the agony of angina, is most common in aortic cases, and is associated with endarteritis, particularly at the root of the aorta, or with dilatation of the left ventricle, or is a neuralgia of the cardiac plexus. In mitral affections, pain arises from over-distension of the left auricle, and its pressure on neighbouring parts, and later on from dilatation of the right ventricle. Dyspnoea may be present in any form of valvular disease, but it is often absent from the earlier history of aortic cases, while some dyspnoea is always present in mitral cases. This difference is due to the absence of pulmonary congestion in aortic affections, whilst it is more or less present from the first in mitral affections. The dyspnoea is a breathlessness rather than a difficulty of breathing. It is panting and gasping in its character, with acceleration of the rate. It is aggravated by any movement, and often compels the patient to sit upright (orthopnoea). Headache, vertigo, dreaming,

night-frights, and sleeplessness are other symptoms, which depend on disordered cerebral circulation. Sleeplessness is one of the most distressing of all symptoms, and can only be relieved when the dyspnoea is lessened. Other more special symptoms will be found in certain cases, and will be traceable to the disturbances in the circulation, which the particular form of valvular disease has engendered.

*Physical signs.*—The physical signs associated with valvular affections may be said to be—first, those of alteration in the size of the heart; and, secondly, those of mechanical disorders of the circulation; together with one or more endocardial murmurs. The persistence of a murmur is the cardinal sign, and if the murmur be either diastolic or præstolic in its rhythm it is of absolute value. A systolic murmur may be caused by poverty of blood—anæmia, especially at the base of the heart. But in such a case—that is, in hæmic murmur—there is no cardiac hypertrophy, as indicated by increased cardiac dulness, though there is often nervous over-action of the heart. There is no accentuation of the pulmonary second sound, inasmuch as there is no extra-fulness of the pulmonary blood-vessels from obstructed circulation. The pulse in anæmia is generally quick, ample, and compressible, but, withal, jerky; while with an organic systolic murmur it is generally slow, rising gradually under the finger, and not very compressible (aortic stenosis), or small, irregular, and unequal (mitral insufficiency). The clinical methods of investigating valvular lesions are mainly inspection, palpation, percussion, and auscultation. The signs of each form of valve-disease are stated below; and on these the special diagnosis rests.

*DIAGNOSIS.*—In *Aortic stenosis* there is often some prominence of the præcordial region, and a steady forcible impulse is perceived below and to the left of its normal position. A thrill, systolic in time, may often be felt at the base of the heart. On auscultation, a loud, frequently rough, rasping, sometimes musical, murmur is heard with the first sound at mid-sternum, and also at the second right intercostal space. The murmur, commencing with the first sound, extends to the succeeding second sound, which is often not very distinct. In relative stenosis with a dilated aorta, the murmur is softer, and the second sound sometimes louder than normal. The murmur of aortic stenosis is audible all over the upper part of the thorax, especially on the right side; is conducted along the great vessels to the left vertebral groove, and it may be even to the lower dorsal vertebræ; and is occasionally heard at the apex of the heart. The pulse is regular, slow, retarded by the narrowing of the aortic orifice, and slowly developed under the finger. The

sphygmogram (*see* fig. 62) shows the line of ascent to be oblique or broken, instead of nearly vertical; the summit is generally blunt; and the line of descent shows small or no secondary waves, and ill-developed dicrotism. Aortic stenosis when moderate, requiring only hypertrophy of the left ventricle for its compensation, is often very perfectly remedied by



FIG. 62.—Pulse-tracing in Aortic Stenosis.

this change, and produces little or no disorder of the circulation. This is also true of cases in which roughness of the valves or vegetations are the cause of even a loud murmur. The amount of hypertrophy of the left ventricle is the surest guide to the gravity of the condition. When the stenosis is very great, epileptiform and syncopal attacks may occur, and lead to sudden death. When the compensation fails, the mitral valve often yields from the dilatation of the left ventricle, and degeneration of the papillary muscles. Then the pulmonary second sound becomes accentuated; the pulmonic circulation is embarrassed; and dyspnoea, bronchial catarrh, pulmonary hæmorrhage, œdema, and cyanosis supervene.

In *Aortic insufficiency*, inspection discovers a more forcible and diffused impulse, lower than natural, sometimes as low as the seventh intercostal space, and outside the nipple line. The præcordial region may be bulging from the violent action of the heart; pulsation may be seen in the upper intercostal spaces at the right edge of the sternum; and a thrill may sometimes be felt there, and occasionally as low down as the heart apex. The great vessels of the neck pulsate visibly. The area of cardiac dulness is increased in all directions, but mainly vertically. On auscultation, a murmur is heard, replacing and following the second sound, of a blowing or hissing character, rarely rough, and lessening in loudness towards its end. It is usually loudest at mid-sternum and in the second right intercostal space; and it is conducted upwards to the right clavicle, but mainly downwards to the xiphoid cartilage. It is not heard at the back of the chest. It may be conducted to the apex of the heart rather than to the ensiform cartilage; and this occurs, in the writer's opinion, when the posterior or mitral segment of the aortic valves is the incompetent one, as the regurgitant current then falls on the mitral valve, and the murmur is thus conducted to the apex. The second sound may be wholly lost at the base of the heart, being replaced by the murmur; but in some cases it is audible—this being due either to normal closure of one or two aortic segments, or to the propagated pulmonic second sound. If audible in the carotids, the second sound is aortic, and is of some value as indicating

partial competency of the valves. The second sound is often audible at the apex. The first sound at the base is almost always modified, being generally murmurish and often obscured by a systolic murmur, due to slight obstruction from thickening of the valve-segments, or to relative stenosis and the vigour of the ventricular systole. In some cases there is no distinct first sound audible at base or apex; its absence being possibly due to noiseless closure of the mitral valve by the intra-ventricular blood-pressure before the systole occurs. Thus in cases of free regurgitation, both first and second sound may be absent over the left heart; but on listening over the femoral artery with a binaural stethoscope lightly applied, the two sounds of the heart can sometimes be heard. In the majority of cases the insufficiency is no doubt associated with some stenosis, and the murmur is double—a short rough systolic portion, with a softer, longer, and more hissing diastolic portion. This double murmur might be well called the *up* and *down* murmur of aortic valve-disease: the two descriptive words indicating the length, and, to some extent, the characters of its component parts.

The signs connected with the pulse in aortic insufficiency are very significant. As the pulsations of the aorta are visible to the right of the sternum, so the arteries often beat visibly all over the body, even to the radial, temporal, and dorsal arteries of the foot. The ophthalmoscope has shown the same phenomenon in the central artery of the retina. This remarkable movement of the arteries is due to two causes—first, to the hypertrophy and dilatation of the left ventricle, which throws an excessive quantity of blood into the vessels at each systole; and, secondly, to the sudden collapse of the arteries, due to the aortic regurgitation. The arterial recoil during the ventricular diastole is not opposed, as in health, by the resistance of the perfectly closed aortic valves, and, consequently, the blood-column is not sustained, and the arteries collapse. These locomotive features in the pulse are generally increased by elevating the arm. The pulse is sudden, short, large, regular, rapidly collapsing, and vibratory. The sphygmographic tracing brings out these characters: the line of ascent is vertical and lofty; the summit is sharp and pointed; the line of descent falls rapidly, and is broken by a series of secondary waves due to vibratory conditions, but has an ill-developed dicrotism. The post-dicrotic portion of the tracing falls rapidly, from the absence of a sustained blood-column. The longer and more oblique this portion of the tracing, *cæteris paribus*, the less copious the regurgitation (*see* fig. 63).

Aortic insufficiency often lasts for many years without producing any obvious disturbance of the systemic and pulmonic circulations; hence the absence of dyspnoea and

cedema. The hypertrophy and dilatation of the left ventricle, which form the compensation, suffice to prevent ill effects. At each



Fig. 63.—Pulse-tracing in Aortic Insufficiency.

systole the dilated ventricle throws sufficient blood into the aorta to allow for the reflux, and to maintain a fair arterial tension. Thus the compensation is perfect. In many cases, however, if the reflux is free, and the coronary segments of the aortic valves are affected, the coronary arteries, which are partly filled by the arterial recoil, are deprived of the full force of the blood-wave, and the nutrition of the heart consequently suffers. This is the great source of failing compensation, and the malnutrition of the cardiac muscle soon leads to dilatation of the ventricle, secondary mitral insufficiency, and atrophy.

When the hypertrophy is excessive (over-compensation), as it is in some cases, there are flushings of the head and face, headache, vertigo, and violent arterial action all over the body.

In *Mitral stenosis* there is rarely any prominence of the præcordial region; and in its earlier stages neither increase of the cardiac dulness, nor alteration in the position of the impulse. The impulse, when regular, is fairly distinct, but it is often very irregular, and is associated with a thrill, which precedes, runs up to, and terminates in the impulse. In advanced cases, the area of cardiac dulness is increased laterally; the impulse is diffused, and may be seen in the epigastrium; this indicates hypertrophy of the right ventricle, which sometimes obscures the left ventricular impulse and gives rise to epigastric and liver pulsation. The left auricular systole may occasionally be noticed, if the chest be thin, in the third left intercostal space. The sounds heard on auscultation in this lesion vary. The pathognomonic sign is a murmur preceding the systole, and ending with its commencement. This is best called the *præsystolic* murmur (also 'auricular systolic'); and is produced when the contracting auricle forces blood under high pressure into the ventricle. It is the passage of a stream of blood in a state of high tension into blood already in the ventricle which causes the murmur. The first portion of the blood passes from the auricle into the ventricle noiselessly; and it is only when a stream of higher tension is forced into it by the true auricular systole that the murmur is developed. The murmur is, therefore, short in most instances, occupying the

last part of the diastole; it runs up to the first sound, and ends abruptly in it. The position in which the murmur is best heard in its typical præsystolic form is limited to the left apex itself or a little above—that is, lower than a mitral regurgitant murmur. The funnel-like shape of the mitral valve-curtains in these cases accounts for this, as the button-hole aperture through which the blood passes is closer to the apex.

This murmur is soft and puffing, but may be harsh. It commonly fills only the last part of the diastolic period. In some cases, with greater roughening and deformity of the valves, the murmur is longer, rougher, more rolling or grinding, and ends abruptly with the first sound, which is very flapping in tone, and might easily be mistaken for the second sound conducted. The careful observation of the impulse or of the carotid pulse with the finger while auscultating, is necessary in order to avoid the error. Mitral stenosis develops three murmurs under different conditions of the valves: (1) A short, softish puff (sounding like *vōōt*) immediately preceding the first sound; (2) a short, rough, grinding murmur (*rrrrb*); and (3) a long murmur filling nearly all the diastole, often rough, but varying in tone in some cases. These several murmurs increase in intensity up to, and end abruptly with, the first sound. The soft, hissing diastolic murmur, occasionally heard at the apex, which lessens in intensity, and ends before the first sound, is a conducted aortic (or pulmonic) murmur.

A special peculiarity of the præsystolic murmur is its variability: it is the only organic murmur which disappears and reappears as the heart-conditions change. For instance, a murmur, inaudible while the patient is at rest, is developed by a little exercise; or, again, an irregular tumultuous action of the heart masks all murmur, which becomes distinct as the heart steadies down under the action of digitalis. In other cases there is no distinct murmur, but only a slightly prolonged and rough or grinding first sound, while the murmur is often lost as the heart-failure advances. In cases of mitral stenosis there is accentuation of the pulmonary second sound, from the greatly increased tension in the lesser circulation; there may also be a doubling or reduplication of the second sound at the base. This reduplication is a sign of great value in cases in which the prolonged or grinding first sound is the only sign. The reduplication of the second sound is due to a want of synchronous closure of the pulmonic and aortic valves, from their altered relative tension. A doubling of the first sound is sometimes noticed, probably due to retarded closure of the mitral valve, from lessened fulness of the left ventricle.

The rhythm of the heart is frequently greatly disordered in mitral stenosis, and

also in mitral insufficiency. A few beats occur regularly, or nearly so, and then a series of very small hurried ones follows, to be again succeeded by stronger and better pulsations. These irregularities are referable to the varying charges of blood on which the ventricle contracts. The over-distended right cavities and the left auricle contract rapidly to expel their contents, but the narrowed mitral orifice does not allow a full charge to pass into the ventricle; the diastole is too short for this purpose; and the wave of contraction passes on to the ventricle from the auricle, producing a series of small ineffective pulse-beats, each representing the small charge sent into the aorta. When the series of ineffective contractions ceases, the next diastole is longer; and the succeeding systole sends, as the sphygmograph shows, a fuller charge into the arteries. During the small irregular beats the præ systolic murmur is often indistinguishable, but it is again heard with the succeeding slower and more effective beats. This irregularity in the heart's rhythm, however, is not present in all cases of mitral



FIG. 64.—Pulse-tracing in Mitral Stenosis.

stenosis. In some instances the heart's action and the pulse are regular. The sphygmograph in such cases records a small pulse of low tension, with a little inequality in the volume of the beats; this inequality is often increased by exercise. In other cases the pulse-tracing is small, irregular, and unequal in its pulsations, and marked by true and false intermissions (see fig. 64).

In the earlier stages of mitral stenosis the face may be pale, and the congestive symptoms which mark mitral insufficiency are absent till the later stages. This form of valvular lesion gives rise more commonly than mitral insufficiency to hæmorrhagic infarction in the lungs; but in other respects the pulmonary and systemic circulations suffer in the same way as described in the other form of mitral disease.

In *Mitral insufficiency* inspection discovers some slight prominence of the præcordial region, with increased impulse, the apex often beating to the left of the nipple line. The area of cardiac dulness is augmented mainly in a lateral direction, from the hypertrophy of the right ventricle. On auscultation, a murmur is heard with the first sound, and following it; loudest at the apex; loud along the left edge of the heart; but absent or not so distinct over the right heart and at the base. The murmur is propagated towards the left axilla, and is audible in most cases

in the left vertebral groove. If not audible posteriorly the murmur signifies regurgitation through a narrowed but patent mitral orifice, and may indicate mitral stenosis, in the absence of either a præ systolic or diastolic murmur. The mitral systolic murmur associated with dilated left ventricle or enfeebled muscle is not usually heard posteriorly. The murmur is usually loud, blowing, and distinct in its character, keeping the same tone throughout. The true first sound is generally obscured by it, but in some cases may be heard through it, and is then due to the partial closure of the mitral, or to the action of the right auriculo-ventricular valves. The pulmonic second sound is commonly accentuated. The pulmonic first sound has in some cases a murmurish character, probably due to hypertrophy and dilatation of the right ventricle, and relative stenosis of the pulmonic orifice.

The radial pulse in cases of fairly perfect compensation is regular, but quick, small, weak, and easily compressible; and the sphygmograph shows low tension, and an



FIG. 65.—Pulse-tracing in Mitral Insufficiency.

inequality in the size of the pulsations. In cases of less perfect compensation, it becomes irregular and intermittent. In all cases the pulse-beat is weak in comparison with the vigour of the ventricular systole (see fig. 65).

There is no unusual fulness of the superficial veins in the earlier stages of mitral insufficiency. Later, when the right cavities become over-distended, the veins of the neck become full and may even pulsate. This is very distinct when the tricuspid valve gives way. In all cases slight exertion is sufficient to induce dyspnoea; and there is an ever-present tendency to bronchial catarrh, from the congested state of the lungs. When asystole comes on, the murmur becomes less distinct; the heart's action is rapid, irregular, and tumultuous; the accentuation of the pulmonary second sound is lost; dyspnoea becomes orthopnoea; and cyanosis, œdema, and hæmorrhagic infarction of the lungs, with general and visceral dropsy, close the case.

Many cases of mitral regurgitation obtain fairly perfect compensation; but the disease, like mitral stenosis, of necessity entails some dyspnoea on exertion, and keeps up constantly an engorged state of the pulmonary vessels.

*Valvular affections of the right heart*, arising from disease, are rare. Those of the *pulmonic valves* are rare; cases are on record, however, of pulmonary stenosis, and

a few of pulmonary insufficiency. In the first case the systolic murmur is loud and superficial, and is heard loudest at the third left costal cartilage close to the sternum, and in the second left intercostal space; it is not usually conducted across the sternum, nor upwards to the right clavicle, as is an aortic murmur. A diastolic pulmonic murmur is soft and blowing; and is heard loudest in the same situations, and downwards towards the ensiform cartilage.

The *tricuspid valves* are more frequently affected. Tricuspid insufficiency is indeed a common sequence of disease of the left side of the heart. Structural changes in these valves are, however, rare. Tricuspid insufficiency does not always produce a murmur; when present this is soft and short, and is heard nearer the middle line than a mitral murmur, at the base of the ensiform cartilage. The pulsations of the cervical veins may indicate the lesion when the murmur is absent.

Tricuspid stenosis causes a præsystolic murmur, harsh in character, loudest at the base of the ensiform cartilage and towards the left edge of the sternum, not propagated towards the left heart, and not audible at the back of the chest, though faintly conducted along the sternum to the base of the heart. A præsystolic thrill may be present. Mitral stenosis has been observed in association with this lesion, and two præsystolic murmurs may be made out in such cases.

The physical signs and the diagnosis of *combined valve-lesions* remain to be described. The mitral and aortic valves may each be affected with stenosis and insufficiency, from a single attack of endocarditis, or from one lesion arising as a consequence of the other. The double aortic murmur, already described, indicates the aortic combination; but it must be always remarked that the systolic murmur in these cases may exist with little or no actual stenosis.

In the double form of mitral disease, either defect may exist alone at first, and afterwards be associated with the signs of the second. In some cases the præsystolic murmur may fail to be heard, and a systolic murmur may alone be audible; in other cases, there is a prolonged apex-murmur which slightly changes tone; in other cases, again, a short grinding præsystolic murmur is followed, occasionally at an interval, by a soft, blowing, systolic one. The combination of aortic with mitral disease may be recognised by the presence of their special murmurs.

**PROGNOSIS.**—It is very difficult to state general rules of prognosis in valvular affections of the heart, as so much depends on the peculiarities of each case. There are, nevertheless, certain broad rules. As regards origin, rheumatic inflammation is less serious than degenerative change, which occurs later

in life, and is necessarily progressive. Accidental rupture is the gravest form of origin. The valve affected is also a prognostic element; but any attempt to arrange cases in order, according to the seat of the valvular defect, must be open to so many exceptions that it must not be too much trusted. Speaking generally, however, tricuspid lesions are gravest, mitral less so, and aortic—especially aortic stenosis—probably least so. When the heart fails, and asystole supervenes, the prognosis is worse, however, in aortic cases than in mitral.

Aortic cases are often free for years from any grave symptoms. When the murmur is conducted to the left apex, the prognosis is more favourable, as, the aortic segment affected being non-coronary, the muscle of the heart is not robbed of its blood. The presence of the second sound over the carotids is favourable. The pulse-tracing also affords valuable aid in prognosis, as it gives, by the size of the dirotic wave and the obliquity of the line of descent, a rough measure of the amount of insufficiency. There is much more risk of sudden death in aortic cases than in mitral.

In mitral lesions the dangers arise from the pulmonary complications; embolism is more common than in aortic affections. Mitral cases can be rescued from asystole more frequently, and die of advanced cardiac cachexia, generally with dropsy. Under favourable conditions of life, requiring little physical exercise and causing no emotional excitement, both forms of mitral disease are compatible with many years of life. When they are conjoined, the prognosis is more unfavourable. In cases of sudden insufficiency, produced by rupture of the valve-curtains or of the tendinous cords, death may occur very rapidly from the disturbance of the circulation.

The whole question of prognosis turns principally on the state of the myocardium. So long as this is sound, compensation may be maintained; the moment degeneration sets in, asystole and all its evil train of symptoms come on. Thus asystole coming on gradually, without any previous overstrain of the heart, is always most grave. Each successive attack becomes graver, and the visceral congestions which accompany it more stubborn. Albuminuria is a marked index of the gravity of the congestion, and is serious in proportion to the frequency with which it has occurred. In some cases a copious flow of limpid urine is a very grave symptom. Dropsy of the extremities, and of the cavities more especially, is bad, as indicating failure in the peripheral circulation. Next to the cardiac muscle, the state of the peripheral vessels is most important; thus, petechiæ are bad signs; while atheroma and other conditions, such as febrile attacks, add to the danger by interfering with the

circulation. The general nutrition of the patient suffering from valvular disease also enters into the prognostic problem. There is a cachexia proper to the end of heart-cases, which is due to the gradual deterioration of the nutritive fluids by the long-continued visceral congestions which hinder assimilation and excretion. Blood is less perfectly made and less perfectly purified; hence the steady deterioration of cardiac cachexia, which is always of evil import as regards duration of life. Valvular diseases are, however, in numerous instances compatible with many years of life—aortic in fairly active, mitral in sedentary persons. In some the healthy expectation of life may be attained; and, in many, years of comparatively active life are enjoyed. In the poor, the prognosis, as to duration, is not favourable; but in the well-to-do, all observers see many cases extending over a great number of years.

**TREATMENT.**—Valvular affections of the heart, whether the result of rheumatic inflammation or of degenerative change, are, as a rule, incurable. Some few cases of rheumatic origin lose the signs of valvular disease, and are practically restored by the after-processes (for example, contraction) in the inflamed valve; and some few cases also of mitral insufficiency, associated with dilated left ventricle and softened muscle, are cured by treatment. These exceptions are, however, few; and as we cannot repair the valve-mischief, in the vast majority of cases, our treatment must be directed to aid the compensatory hypertrophy, and to check the development of the consequences of the defect. The maintenance of the nutrition of the substance of the heart is, therefore, the main object of treatment; just as the state of the nutrition of the heart is the key to the prognosis. On this account the general regimen of heart-cases is very important.

**General regimen.**—The diet should in all cases of valvular disease of the heart be unstimulating but sustaining, consisting of a good proportion of albuminous food (underdone meat, eggs, poultry, and fish), with wine in moderate quantity, and some chalybeate water. There should be no unnecessary excitement of the heart, either by exercise or emotion. All athletics and violent efforts should be avoided by the young, especially in mitral cases. In aortic cases, steady exercise without strain is beneficial. The residence should be so situated as to avoid the necessity of exertion, sudden changes of temperature, cold, and damp. The chief object of the regimen should be to prevent anæmia; hence plenty of fresh air is essential. Tobacco is injurious. In early life over-exertion and exposure to cold—in adult life, emotional, sensual, and dietetic excesses are the chief dangers. The propriety of marriage must be considered in each case on its merits.

Women, as a rule, should not marry; when affected with mitral disease they are often barren. To men marriage is more generally permissible.

**Medicinal treatment.**—The therapeutical treatment varies according to the stage of the cardiac disease. The mechanical defect of a valve first makes itself felt by palpitation and præcordial pain; these symptoms pass away when compensation is effected, but till then require treatment. In cases of mitral disease, tincture of digitalis (℥ x doses) relieves the palpitation; chloric ether is also a useful adjunct. In aortic cases, ether, diffusible stimulants, small doses of opium and belladonna, with the local application of belladonna to the præcordial region, are valuable remedies.

The præcordial pain, mostly retro-sternal, may, when severe, require a few leeches or cupping, but generally yields to mild counter-irritants, such as turpentine, iodine, or mustard. Internally the bromides are useful; when the pain occurs paroxysmally, ethereal preparations and ammonia act well.

When the compensatory changes in the heart are effected, the palpitation and pain decline, and the chief indication is to keep up the nutrition of the heart by the hygienic rules above given, and by the administration of preparations of iron, combined with arsenic, strychnine, quinine, and mineral acids. Chalybeate waters are also useful adjuncts. The syrup, infusion, and tincture of the prunus virginica, are preparations of value in some cases after the use of digitalis. The secretions should be carefully watched, and the bowels opened freely every day, so as to avoid straining, and to relieve the portal circulation. The quantity of urine should be daily noticed, as it is a capital index of the state of arterial tension. Patients in whom the most perfect compensation exists are, nevertheless, in a state of imminent trouble, for an exaggeration of a physiological act or emotion may disturb the unstable equilibrium of their health. In most cases the compensation breaks down sooner or later; and then begin the symptoms depending on pulmonary congestion and general visceral engorgement, with the consequent impoverishment of the blood. Dyspnoea marks the beginning of these troubles; anæmia and dropsy the close. The pulmonary congestion soon manifests itself by bronchial catarrh, which requires expectorants in various combinations, while friction, poultices, and counter-irritation are applied to the chest-walls. In capillary bronchitis with rapid pulmonary congestion, it is sometimes necessary to bleed from the arm to relieve the over-distended cavities of the right side of the heart. Nauseating doses of ipecacuanha, or actual emetics of sulphate of zinc, are sometimes very useful.

For the general visceral congestions our

chief remedies are, firstly, diuretics; and, failing these, hydragogue cathartics. Of diuretics, the salts of potassium, digitalis, squill, broom, chimaphila, spirit of nitrous ether, and juniper are the most useful. The hydragogue cathartics, which relieve the over-distended portal vessels primarily, and the general circulation secondarily, are also most valuable: of these the compound powders of scammony and jalap in 20- to 40-grain doses; bitartrate of potassium in electuary, one to two drachms, every morning; sulphate of magnesium; compound scammony pill; elaterin; senna; and gamboge are the most trustworthy. By the judicious use of an occasional purgative, and the administration of a suitable diuretic, aided by cupping, poultices, and sometimes a small blister over the loins, combined with rest and stimulants, the worst cases of dropsy from cardiac failure are often saved.

For the dyspnoea and the insomnia, two of the worst symptoms, we have a remedy of great power in the subcutaneous injection of morphine in doses of one-sixth of a grain upwards. This remedy acts often like a charm, and may be used even in the worst cases of both mitral and aortic disease, but always with caution. Albumen in the urine does not necessarily contra-indicate its use. In some cases chloral hydrate and bromide of potassium, alone or in combination, are valuable remedies for the insomnia; of new remedies, urethan and sulphonal are the best; paraldehyde is also useful; but they must all be given cautiously. The bromides may be prescribed with other sedatives such as nitroglycerine for the dyspnoea. The compressed-air bath also relieves the last symptom.

Dropsy, like the visceral congestions with which it is associated, requires the use of diuretics and hydragogue cathartics. When these fail, the swollen limbs may be punctured with benefit. Continued friction of the limbs, by stimulating the vessels, will often cause considerable anasarca to disappear. The drug on which main reliance must be placed when general dropsy supervenes is digitalis. It is *par excellence* the cardiac tonic. Convallaria, strophanthus, casca, caffeine, and adonis vernalis are other drugs having similar action—strengthening, but slowing, heart-beat. They are all less trustworthy than digitalis. Caffeine is a good diuretic, and so occasionally is convallaria, even when digitalis has failed. Strophanthus is generally the most useful. Digitalis has so great a share in the therapeutics of heart-disease, and a knowledge of its action is so important, that it must be discussed separately and last. Whatever views may be held as to its physiological action, its greatest triumphs are seen clinically in the treatment of valvular diseases, when cyanosis, distended jugulars, dyspnoea, congested viscera, dropsy-

cal limbs, scanty urine, tumultuous heart-action, and quick, irregular, and failing pulse, indicate asystole. This assemblage of symptoms is mostly seen in mitral cases, and it is precisely in this class that the drug is most valuable. Under its use 'the pulse grows in force, fulness, and regularity; the arterial tension rises; the pulmonary congestion diminishes; the kidneys, before inactive, wake up to their work; and the advancing dropsy recognises its master and beats a sullen retreat.' In mitral stenosis these good results are due not only to the increased vigour given by the drug to the contractile power of the heart, but also to the fact that by its slowing action the diastolic period of each revolution is lengthened, and the time thus increased during which the distended auricle can force its contents through the narrowed mitral orifice into the left ventricle. Digitalis here not only obtains a better filling of the ventricle, but a more effective discharge of its contents when filled: and thus, under its use, beat by beat, the general and pulmonary venous congestion is relieved. In mitral insufficiency it is almost equally potent. In both forms certain of its good effects would seem to be due to some influence, probably through the pneumogastric nerves, in producing contraction of the pulmonary blood-vessels. It is perhaps this property which makes it valuable in pulmonary hæmorrhages, independent of heart-disease.

In aortic valvular diseases digitalis is not so valuable a remedy. In these cases the assemblage of symptoms mentioned above is not met with, except sometimes in the later stages, when the mitral valve is secondarily affected, and the case is not one of pure aortic disease. In these compound cases the drug is valuable, especially in combination with stimulants. In aortic insufficiency alone, the slowing action of the digitalis produces evil by increasing the length of the diastolic period of each revolution, during which the regurgitation takes place. The force it may give to the systole is no gain in the face of this slowing action, inasmuch as the aortic recoil gains in the same proportion as the ventricular systole, and thus forces blood back into the ventricle with increased vigour during the lengthened diastole. It is important in aortic insufficiency to maintain the frequency of the cardiac action; hence these cases are so constantly the better for bodily activity; and so, when the toning effects of digitalis are required, it should always be given in combination with ether and ammonia, to keep up quick action of the heart, and to prevent the vertigo and syncope which may otherwise occur. When there is excessive hypertrophy in cases of aortic insufficiency, digitalis is useful sometimes in quieting palpitation, reducing excessive frequency, and lessening headache and vascular

excitement. Caffeine given with senega, aconite in small doses, and veratrum viride also relieve these symptoms; but a few drops of nitrite of amyl inhaled from cotton-wool, or nitroglycerine tablets, are more rapidly and surely beneficial than any other remedy. In aortic stenosis digitalis is rarely required. The simple mode of compensation makes these cases require little treatment. Digitalis is sometimes useful in combination with stimulants to give vigour to the myocardium, and check the tendency to dilatation. If it slows the action of the heart notably, its effect becomes hurtful. Nux vomica often prevents this.

In combined valvular lesions, the predominant lesion must be the guide in the use of digitalis; but it may be given advantageously whenever the general signs of venous stasis are present. The diuretic power of the drug is one of the best tests of its beneficial action. Relying on this test, the writer often gives digitalis for weeks, nay, months at a time, and obtains improvement in the nutrition of the heart which lasts long after its discontinuance. Digitalis effects this improvement by increasing the vigour of the coronary circulation, and thus builds up new heart-muscle to compensate a valvular defect. In aortic cases the nutrition of the muscle must be kept up by iron, arsenic, quinine, strychnine, and stimulants.

The preparations of digitalis which may be used are, the powder in  $\frac{1}{2}$  to 1 grain; the tincture in  $\mathfrak{m}$ v to xxx; and the infusion in  $\mathfrak{z}$ j to  $\mathfrak{z}$ vj doses. As a diuretic in cases of dropsy, the old combination of squill, digitalis, and blue pill is invaluable.

B. WALTER FOSTER.

### HEART, Valves of, Aneurysm of.—

**DEFINITION.**—A valvular aneurysm is a circumscribed pouching or sacculatation of one of the valve-segments.

**DESCRIPTION.**—Two forms of aneurysm of the valves of the heart are met with. In the one, the whole thickness of the valve is dilated by the blood-pressure to form the pouch; in the other, one of the lamellæ being ulcerated by endocarditis, the blood pushes the remaining lamella before it to form a sac. The second form, which is sometimes called 'acute valvular aneurysm,' occurs most commonly in ulcerative endocarditis. Valvular aneurysms vary in size from a pea up to a pigeon's egg. The orifice is almost invariably towards the greatest blood-pressure—those on the mitral valve opening towards the left ventricle, those on the aortic valves towards the aorta. They are usually rounded in shape, but may have irregular prolongations between the lamellæ of the valves. Valvular aneurysms are sometimes multiple. The valves of the right side of the heart are seldom affected. The mitral valves are the seat of the larger aneurysms,

and are twice as often aneurysmal as the aortic valves.

Valvular aneurysms terminate commonly by early rupture, giving rise to perforation and consequent insufficiency of the valve, and often leading to considerable laceration. Rupture occurs more rapidly in aneurysm of the aortic valves. Mitral aneurysms occasionally become chronic, and filled with coagulum; and aortic valve aneurysm may also be found filled with solid clot.

**SYMPTOMS.**—The clinical history of this form of disease is defective. When seated on the mitral valve, aneurysms usually give rise to no signs until the perforation and laceration of the valve suddenly develop the murmur of mitral insufficiency. An aneurysm of one of the aortic segments causes a soft systolic murmur over the valves, which one day, as the sac ruptures, is supplemented by a diastolic murmur, and accompanied by the symptoms of aortic insufficiency. The phenomena of this accident are similar to those of sudden rupture of an aortic valve.

B. WALTER FOSTER.

### HEART, Wounds of.—SYNON.: Fr. *Blessures du Cœur*; Ger. *Herzwunden*.

The subject of wounds and other injuries of the heart belongs more properly to the domain of Surgery, but, being in several respects of much medical interest, it requires to be briefly discussed here.

#### ÆTIOLOGY AND ANATOMICAL CHARACTERS.—

Wounds of the heart may be punctured, incised, or lacerated; and inflicted with a variety of weapons or other sharp bodies, as well as with projectiles, especially bullets. Traumatic ruptures and contusions form another considerable class of injuries of the heart, which chiefly result from falls, crushing accidents (for example, being 'run over'), kicks, and blows. Injuries of the heart due to the entrance of foreign bodies, such as a needle or a bone, may be inflicted in some rare cases from the interior of the œsophagus or stomach; the most remarkable case of this kind being one of wound of the pericardium from behind by the point of a sword which a juggler had attempted to 'swallow.'

*Post mortem*, the chest-wall generally presents evidence of the wound that has been inflicted. The pericardium rarely escapes injury. Its sac is found to contain blood in recent cases, or effused lymph or pus when life has been preserved for a few days or more. The walls of the heart at the seat of injury present different appearances, according to the precise nature of the lesion. Punctured and incised wounds may be of all sizes; may take either a direct or an oblique direction through the muscular fibres; and are generally penetrating. Bullet-wounds cut away a portion of the heart, whether at the borders or from the thickness of the organ. Traumatic ruptures present special

characters (*see* HEART, Rupture of). In all the varieties of injury of the heart, the wound is found plugged with blood-clot, the edges being either infiltrated or ecchymosed and torn. In cases that do not prove rapidly fatal, the usual signs of inflammation, or healing, and even cicatrization, are found in the heart; or aneurysm of the cardiac walls may be developed as a result of the latter. The valves and their appendages are frequently incised or otherwise injured. In some cases a portion of the weapon, projectile, or foreign body may be found in the heart. The ventricles—and especially the right ventricle—are the parts of the heart most commonly injured. The great vessels, lungs, and the arteries of the chest-wall, may also be wounded in different instances.

It must be observed that a blow over the heart has proved instantly fatal without leaving any lesion discoverable *post mortem*.

From these results it is evident that wounds of the heart may prove rapidly fatal by loss of blood, by compression of the heart resulting from hæmorrhage into the pericardium, or by shock; whilst, at a later stage, pericarditis, myocarditis, and secondary hæmorrhage may be expected to supervene.

**SYMPTOMS, COURSE, AND TERMINATIONS.**—In about one-third of recorded cases of injury of the heart, either death is immediate—fainting, convulsions, and the other symptoms of syncope, as well as those of shock, being the prominent phenomena, along with external hæmorrhage; or the patient drops dead after a few moments, during which time he may have undergone considerable exertion. In a second class of cases, the symptoms of syncope or of shock occur immediately, but death does not ensue at once. The patient then lies in a state either of unconsciousness or of complete prostration. In the latter event he complains of a sense of great debility, præcordial oppression, dyspnoea, and suffocation; the surface is cold, pallid, and trembling; vomiting may occur; and there is usually hæmorrhage from the region of the heart. Death occurs after minutes or hours, either from exhaustion due chiefly to continued or repeated loss of blood, or from compression of the heart. In a third series of cases, the course is more protracted. The patient, after suffering from the symptoms just enumerated, but in a less degree, passes through the various phases of constitutional disturbance commonly observed in severe wounds, complicated, however, with pericarditis, myocarditis, and repeated hæmorrhage; and dies of exhaustion after an illness of weeks or months. Lastly, in a small proportion of cases, the patient survives the various accidents and complications just described, and the wound of the heart heals; but it sometimes happens that symptoms of aneurysm of the cardiac walls, or of incom-

petence of the valvular apparatus, are developed as the result of the lesion.

**DIAGNOSIS.**—Wounds of the heart can usually be diagnosed without difficulty by the situation of the external injury, and the severity of the symptoms. Similar symptoms may, however, follow injuries of the great vessels in the neighbourhood of the heart, or of the arteries of the walls of the chest, if the hæmorrhage be profuse; still, unless there be almost complete certainty that a vessel within reach is the only seat of the bleeding, the diagnosis should be left undecided, and all dangerous interference avoided.

**PROGNOSIS.**—Injury of the heart is generally to be regarded as certain to end in death; but it should not be forgotten that as many as 12 per cent. of recorded cases are said to have recovered. Some very remarkable instances have occurred of recovery after very severe injury to this organ; for example, a case in which a bullet was lodged in the substance of the heart for twenty years (*Brit. Med. Journ.*, March 23, 1867). The prognosis may be broadly estimated by the severity of the immediate symptoms. Traumatic rupture of the heart is said to have invariably proved fatal.

**TREATMENT.**—In wounds of the heart, the hæmorrhage must be at once arrested by the usual surgical means; immediate death must be prevented by cautious stimulation; and the patient must be kept in such a condition that, whilst life is preserved, the danger of inflammatory reaction in the region of the heart, and of fresh hæmorrhage, is reduced to a minimum. It is on this account that restorative measures are to be guardedly employed at first, and resorted to in the further progress of the case only when urgently indicated. Absolute rest of body and mind is indispensable. Nervine and cardiac sedatives, such as bromide of potassium, morphine, chloral-hydrate, and belladonna, may be of great service when used with judgment.

J. MITCHELL BRUCE.

**HEARTBURN.**—**SYNON.**: Cardialgia.—Heartburn is a hot or scalding sensation, usually referred to the cardiac orifice of the stomach, but in some cases diffused over the whole abdomen. It is a marked symptom of indigestion, and is frequently accompanied by eructations of a very acid character; and the fluid rejected from the stomach produces a sensation of scalding in the throat and œsophagus.

**ÆTIOLOGY.**—Heartburn exists in a very marked degree in dilatation of the stomach, being produced by the decomposition of indigestible food retained in this organ. It constantly accompanies chronic catarrhal gastritis, the retained mucus enclosing particles of food in a state of decomposition,

which set up fermentation in the materials of each meal as soon as it is swallowed. Heartburn is common in the later period of pregnancy, probably because the stomach is so displaced that it is unable properly to expel its contents.

**TREATMENT.**—This sensation is best treated by antacids, such as chalk, magnesia, alkalis, and alkaline waters. Aperients and alteratives may be necessary. In some cases powdered charcoal relieves it. The diet also requires careful regulation. All articles of food containing much fat, sugar, or starch, should be avoided. The use of tobacco should be interdicted; the writer has frequently seen obstinate cases kept up by smoking and chewing. Alcoholic stimulants should be used very sparingly, the least likely to do harm being brandy mixed with Vichy or potass water. The writer has obtained good results by substituting gluten bread for the ordinary baker's bread; and, where this could not be taken, has found advantage from the use of aerated or some other kind of unfermented bread. Some women who suffer severely from heartburn when pregnant find relief from eating lettuce. S. FENWICK.

**HEART-CLOTS.**—See HEART, Thrombosis of.

**HEAT, Ætiology of.**—See DISEASE, Causes of; and the following article.

**HEAT, Effects of Severe or Extreme.**—(A) **Constitutional Effects of Severe Heat acting generally.**—The constitutional or general effects of exposure of the whole body to high temperatures vary with the source and degree of heat, the slowness or rapidity of transition from lower temperatures, and the length of exposure, as well as with the age, constitution, habit and health of the body; and they are liable to be more or less modified or obscured by various concomitant circumstances and conditions, such as the barometric pressure, the hygrometric state, and the purity or impurity of the atmosphere.

The range of temperature within the limits of which life can be maintained appears to be greater in the case of man than in that of most of the lower animals, in virtue of greater power of accommodation to external influences, without undue elevation or lowering of the temperature of the body. But in every case any such combination of external circumstances as causes the temperature of the body to rise  $10^{\circ}$  to  $15^{\circ}$  above the normal standard speedily proves fatal.

1. **Artificial heat.**—Numerous observations and experiments show that in *dry air* exposure to very high temperatures can be borne, during periods varying with circumstances, without danger or even serious inconvenience, the temperature of the body being kept down within safe limits by evapo-

ration from the surface and from the lungs. Thus, in Dobson's experiments, a temperature of  $210^{\circ}$  was sustained during twenty minutes; Blagden exposed himself during eight minutes to a temperature of  $260^{\circ}$ ; Chantrey's workmen were accustomed to enter a drying oven in which the thermometer stood at  $350^{\circ}$ ; and Chabert, the 'Fire King,' is said to have frequently exposed himself to a temperature of from  $400^{\circ}$  to  $600^{\circ}$ . Glass-workers, metal-founders, gas-stokers, engineers in steam-boats—especially in the tropics—bakers' oven-builders, and others constantly carry on their work in temperatures of from  $120^{\circ}$  to  $160^{\circ}$  or even higher—to say nothing of the blasts of radiant heat to which some are from time to time exposed. In *moist air*, evaporation from the surface and its cooling influence being diminished or prevented, much lower temperatures speedily become insupportable. Berger was unable to remain in a vapour bath the temperature of which had risen from  $106^{\circ}$  to  $120^{\circ}$ , although he had easily borne a temperature of  $230^{\circ}$  in dry air for five minutes. When the natural loss of heat is prevented by the moisture of a hot atmosphere—as in a vapour bath, or by immersion in a hot-water bath, or otherwise—the temperature of the body rises. It has been found, taken in the rectum, to rise as high as  $103^{\circ}$  in eight minutes, and even to  $107^{\circ}$  (?) in thirty minutes in a vapour bath of  $127^{\circ}$  (Bartels); and in a hot-water bath of  $104^{\circ}$  to  $111^{\circ}$  the temperature taken in the mouth has been found to rise to  $101.6^{\circ}$  (Mosler).<sup>1</sup>

Dr. Fleming<sup>2</sup> found that exposure in the Turkish bath during an hour to a temperature commencing at  $170^{\circ}$  and gradually lowered to about  $130^{\circ}$ , caused the temperature of his body (taken by a specially devised thermometer in the mouth) to rise rapidly during the first ten minutes from a normal average of  $97.65^{\circ}$  to  $99.2^{\circ}$  (a rise of  $1.55^{\circ}$ ); and then more slowly until the end of fifty minutes, when the highest point,  $101.3^{\circ}$  (a rise of  $3.65^{\circ}$ ) was reached. His pulse rose during the first ten minutes from 78 to 91 beats in the minute, and like the temperature attained its maximum, 115, at the end of fifty minutes. His breathing first diminished in rapidity from 22.5 to 20.8, and then increased to 25.4 in actual rapidity, but maintained a diminished ratio to the pulse. The arterial tension seemed to be increased by the greater rapidity of the heart's action, combined with the gorged state of the capillary circulation. The quantity of material eliminated during the hour amounted to forty-four ounces. The proportion of chlorides in the urine passed after the bath (3.65 in the 1,000) was little

<sup>1</sup> Leichtenstein. *General Balneotherapeutics*. Smith, Elder & Co., 1885.

<sup>2</sup> *Journal of Anatomy and Physiology*, July 1879.

more than half that in the sweat (6.05) collected during the bath, and much less than in the urine previously passed (5.68). The proportion of urea in the urine was slightly increased, and the sweat contained 1.55 in the 1,000.

The effects felt by those exposed to great heat vary with the temperature, the length of exposure, and collateral circumstances, such as impure atmosphere, physical exertion, &c. A sensation of warmth, at first agreeable, is succeeded by one of oppressive and then painful heat, until this is again relieved by the establishment of copious evaporation from the surface. Pleasant stimulation, and some degree of excitement of the nervous and muscular systems, are quickly followed by languor, lassitude, listlessness, feelings of exhaustion, indisposition to mental effort or muscular exertion, dizziness, tendency to sleep, faintness and unconsciousness, sometimes accompanied or preceded by convulsions. If relief is not afforded death ensues. If timely relief is afforded, more or less speedy and complete recovery may be brought about.

If the symptoms have been severe and persistent, or if the sufferer has been exposed to repeated attacks, permanent damage to the health, and especially to the nervous system, almost certainly results, in spite of apparent temporary recovery. In many cases general debility and deterioration, in some cardiac troubles, and in others insanity, have been recorded as the persistent after-effects. Symptoms and conditions closely or exactly resembling those of sunstroke, may be produced by exposure to an artificially heated atmosphere, or to blasts of radiant heat from fires or furnaces. See SUNSTROKE.

Bernard, Delaroché, and others have shown that animals exposed to temperatures of from 130° or lower, to 150° and upwards, quickly die.

The *post-mortem* examination of animals so killed showed that the heart had entirely ceased to beat at the moment of death, and that neither it nor the muscular coat of the intestines could by any means be stimulated to contract again. The muscular fibres of the heart examined microscopically appeared rigid and coagulated (Kühne and Ranvier). Further, the blood in both arteries and veins was dark coloured. *Rigor mortis* set in very early, and general decomposition very speedily commenced. It would thus appear that (apart from sudden shock to the system generally) the cause of death from exposure to high temperatures is to be found in the effect produced on the muscular system of organic life, and especially on the heart. The cardiac myosin coagulates at 115°; and at a temperature considerably short of this its condition must be seriously affected.

ANATOMICAL CHARACTERS. — The *post-mortem* appearances met with in the human

subject in cases in which death has been attributed to the general effects of heat have not been uniform, probably because there has been in most cases simultaneous exposure to other deleterious influences. The following case, however, recorded by C. Speck<sup>1</sup> is noteworthy. A girl, æt. fourteen, the subject of chronic disease of many of her joints, was, on the advice of a quack, wrapped in a sheep-skin warm from the carcass, laid in bed, surrounded with hot loaves fresh from the oven, and covered by the clothes. In about an hour she complained of pains, especially in one arm. She soon fell asleep. It was noticed that her chest rose and fell strongly. She perspired freely, and the sweat was frequently wiped off. She became very pale, and about three hours after the commencement of the treatment she expired, without having recovered, or at any rate manifested consciousness. On *post mortem* examination the next day (the weather being cool and dry) advanced decomposition was found; the blood being black and fluid, and the blood-vessels, cavities, and tissues generally full of gas. The heat of loaves fresh from the oven, such as were put round the child, was found to be 185°.

TREATMENT.—The *immediate treatment* to be adopted in the case of those suffering from exposure of the whole body to heat consists essentially in removal into a cooler and purer atmosphere; quiet rest in the recumbent position; fanning; cool or even cold affusions or sponging, especially over the head and spine—the effect, however, being carefully watched; and the administration of cool or lukewarm fluid in small quantity at a time, with some stimulant. Copious draughts of cold water in a highly heated state of body are liable to give rise to dangerous or even fatal results. Bleeding is not to be recommended.

The *after-treatment* must be conducted on general principles, and determined by the condition of the patient, and the indications afforded in the particular case.

2. Climatic and Solar Heat.—The effects of climatic heat experienced on transition from temperate to tropical regions are as follows: 1st. The average temperature of the body rises .5° to 1° according to Davy and Crombie, or to a somewhat greater extent according to others. The daily fluctuations of bodily temperature in health in India correspond to those in England. The normal temperature of native Indians is about half a degree higher than that of Europeans (Crombie). 2nd. The pulse is quickened according to most observers, but Rattray says this is incorrect. 3rd. The breathing becomes slower and less deep, falling from about sixteen to about twelve or thirteen per minute. Less carbonic acid and less water

<sup>1</sup> *Vierteljahrsschrift für gerichtliche Medicin*, 1874, p. 249.

are thus exhaled. 4th. The skin acts much more freely, its excretion being increased by about 24 per cent. (Rattray). The continued hyperæmia and over-action of the skin, however, are liable to be followed by congestion and obstruction of the sweat follicles, giving rise to 'prickly heat.' 5th. The urine is diminished in quantity, and in amount of urea and chlorides. The experiments of Becher (confirmed by those of Forbes Watson) showed a constant relation between the temperature, and the urea and chloride of sodium got rid of by the kidneys. As the temperature rose from 50° to 70°, an increase was found; but with a further rise from 70° to 90°, an almost equally constant diminution occurred. 6th. The appetite, especially for animal food, is diminished, and the digestive powers seem lowered. 7th. Lassitude, languor, want of vigour, indisposition to exertion, and a sense of exhaustion of mind and body are experienced in degrees varying with circumstances; the depressing effects being most felt when the heat is not only great but continuous day and night, and when the atmosphere is moist.

The effects of the radiant heat of the sun, as distinguished from those of atmospheric heat, are not well made out. It would appear probable, however, that a physiological effect adverse to perspiration is produced by the direct rays (Parkes), and that thus, as well as from rapid evaporation, the skin becomes dry, and liable to certain structural changes, such as the formation of pigment.

Exposure to the direct rays of the sun, or to great or continued heat in the shade, especially under unfavourable atmospheric and general conditions, may give rise to heat-fever, heat-apoplexy, or one or other of the forms of sunstroke. See SUNSTROKE.

(B) **Local Effects of Severe Artificial Heat: Burns and Scalds.**—The local effects of heat vary with the degree, the length of exposure, the medium of application, and the part acted upon. Burns result from 'dry,' Scalds from 'moist' heat.

SIGNS AND SYMPTOMS.—First: a comparatively slight degree of heat causes vascular turgescence, redness, tingling, pain, and tenderness, which soon subside. Desquamation of the epidermis may follow, but no permanent trace of injury is left. Secondly: a higher degree causes severe burning pain, and great redness of surface, followed by effusion of serum beneath the cuticle (vesication). Complete restoration without scar is usually effected. Thirdly: still higher degrees of heat or longer exposure cause intense pain, and immediate destruction, or consecutive destructive inflammation, of the true skin to a greater or less depth. Sloughing and supuration follow, and permanent scarring results. Fourthly: violent heat and prolonged exposure cause complete disintegration and

charring of the structures especially acted upon, followed by destructive inflammation and sloughing of others to a still greater depth and extent. Loss of parts and more or less serious deformity and scarring necessarily result.

The separation of sloughs, and the processes of repair after severe burns, take place slowly; and as a rule the patient suffers much more acutely, and during a much longer period, than after other forms of injury involving equally extensive destruction of tissue.

The *constitutional symptoms and effects* associated with burns and scalds vary with the superficial extent and situation, rather than with the depth of the injury. Thus an extensive burn or scald over the abdomen affecting only the skin is much more likely to prove fatal than a deep burn of one of the extremities, penetrating even to the bone, but of comparatively small superficial area; and burns over the front of the abdomen or chest are more likely to prove fatal than those of similar extent over the back. If more than half the surface of the body is affected the sufferer rarely recovers.

In severe cases death may result from shock to the system, either immediately on receipt of the injury, or after a period of from two or three to forty-eight hours or more. During such time the sufferer remains in a state of collapse or prostration, with pallor of complexion, lowness of temperature (dependent probably on decreased production of heat), coolness of breath, small or imperceptible pulse, dryness of tongue and mouth, and sometimes delirium, rigors, or convulsions. In about half the fatal cases of burns death is attributable to shock. In such cases, *post-mortem* examination shows only congestion of the viscera, and especially of the brain. In some instances, however, characterised by painfully laboured and frequent efforts at respiration, tumultuous, irregular, feeble, and very frequent action of the heart, and great præcordial distress, death would appear to be due to cardiac thrombosis, rather than to simple nervous shock (Brown). In many severe cases the blood has been found on microscopical examination changed in appearance, the red corpuscles being altered in form, or separated into 'numerous little bits' (Ponfick and Schmidt). And it is readily conceivable that such destruction of corpuscles may cause thrombosis and stasis in various organs, especially in the smaller branches of the pulmonary artery (Silbermann), and so give rise to severe symptoms, and even lead on to death.

In about forty-eight hours, more or less, if the immediate effects of the injury have been survived, the stage of reaction and inflammation sets in. The patient revives, and some degree of general pyrexia becomes manifest. The pulse becomes quicker and fuller; the

temperature rises; and the burnt part begins to discharge pus, usually of an offensive odour. Thirst, with dry red tongue, want of appetite, vomiting and constipation, followed by diarrhoea—sometimes with blood in the evacuations—are commonly experienced during the ensuing period; and inflammation of internal parts often occurs, although the special signs and symptoms afforded may be obscure. The pleuræ and lungs, the peritoneum, and the gastro-intestinal mucous membrane (particularly that of the duodenum) are especially liable to be affected. Evidence of inflammation of one or more of these parts, and not uncommonly of ulceration of the duodenum, is afforded on *post-mortem* examination in cases in which death has occurred during this period. It has been suggested that capillary embolism, from the presence of disintegrated blood in the vessels, may cause in some cases the lesions of the internal organs (Brown). During this period, and from the causes indicated, death occurs in about 30 per cent. of the fatal cases.

In the course of about a fortnight or so under appropriate treatment, as a general rule, the acute symptoms will have subsided, the sloughs will have separated, and granulation and suppuration will have become established and healing will gradually take place. But a low form of chronic inflammatory mischief in the internal organs may still be going on, and lead to a fatal result sooner or later; or the patient may sink, worn out by suffering, and exhausted by the profuse discharge from the suppurating surface, or by persistent diarrhoea, accompanied or not by blood in the motions. Sometimes the kidneys are affected, and blood or bloody casts may be found in the urine. Pyæmia, erysipelas, or tetanus may occur and cause death; but there would not appear to be any special liability to these diseases after burns or scalds.

**TREATMENT.**—*Local treatment.*—Slight superficial burns of small extent require little in the way of treatment. Immersion in cold water according to some, in hot water according to others, or exposure before the fire, affords the readiest means of obtaining immediate relief. Afterwards the part may be covered with flour, starch, oxide of zinc, bismuth, or collodion, and wrapped round with cotton-wool to protect it from the air and from accidental irritation. In cases in which there is vesication, the blisters should be pricked, the serum evacuated, and the cuticle left to form a natural protective covering, which may be advantageously strengthened and kept in position by a layer of collodion. But the punctures should be left open. Lint soaked in oil, or smeared with vaseline or some such material, should be applied, and the whole covered with cotton-wool. A mixture of chalk or

whitening and vinegar, of the consistence of thick cream, is said to form an excellent application in such cases, speedily relieving pain, and helping to constitute a good protective covering.

Severe and extensive burns and scalds demand in their treatment the most careful management, and the greatest possible patience, gentleness, and firmness; for even if life be preserved, the most pitiable disfigurements and deformities are liable to result from cicatricial contractions, unless proper preventive measures are perseveringly carried out.

So soon as may be after the injury, the clothes should be removed from the patient with the greatest care—being cut away piecemeal if needful, and not removed in such way as to tear off epidermis or scorched or charred parts. The whole burnt surface, having been cleansed as seems needful by some antiseptic wash or spray, should then be covered as quickly as possible with the dressing considered best, and enveloped in thick layers of antiseptic cotton-wool lightly bandaged on. For cleansing wash or spray solutions of borax or carbolic acid (weak), with the addition or previous application of cocaine, are perhaps best. Different dressings have been advocated from time to time. One seems to suit best in some cases, another in others. But antiseptics should always be used. Among the applications variously recommended may be mentioned the old-fashioned favourite in hospital practice—Carron oil, a mixture in equal parts of linseed oil and lime water. Almond oil or olive oil may be substituted for the linseed oil, and iodoform or a little carbolic acid added with advantage. White lead, putty made thin by addition of oil, calamine ointment, carbolised oil, solution of carbonate of sodium, carbolic lotion, flour, and starch, are among the other materials that have been recommended. Among the best probably may be recommended a combination of boric acid and iodoform with vaseline, or glycerine and oil, as a basis. Whatever the material selected, it should be slightly warmed, and applied very thickly spread on separate strips of lint in such way as to facilitate future dressing bit by bit, and so as to avoid extensive exposure of raw surface. The first dressing should be allowed to remain undisturbed as long as possible—until, indeed, the offensiveness of the discharge, or the discomfort of the patient indicates the necessity for its removal. The earlier dressings, however gently carried out, occasion so much suffering to the patient that—in the case of children especially—it is often desirable to administer chloroform or to spray the parts with a 5 per cent. solution of cocaine. Poultices are sometimes useful in aiding the separation of sloughs. In such case boracic lint soaked with warm

water forms an excellent substitute for the old linseed-meal poultice. Any needful washing or cleansing is best done by aid of the steam spray-producer, a weak solution of carbolic acid or of borax being used. When suppuration is established, and the surface clean, the application may be varied according to the indications afforded. Calamine or zinc or lead ointment, with or without the addition of some anodyne; and lotions of lead, morphine, and glycerine, or of sulphate of zinc, are amongst those commonly employed. Iodoform, with extract of hemlock and spermaceti ointment, has been strongly recommended as tending to soothe pain, to deodorise the discharge, and favour healing. Exuberant granulations may be treated with nitrate of silver in solution, or by the application of the solid stick. When the granulating surface is in a healthy state, cicatrisation may be very materially expedited by skin-grafting; and in cases in which there is extensive healthy granulating surface, by transplantation of large portions of healthy skin from some adjoining part.

During cicatrisation, and even for some time afterwards, it is of the greatest importance to keep all parts in such position as that there shall be as little deformity as possible from growing together of surfaces, and contraction of scars. This is to be effected or attempted by position, by mechanical apparatus, and by the application of strips of adhesive plaster and bandages in manner determined by the circumstances and conditions in each particular case.

*Constitutional treatment.*—In the early stages, alcoholic stimulants, or ammonia, and external warmth are especially requisite, and such light nourishment as can be taken. Opiates or other anodynes, as chloral or bromide of potassium, are to be administered according to the indications afforded, for the purpose of allaying pain and soothing the nervous system. When suppuration is established, a full allowance of good nourishing food, with some alcoholic stimulant, should be given, and such tonics as seem most suitable. Small doses of opium at regular intervals often prove very beneficial. The complications that may arise, such as affections of the internal organs, &c., must be treated in accordance with general principles, but all depressing medicines as a rule should be avoided, and if there should be indications of intestinal inflammation great care should be exercised in the administration of aperients.

(C) **Local Effects of Severe Solar Heat: Sunburn.** See SUNBURN.

ARTHUR E. DURHAM.

**HEAT, Therapeutics of.**—PRINCIPLES. The primary effect of external heat applied *locally* to the animal body is that of an excitant or stimulant. There occurs redness,

with turgescence of the small vessels, in the part to which the heat is applied, along with slight augmentation of temperature, and pain. Increased beyond a certain degree, heat ceases to be a stimulant, its prolonged action causing greater pain, exhaustion, and depression; and, if the action be very intense, it becomes an escharotic.

Experiments have shown that by increased heat the electric currents in the nerves are destroyed. It may therefore be assumed that the nerves become less able to conduct impressions either to or from the brain, and that heat may act as a sedative to painful nerves.

The action of medicines on the system appears to be influenced by temperature. Ringer found that a small dose of veratrine greatly affects the ventricle at a high temperature, but at a low one produces no effect. Brunton and Cash found that the roots of the vagus in the medulla are paralysed by a high temperature. This may explain the observation of Thomas as to the failure of digitalis to act on the pulse in pneumonia with high temperature.

Heat applied generally—that is, to the whole body—produces a number of important physiological effects which are described in the preceding article; and in that on BATHS.

**APPLICATIONS AND USES.**—Heat is employed in the treatment of disease as a general or local stimulant, a local depressant, a caustic, or a counter-irritant; and that in the form either of *dry* or of *moist* heat.

*Dry heat.*—The primary exciting and stimulating action of heat may be made available to rouse the nervous and vascular systems. The use of the hot-air bath (Turkish bath) and that of the sand bath are discussed in the article on BATHS. In some parts of the South of France, baths of hot sand (*arena calida*) are used in the treatment of rheumatism, paralysis, and spasm; the sand acting as a stimulant and sudorific. Bottles of hot water are placed in the axilla, and against the feet and thighs, to restore the circulation, in cases of collapse of the system with coldness of the extremities and great failure of circulation, as in the treatment of collapse from the shock of an injury, or from such diseases as cholera, or of the apparently drowned.

Dry heat may also be applied to the abdomen, in the form of tins or bottles of hot water, or bags of heated salt or sand, to relieve painful spasm and colic. Hot water enclosed in an indiarubber bag is sometimes of service to allay undue irritability of the spinal nerves. The therapeutical application of heat as a counter-irritant will be found described elsewhere. See COUNTER-IRRITANTS.

*Moist heat.*—Heat and moisture together tend to cause relaxation of the tissues, thus removing the tension and pain due to inflam-

mation. Moist heat is employed locally in the form of the local vapour bath, fomentations, and poultices. See FOMENTATIONS; and POULTICES.

Moist heat is applied to the surface of the body generally, chiefly in the form of the vapour bath. See BATHS.

JOHN C. THOROWGOOD.

**HEAT-STROKE.**—A synonym for sunstroke. See SUNSTROKE.

**HECTIC FEVER** (ἐκτικός, habitual).—  
SYNON.: Fr. *Hectique*; Ger. *Hectisch*.

**ÆTIOLGY.**—The variety of fever thus named has long received special recognition, inasmuch as it presents certain prominent and peculiar features, as regards its course and attendant phenomena. It usually occurs in association with some wasting and exhausting disease, especially when this is accompanied by a profuse and constant drain from the system, and more particularly when there is chronic suppuration, with an abundant discharge of pus. Hectic fever is most frequently noticed in cases of pulmonary phthisis, in a large proportion of which it appears in various degrees during some part of their course, chiefly in the advanced stages. Other conditions deserving of mention in connexion with which it may supervene are empyema, especially if there is an external fistula, tubercular ulceration of the intestines, chronic purulent discharge from the kidneys, hepatic abscess, chronic dysentery, and any form of external chronic abscess attended with an abundant formation of pus. Fever of a hectic type sometimes occurs in cases of acute inflammation; and it is occasionally observed in chronic affections unattended with suppuration, such as cancer and lymphadenoma.

**SYMPTOMS.**—Hectic fever is usually established gradually, becoming more and more distinct, until it assumes its typical characters. It is more or less paroxysmal, being at first indicated by slight pyrexia towards evening and during the night, the temperature being a little raised, and the pulse hurried. During the day there is no fever at this time, but as the case progresses it becomes constant, though exacerbations occur at night, and, it may be, also in the morning, the paroxysms thus occurring either once or twice within the twenty-four hours, and the pyrexia being remittent. In typical hectic there is a complete febrile cycle, beginning with chills or even a distinct rigor, followed by considerable heat of skin, the temperature continuing to rise, and ending in more or less profuse perspiration, especially about the head and chest, sometimes so abundant as to saturate the bedclothes or even the bedding. Patients often feel subjectively very hot, the palms of the hands and soles of the feet having a burning sensation. The pulse tends to become very frequent and quick, and is easily

hurried and excited, being at the same time weak, soft, and compressible. Not uncommonly a bright red or pink spot appears on each cheek during the paroxysm, known as the *hectic flush*, and this may contrast markedly with the general pallor of the face, the eyes being also bright, clear, and sparkling. The mind is unaffected, and the mental faculties may be unusually cheerful and vivid. After a febrile exacerbation the urine may present excess of lithates. Hectic fever does not always show all its typical features, and even in the same case variations are noticed in the precise characters of the paroxysms. It is usually accompanied with other symptoms due to the disease with which it is associated; while it itself tends to cause wasting and debility, as well as a sense of much exhaustion after each attack. In most instances a fatal termination ultimately ensues, but if the condition upon which hectic fever depends is curable, recovery will generally take place.

**TREATMENT.**—The first principle in the treatment of hectic fever is to attend to, and cure, if possible, the condition with which it is associated, and especially to diminish or check suppuration, to prevent retention of unhealthy pus, or to employ antiseptic applications. General tonic treatment will also help in preventing the attacks. These may be directly checked in appropriate cases by full doses of quinine, salicine, salicylates, or other antipyretics, given before the usual time for their occurrence. Sponging the skin freely may also prove of service in some instances. The treatment of hectic in phthisis runs into that of night-sweats, and can be more conveniently discussed under that disease. See FEVER; and PHTHISIS.

FREDERICK T. ROBERTS.

**HELIOSIS** (ἡλιώω, I expose to the sun). A method of treatment for certain diseases, which consists in exposing the patient to the rays of the sun. The term is also employed as a synonym for sunstroke. See SUNSTROKE.

**HELMINTHIASIS** (ἐλμύς, a worm). The condition of system upon which the development of worms in any part of the body depends. The term is also applied to diseases characterised by the presence of worms.

**HELMINTHICS** (ἐλμύς, a worm).—Of or belonging to worms. A synonym for anthelmintics. See ANTHELMINTICS.

**HELMINTHS** (ἐλμύς, a worm).—This term is often employed in preference to one or other of the various synonyms with which it is regarded as equivalent (worms, intestinal worms, vermes, entozoa, internal parasites, and so forth). Thus, von Siebold (who speaks of the Helminths as forming a *class* of animals, nearly all of whose members are

parasitic) admits that the only character common to the greater part of the whole group is their peculiar mode of life. The study of the helminths forms what is often called the science of *Helminthology*. It has been thought desirable, in the present work, to speak of the helminths as constituting part of the class ENTOMOZOA, under which heading, therefore, more detailed references are given.

T. S. COBBOLD.

**HELOUAN, near Cairo, Egypt.**—Thermal sulphurous muriated saline waters. See MINERAL WATERS.

**HEMERALOPIA.**—DEFINITION.—Day blindness: a disorder of vision in which objects cannot be seen well or comfortably in ordinary daylight or by strong artificial light, but are seen more clearly or more comfortably in shade or in twilight.

Most modern authors use the term in the opposite sense of *night blindness*, regarding it as derived from *ἡμέρα*, the *day*, and *ὄψ*, the *eye* or *sight*. In this work it has, from the first, been used in the sense given above, the *al* being regarded as representing *alpha privativa*, or as a contraction of *ἀλαός*, *blind*. This meaning has since been adopted in the *Nomenclature of Diseases* of the Royal College of Physicians. The word 'hemeralopia' does not occur in any of the genuine ancient Greek or Latin medical writings. It is once mentioned, without being defined, in the pseudo-Galenic treatise, *Introductio seu Medicus* (ed. Kühn, xiv. 768). It has, however, long been used as the antonym of *nyctalopia*. See NYCTALOPIA.

**CAUSES.**—Day blindness is a symptom rather than an idiopathic affection, and is sometimes the effect of mere intolerance of light (photophobia), dependent upon irritation or inflammation of the cornea, &c. The various causes may be grouped as follows:—

1. Opacity in some of the refractive media, such as central nebula or leucoma of the cornea, or circumscribed opacities occupying the pupillary region of the lens or the vitreous. In such cases, vision is obscured when the pupil is contracted, but becomes clearer when the pupil dilates in shade.
2. Irritation or inflammation of the parts of the eye innervated by the sensory fibres of the fifth nerve—the lids, the conjunctiva, the cornea, or the iris; neuropathic affections of these textures; and reflex irritation from nose, teeth, uterus, &c.
3. Hyperæsthesia of the retina, which may be due to reflex irritation, or (rarely) to inflammation of the retina, the optic nerve, or the choroid, to insufficient pigmentation of the ocular fundus as in albinism, to dazzling from coloboma of the iris or preternatural dilatation of the iris from any cause, to exposure to bright lights or glare of any kind, to prolonged seclusion in darkness or dimly lighted places.

A transient physiological form of hemeralopia occurs whenever the eyes suddenly pass from darkness into bright light.

**TREATMENT.**—The treatment of hemeralopia should follow the special indications. Impairment of sight due to central opacity in any of the refractive media may often be relieved by dilatation of the pupil with a weak solution (gr.  $\frac{2}{10}$  to gr.  $\frac{1}{2}$  to an ounce) of atropine or other mydriatic, or by making an artificial pupil. Inflammations of the various textures of the eye and reflex irritations are to be treated by appropriate means. The eyes should be protected by a shade or by neutral-tinted glasses. The general treatment must be adapted to the associated local and constitutional conditions.

JOHN TWEEDY.

**HEMIANÆSTHESIA** (*ἡμι-*, half; *ἀ*, priv.; and *αἰσθάνομαι*, I feel).—Paralysis of sensation, affecting one side of the body. See SENSATION, Disorders of.

**HEMIANALGESIA** (*ἡμι-*, half; *ἀ*, priv.; and *ἄλγος*, pain).—Insensibility to painful impressions, affecting one side of the body. See SENSATION, Disorders of.

**HEMIANOPIA** (*ἡμι-*, half; *ἀ*, priv.; and *ὄψ*, sight).—The term 'hemioopia' is now generally taken to mean loss of perceptive power in one lateral or vertical half of the *retina*; while by hemianopia is understood obscuration of vision on one lateral or vertical half of the *visual field*. Thus, owing to the fact that rays of light cross within the eye, right lateral hemianopia is produced where left lateral hemioopia exists, as a result of intracranial disease of one or other kind.

It is well known that in the lower vertebrate animals the optic nerves undergo a complete decussation at the optic chiasma, so that all impressions from each eye go to the opposite optic lobe. Some believe, even to the present day, that a similarly complete decussation of the optic nerve-fibres takes place at the chiasma in man; others (constituting a large majority) altogether reject this notion, and adopt the view originally propounded by Newton and reinforced by Wollaston, that at the optic chiasma there occurs a semi-decussation only. Neither view can be said to be definitely proved to the exclusion of the other; and it seems quite possible that these discrepancies in doctrine may be in part dependent upon the fact that the course of the optic fibres is not the same in all men. There may, in fact, be variations in the degree of completeness of the decussation of the optic nerve-fibres at the chiasma, just as, from the researches of Flechsig, we now know that not infrequently considerable variations occur in the amount of decussation of motor fibres in the anterior pyramids of the medulla.

The effects resulting from congenital or

early blindness of one eye supply us with some information bearing upon this point. Where a semi-decussation exists, the effects of the atrophy of the optic fibres proceeding from the affected eye should, beyond the chiasma, be pretty equally distributed, so that, though in such a case the two optic nerves will be very dissimilar in size, the optic tracts ought to be of equal bulk; and this is what is most commonly met with. On the other hand, it is undoubtedly the fact that in some of such cases the optic tract on the opposite side is found to be notably atrophied, while that on the same side as the atrophied optic nerve presents a healthy appearance—signs which betoken a pretty complete decussation of the optic fibres at the chiasma.

*Different forms of Hemianopia and their causes.*—In the cases where the common arrangement obtains—that is, the semi-decussation—it seems to be the inner fibres that cross one another, whilst the outer fibres of each nerve are continued on into the outer part of the corresponding optic tract. The result of this arrangement would be that the fibres from the right half of each retina would proceed to the right optic tract, while those from the left side of each retina would go to the left optic tract; so that if the image of any object should fall simultaneously either on the right side of the two retinae, or on the left side of the two retinae, the impressions in either case will be transmitted along one and the same tractus opticus—the right or the left, as the case may be. Therefore it seems clear that, in all cases in which the semi-decussation of the optic fibres obtains, any intracranial lesion which either destroys, seriously presses upon, or disturbs the nutrition of the optic tract on one side, will give rise to an opposite *lateral hemianopia*. If the decussation had chanced to be total there would, on the contrary, have been produced a ‘crossed amblyopia.’ The optic tract is apt to be involved in some part of its course between the chiasma and the geniculate bodies by different kinds of lesions. Thus, we may have tumours originating in parts of the tract itself; or the tract may become involved secondarily, or pressed upon, by some tumour or hæmorrhage occurring in the crus cerebri or in the posterior tubercle (pulvinar) of the thalamus, or by a tumour originating in the inner and under part of the temporal lobe, or from the bone itself in the middle fossa of the skull.

Two other, much rarer, forms of hemianopia require to be mentioned, which are produced by lesions affecting different parts of the chiasma. One of these is double *temporal hemianopia*, which may be produced by a lesion pressing upon the anterior median portion of the chiasma. Any lesion capable of doing this would probably be a tumour, and it would produce the same effects whether there is a total or a semi-decussation in the

chiasma. The lesion would involve the decussating fibres that come from the inner half of the retina on the two sides, and thus would lead to double temporal hemianopia. The other form is known as *nasal hemianopia*, and where semi-decussation of the optic fibres exists it would be produced by a tumour or an aneurysm causing an injurious amount of pressure upon either outer angle of the chiasma, since there would then be paralysis of the fibres emanating from the outer part of the retina, and consequently the suppression of the inner part of the visual field of the corresponding eye. It is only with extreme rarity that this form of hemianopia is double.

We have now to turn to another side of the question. It is natural to ask whether the lateral hemianopia that develops as a consequence of disease of the optic tract will also result from a lesion which involves the optic fibres beyond the corpora geniculata—that is, in their deep intracerebral course. This was the view put forward by von Graefe in 1860, and which was very commonly accepted till it was challenged by Charcot some few years since. Relying apparently upon the paucity of cases on record in which lateral hemianopia had resulted from lesions of the cerebral hemispheres themselves, and upon the many cases that had come under his own observation in which ‘crossed amblyopia’ had been produced by functional or structural lesions involving the posterior or sensory segment of the internal capsule, he came to the theoretical conclusion that all the optic nerve-fibres from each eye went to the opposite cerebral hemisphere, owing to the supposed fact that those fibres which had not decussated at the chiasma did decussate somewhere in the corpora quadrigemina. These views he embodied in a well-known diagram. The subsequent progress of knowledge has, however, not been compatible with this theory. Thus Munk’s original statement has been confirmed by Schäfer and S. Brown, to the effect that removal of one occipital lobe in the monkey produces, not crossed amblyopia, but opposite lateral hemianopia (*Brain*, Jan. 1888, p. 367). Again, Seguin, in a valuable memoir (*Journ. of Nerv. and Ment. Dis.*, vol. xiii, Jan. 1886), has shown that opposite lateral hemianopia is more especially produced by lesions in that inner portion of the occipital lobe known as the ‘cuneus,’ a region supplied by a rather large branch of the posterior cerebral artery which passes along the calcarine fissure. Still it would appear that lesions anywhere in the posterior parietal, temporal, or occipital region of the hemisphere, when they implicate the ‘optic radiations’ of Gratiolet (*i.e.* fibres passing backwards from the corpora quadrigemina and geniculata to the occipital cortex), are also capable of producing an opposite hemianopia in man of a permanent character. In the lower animals (monkey,

dog, &c.), for the production of a similar defect the removal of the occipital lobe must be complete, and even then the defect is of a comparatively brief duration—two or three weeks at most.

There is at present an apparent contradiction between these well-attested facts, as to the production of lateral hemianopia from disease of the occipital lobe (implying as they do that the semi-decussation persists even to the cortical termini of the optic fibres), and the fact of the existence of a 'crossed amblyopia' in association with functional or structural disease of the hinder portion of the internal capsule. A solution of this difficulty cannot as yet be readily found. Ferrier still maintains that crossed amblyopia is produced by destruction of one angular gyrus. He asserts, indeed, that 'the angular gyri are more particularly the centres for clear vision, each mainly for the eye of the opposite side' (*Brit. Med. Journ.*, June 21, 1890, p. 1415).

DIAGNOSIS.—Under this head reference has to be made to different sides of the problem. The first point to be considered is the mode of recognition of the different varieties of hemianopia. In some cases, where the defect is well marked, and the patient is intelligent, he will himself call the observer's attention to it; very frequently, however, such defects are only discovered by being specially looked for. This subject will be referred to under Vision, Defects of. Here it must suffice to say that for all accurate observation an instrument named a 'perimeter' is required, by the aid of which the exact nature and extent of the limitation of the visual field can be discovered; but, for a first rough examination, the following simple method will suffice for the detection of any great narrowing of the visual field in particular directions. The patient stands with his back to the light, and, covering one eye, looks steadily from a distance of eighteen inches at the nose or eye of the observer, who then moves his forefinger about in different parts of the field of vision, and notes any part where the patient says it is invisible or badly seen. Anything like a hemianopic defect, of either variety, may be easily appreciated in this manner.

The fact of the existence of hemianopia having thus been established, we must next seek to determine the site of the lesion by which it has been caused. The problems of regional and of pathological diagnosis are in these cases often intimately related to one another. With respect to the latter problem some hints have already been given.

For purposes of regional diagnosis the first thing is to determine whether the case before us belongs to one or the other of two main categories into which cases of hemianopia are divisible—that is, whether it is a case of *tract* or of *central hemianopia*; and this may be determined by a delicate test first used by

Wernicke. This test is based upon the fact that, in the case of lesions in the primary optic centres of one side (the corpus geniculatum and one anterior quadrigeminal body), or of their afferent fibres, there is an interference not only with visual impressions, but also with the path for impressions that excite pupillary contractions through these oculo-motor centres; whereas in the case of deeper lesions, situated in parts of the cerebral hemisphere involving either the optic radiations or the occipital cortex, the pupillary reflex would not be at all interfered with. Thus in cases of 'tract hemianopia' there exists what has been termed hemiopic pupillary inaction; while in cases of 'central hemianopia' this sign does not exist. The sign itself is searched for by throwing a pencil of light obliquely (at an angle of 40°–60°), by means of an ophthalmoscope mirror, on to the blind side of the retina, so as to see whether contraction of the pupil is, or is not, produced. If carefully performed, no reaction of the pupil occurs in cases of *tract hemianopia*—that is, where the defect is due either to disease of the afferent fibres, or of the primary optic centre itself. But where the hemianopia is caused by disease in the cerebral hemisphere itself (*central variety*) the contraction of the pupil occurs equally well whether the light be thrown upon the blind or upon the unaffected half of the retina.

From the point of view of a regional diagnosis, we may enumerate four fairly marked types of *central hemianopia*.

1. There are cases in which hemianopia exists alone, or as the most prominent symptom, or in association with certain general signs indicative of the existence of tumor cerebri. Such are the cases where the occipital lobe is affected in or about the region of the cuneus.

2. Cases in which hemianopia coexists with hemianæsthesia and choreiform or ataxic movements of one half of the body, without marked hemiplegia. These are probably due to a lesion of the caudo-lateral part of the thalamus, or of the caudal division of the internal capsule, on the side of the brain opposite to the dark half-fields and the hemianæsthesia.

3. Cases of hemianopia with complete hemiplegia and hemianæsthesia are probably caused by an extensive lesion of the internal capsule in its knee and caudal part.

4. Cases of hemianopia with aphasia and hemiplegia, with or without hemianæsthesia, are probably due to an extensive superficial lesion of the area supplied by the left middle cerebral artery, such as may be brought about by its plugging either from thrombosis or embolism.

In all these cases the pupillary reaction would not be interfered with, and there would be no atrophy of the optic nerves unless as a

sequence of choked disc. In addition to these forms, five types of *tract hemianopia* may be enumerated.

1. The first type of tract hemianopia would be caused by lesions in the primary optic centres on one side (the corpus geniculatum and one anterior quadrigeminal body). Little or nothing is known of lesions thus limited, which are excessively rare. Still, such parts may be involved, primarily or secondarily, by a tumour; when amongst a varying group of symptoms we might have, as results of the lesion in the parts specified, lateral hemianopia, hemiopic pupillary inaction, together with early atrophy of the optic nerves.

2. In other cases where disease involves the optic tract and the crus cerebri, we may have hemianopia coexisting with some form of cross-paralysis. A probable combination would be paralysis of the third, fourth, and sixth cranial nerves on the side of the lesion (some one or all of these nerves), together with partial hemiplegia (without anaesthesia), and hemianopia on the opposite side. In some cases choked discs would appear early, without interfering with the hemiopic symptoms until optic atrophy with complete blindness had become established.

3. In cases where the optic tract is involved more anteriorly, other symptoms coexisting with the hemianopia may be very vague and uncertain; but the hemiopic pupillary inaction should be marked, and there should be early partial atrophy of the optic nerves. These latter signs suffice to distinguish this group of cases from those belonging to the first category of 'central hemianopia,' with which otherwise they might be confounded.

4. Cases of bitemporal hemianopia due to lesions pressing upon the anterior part of the chiasma.

5. Cases of nasal hemianopia, mostly single, due to a lesion involving the outer part of the chiasma.

The only other forms of monocular hemianopia are also very rare, and are due to lesions of the optic nerve itself in front of the chiasma.

Other very rare forms of hemianopia affect the superior or inferior, rather than the lateral, segments of the field of vision. As Seguin points out, when these forms exist with a very irregular demarcation line, they are probably still more peripheral in their origin and due to embolism or thrombosis of the central artery of the retina.

In another class of cases, instead of a definite hemiopic defect of sight, we meet with partial, quadrant, or sector-like defects in the upper or lower halves of the visual fields. Little or nothing is known as to the causation of such defects. They might conceivably be caused by disease in certain limited portions of the occipital cortex, or of some only of the fibres constituting the

optic radiations. Again, they might be due to a localised damage to some of the fibres of one crus or one optic nerve; or to degeneration of the retina caused by the plugging of some one branch of its central artery.

PROGNOSIS AND TREATMENT. — Nothing special can be said under these heads concerning the hemianopia itself. Both the prognosis and the treatment of this defect are absolutely dependent upon the causative conditions, and the nature and severity of the collateral symptoms with which it is associated. We must, in fact, always seek to mitigate or remove the underlying causal conditions, and upon our chance of being able to do this the prognosis in any given case entirely depends.

H. CHARLTON BASTIAN.

**HEMICRANIA** (*ἡμι-*, half; and *κράνιον*, the head).—Pain limited to one side of the head. The term is, however, generally used as synonymous with *megrim*. See *MEGRIM*.

**HEMIOPIA** } (*ἡμι-*, half; and *ὤψ*,  
**HEMIOPSIA** } sight).—A loss of perceptive power in one lateral or vertical half of the retina. See *HEMIANOPIA*; and *VISION*, Disorders of.

**HEMIPLÉGIA** (*ἡμι-*, half; and *πλήσσω*, I strike).—Paralysis of motion of one side of the body; sometimes applied to loss both of motion and of sensation. See *PARALYSIS*.

**HEPATALGIA** (*ἥπαρ*, the liver; and *ἄλγος*, pain).—Strictly this word signifies pain in connexion with the liver. It has, however, been specially applied to a supposed neuralgic pain referred to this organ, coming on in paroxysms, and said to be of a severe character in some instances, so as to simulate hepatic colic. Whether there is any such affection is exceedingly doubtful; and probably in cases of supposed hepatalgia the neuralgia is either superficial, or there is some tangible but undiscovered cause for the pain, connected with the hepatic apparatus or some neighbouring structure.

FREDERICK T. ROBERTS.

**HEPATISATION** (*ἥπαρ*, the liver).—A term applied to the condition produced by acute inflammation of the lung, in which the pulmonary substance becomes solid and friable, resembling somewhat the liver in its physical characters. See *LUNGS*, Inflammation of.

**HEPATITIS** (*ἥπαρ*, the liver).—Inflammation of the liver. See *LIVER*, Inflammation of.

**HEPATOCELE** (*ἥπαρ*, the liver; and *κήλη*, a tumour).—Hernia of the liver. See *LIVER*, Displacements of.

**HERCULESBAD (HERCULES-FURDO), in Hungary.**—Thermal sulphur and muriated saline waters. See MINERAL WATERS.

**HEREDITY.**—By heredity is meant that special property through which the characters and qualities of organisms are transmitted to their descendants throughout successive generations, so that the offspring in their main features resemble their parents. The transmission of parental characters to offspring is a subject for scientific inquiry. It has long been known that a young organism is derived from an ovum formed within the body of the female parent, so that a descent from the mother and a direct continuity through the egg with her structure was apparent. But it is within a comparatively recent period that the direct participation of the male parent in the production of the offspring has been traced. The older writers thought that the semen evolved a vapour or spirit, which influenced the ovum, or that the fluid part of the semen penetrated the vitelline membrane, and induced germinative changes, which resulted in the production of a young organism, without the male contributing any definite structure in the process. The discovery, in 1842, by Dr. Martin Barry, of the penetration of the vitelline membrane by the spermatozoon put the subject on a more definite basis, and the researches of Bütschli, Auerbach, Fol, Strasburger, Flemming, Hertwig, E. van Beneden, and others, have given greater precision and completeness to our conceptions of the process of impregnation. It has now been demonstrated that the young organism arises within the egg by the incorporation, or conjugation, of an extremely minute particle derived from the male parent with an almost equally minute particle derived from the female parent. The particle derived from the male is the head of the spermatozoon, which, after it has penetrated within the ovum, is called the male pronucleus. The particle derived from the female is a portion of the germinal vesicle, which has remained included within the egg, and is called the female pronucleus. The male and female pronuclei gradually approximate, touch, and then become incorporated with each other, and form what is called the segmentation-nucleus. The segmentation-nucleus consists of chromatin fibres and nucleoplasm, which are derived both from the nucleus of a male sperm cell, *i.e.* the head of a spermatozoon, and from the nucleus of a female germ cell, *i.e.* the germinal vesicle. The segmentation-nucleus is therefore a composite or hermaphrodite nucleus, and consists of matter derived from both parents. This composite nucleus then rapidly divides and subdivides within the ovum, so as to produce daughter-nuclei, and the protoplasm of the yolk undergoes a corresponding

process of division. Each of the daughter-nuclei becomes surrounded by a layer of protoplasm, and numerous new cells are produced. The nucleus of each of these new cells contains chromatin fibres derived from the segmentation-nucleus, and through it from the corresponding fibres of the nucleus of the male sperm cell and of the female germ cell. It is believed that the male and female chromatin fibres enter in equal proportions into the structure of the nuclei derived by division from the segmentation-nucleus, so that each of the nuclei of the new cells, like the segmentation-nucleus, is composite or hermaphrodite, *i.e.* represents both parents. The cells produced by the division of the segmentation-nucleus and its envelope of yolk-protoplasm form the blastodermic membrane. In man and the higher organisms this membrane consists of three layers of cells, named epiblast, mesoblast, and hypoblast. From these layers, by a continuous process of division and subdivision of the nuclei of their constituent cells, accompanied by a differentiation of the protoplasm, all the tissues and organs of the body are descended. As the nuclei of all the cells in the body are derived by continuous descent from the nucleated cells of the blastoderm, and as these again are the descendants of the segmentation-nucleus, one may infer that the nucleus of each cell is composite or hermaphrodite and represents both parents.

The changes which take place in a nucleus during its division constitute the process of *karyokinesis* or *karyomitosis*. They consist at first in an enlargement and rearrangement of the chromatin fibres of the nucleus, and then in a splitting of each fibre into two daughter-threads, so that each daughter-thread divides itself equally between the two daughter-nuclei which are formed by the splitting of the parent nucleus. As this process goes on throughout the whole period of cell-multiplication, there is an actual transmission of structure from the segmentation-nucleus to the nuclei of all the cells derived both directly and indirectly from it. Now, as this descent in structure is in all probability associated with a power of transmitting properties, and as the segmentation-nucleus consists of material derived from both parents, a structural medium is provided for the transmission of the characters of the parents to their offspring, and the phenomena of heredity may be said to rest on a physical basis.

The material which forms the structure of the male and female pronuclei and the segmentation-nucleus has been termed by Weismann the *germ-plasm*. He believes that in each individual produced by sexual generation, a portion of the germ-plasm derived from both parents is not employed in the construction of the nuclei of the cells and tissues of the soma or personal structure of the individual, but is set aside unchanged for the formation

of the germ cells of the succeeding generation. According to this theory there is a direct continuity of germ-plasm from one generation to another, which acts as the conveyor of hereditary characters. Weismann maintains that the germ-plasm is not modified by the habits and mode of life of the individual, and he conceives it to be transmitted from one generation to another uninfluenced by the conditions of life in which the individual is placed. He assumes that the reproductive cells which contain the germ-plasm are not acted on by the organs and tissues of the body in which they are situated, except in so far as they may be affected by general conditions of growth and nutrition. Hence, he concludes that characters acquired by individuals during their lifetime from any external cause, which modify the persona or soma, cannot be transmitted to their offspring.

Several arguments may, however, be advanced in opposition to Weismann's conclusion, which seems to require for its confirmation that the germ-plasm of the reproductive cells should, from the commencement of the development of the embryo, be isolated from the cells from which the other organs of the body are derived. But both reproductive cells and soma cells are formed in the fertilised ovum by the segmentation of the nucleus, and the reproductive cells in man and other vertebrates do not appear as isolated organs until after the rudiments of all the great organic systems have been mapped out. Hence, before they are set aside as germ cells or sperm cells, the germ-plasm has apparently been in a stage of diffusion, and under precisely the same influences as those which in the embryo affect the formative cells of the body generally, so that the conditions which would secure the germ-plasm and the soma cells from mutual interaction are not complied with.

Further, there is experimental evidence to show that in placental mammals an interchange of material takes place during gestation in opposite directions from fetus to mother, as well as from mother to fetus. A character derived by a fetus by descent from its male parent may thus be acquired by the mother from the foetus, and influence the germ-plasm of her reproductive cells so as to be transmitted to her future offspring, even though they may not have the same male parent. Hence, it is an axiom with those engaged in the breeding of particular kinds of stock, that, if they wish to keep the strain pure, the mother must not at any time be allowed to beget offspring by sires of another blood.

The hereditary transmission of characters from parents to offspring is not limited to such as are normal and physiological, and of service in the perpetuation of the species and of the race. Characters which are of no

service, and, indeed, are detrimental to the individuals in whom they occur, may also be transmitted, and it is from this point of view that the subject of HEREDITY requires to be considered in this place. The study of those modifications in the structure of the body, which pathologists group together under the name of Congenital Malformations, has furnished numerous illustrations of the descent in families of variations from the normal structure. Familiar examples are furnished in the presence of supernumerary digits on the hands or feet in some families, or a diminution in their number in others. But modifications or variations in structure that can be transmitted from parents to offspring are by no means limited to changes which can be recognised by the naked eye. They are sometimes so minute as to be determined rather by the modifications which they occasion in the functions of an organ than by the ready recognition of structural variations. One might adduce as an example Daltonism, or colour-blindness, which has distinctly been shown to be hereditary, and which is due apparently to a minute structural defect in the development of the retina, or the optic nerve, or the brain itself, occurring in particular families.

But there are certain diseases which occur in some families more than in others, and which are regarded as hereditary. Some of these are discussed in the article PREDISPOSITION TO DISEASE, but in addition to the examples there given, it may be stated that in some families there is a strong tendency to the production of cataract in successive generations, and in other families an equally strong predisposition to the hæmorrhagic diathesis. When we speak of tendencies, susceptibilities, proclivities, or predisposition to the transmission of characters, whether they be those of health or of disease, we employ terms which undoubtedly have a certain vagueness. We are quite unable to recognise, even with the highest powers of the microscope, any structural difference in the germ-plasm in different persons which would enable us to say that in one family a particular feature should have one form, in another family a different form; or that in one family there should be a tendency or susceptibility to one kind of disease, whilst another family should display a special capacity for transmitting another form of disease. We can only determine that such a sequence will take place by tracing the life-history of the individuals belonging to these families. Though, as we have said, these terms have a certain vagueness, and are wanting in scientific precision, yet it is not the less true that they express a something of the importance of which we are all conscious, though we may not be able to formulate it in a precise definition.

WILLIAM TURNER.

**HERMAPHRODITE** (*Ἑρμῆς*, Mercury; and *Ἀφροδίτη*, Venus).—A term applied to an individual in whom the formation of the sexual organs is such as to give rise to the impression that both the male and the female organs are present. See MALFORMATIONS.

**HERNIA** (*hernia*, a rupture).—SYNON.: Fr. *Hernie*; Ger. *Bruch*.

**DEFINITION.**—This word is used in surgery to express the protrusion of any viscus from the cavity in which it is naturally placed. In this article, however, the observations are exclusively restricted to protrusions of the viscera of the abdomen through the walls of that cavity, and it is only intended to give a mere outline of the subject, its full discussion being beyond the scope of this work, as it is mainly connected with surgery.

**GENERAL REMARKS.**—Hernial displacements take place in both sexes and at all ages. The most striking objective sign of the existence of a hernia is a fullness or swelling in one or other of those regions of the abdomen where, from the anatomical construction of its walls, the tissues are weakest. When the parietes are defective, in consequence of local disease or injury, visceral protrusions may occur at those spots; and also as the result of congenital malformation in both sexes, a fruitful cause of hernia being the non-closure of the vaginal process of the peritoneum at birth, or a patulous state of the umbilical aperture. Such a protruded viscus forms a hernial swelling or tumour. The tumour is composed of a sac, its contents, and the tissues outside the sac. The sac is composed of a prolongation of the peritoneum in most cases; and its orifice, neck, or abdominal aperture, constitutes, with the tissues around it, a frequent cause of impediment to replacement or 'reduction' of the hernia. The hernia may be a part of any abdominal viscus, but those most mobile are usually displaced. Thus, in the majority of cases, either the omentum or small intestines, together or singly, form the hernia. The tissues outside the sac are those which exist in the region where the tumour is formed, and they are frequently described as the coverings of the sac.

Every hernial tumour possesses a neck, body, and fundus.

The first or earliest objective symptom of a hernial protrusion is an unusual fullness, as, for example, in the groin in a case of inguinal hernia. This swelling is transient, appearing and disappearing in relation to the actions or posture of the individual. When pressed with the finger the swelling disappears; but it is readily reproduced if the patient contracts the abdominal muscles, and then with the finger an impulse is felt, produced by the protruding viscus. By slow degrees the bulk of the swelling increases, until, if no support

or 'truss' be used, very large tumours are formed.

**CLASSIFICATION.**—The most practical classification of hernial protrusions is based upon the usual triple division of the abdomen into regions—namely, the epigastrium, mesogastrium, and hypogastrium.

1. Protrusions in the epigastric region are very rare. They are (a) *diaphragmatic*; and (b) *epigastric*.

*Diaphragmatic* hernia is due either to relaxation of the tissue of the diaphragm muscle, or to its laceration. In some cases congenital deficiency of the muscle is the primary cause; in others the natural openings in the muscle become dilated. Signs of this hernia are very obscure; but when the protrusion depends upon laceration of the muscle, the occurrence of a recent injury may excite suspicion, if associated with abnormal sounds in the thorax.

*Epigastric* hernia escapes at the region formed by the cartilages of the false ribs on either side of the linea alba. It is very rare, and, as the abdominal orifice of the sac is usually large, the hernia is easily reduced.

2. The hernias in the mesogastrium are (a) *ventral*; (b) *umbilical*; and (c) *lumbar*.

The term *ventral* is given to any hernial protrusions escaping through abnormal openings in the walls of the abdomen to which no special name is given. They are seen in the region of the linea alba, above the umbilicus, but most frequently below it; in the line of the linea semilunaris; and even opposite the muscular walls. Commonly of traumatic origin, their nature is clearly shown by the ready manner in which the protruded viscus can be pressed back into the abdomen. They sometimes ensue upon the weakening of the walls after distension, or upon the loss of tissue following abscess.

*Umbilical* hernia is met with at all ages and in both sexes. It forms a tumour at the site of the umbilicus in the first instance, and gradually descends over the linea alba as its bulk increases. Very soon after birth this variety of hernia appears. The protrusion takes place at the umbilical ring, and, pushing before it the peritoneum, an acquired hernial sac is formed. To prevent, therefore, the development of the sac in infancy, and to assist the closure of the ring in the linea alba, a slightly convex disc of cork, enclosed in washleather, should be strapped over the umbilical aperture. The prognosis of infantile umbilical hernia is favourable, for the aperture closes with age, and the tissues continue firm.

In adult life this kind of hernia is frequent in fat individuals. The tumour often acquires enormous proportions. Its contents consist of small intestine and omentum, with not uncommonly a portion of the transverse colon. Accumulations of fecal matter therein often give rise to obstruction, and the symp-

toms arising in consequence of this state more or less resemble those of strangulated small intestine. A correct diagnosis of their cause may usually, however, be arrived at from the history of the attack, the comparative mildness of the malady, and the alleviation of the symptoms by exciting the action of the bowels. The contents of this form of hernia, when of long standing, often become adherent or bound by bands to the sac, in which state they remain permanently irreducible.

When the protruded viscus can be entirely reduced within the abdominal cavity, a suitable well-fitting truss should be worn constantly; if irreducible, one adapted to the circumstances of the case must be used.

*Lumbar* hernia takes place in the loins. It is a very rare variety, and usually occurs as the result of an injury.

3. The herniæ in the hypogastrium are the most numerous and the most common. They include (a) *inguino-scrotal* or *inguino-labial*, above Poupart's ligament; (b) *femoral*, below Poupart's ligament; (c) protrusions through the apertures of the pelvis in front, beneath the horizontal ramus of the pubes—*obturator*; (d) beneath the arch of the pubes—*perineal*, *puddental*, *vaginal*; and (e) behind, through the ischiatic notch—*ischiatic*.

(a) *Inguinal* hernia is seen at all ages and in both sexes. The following varieties are described: the *oblique* or *external*; and the *direct* or *internal*. In the first, the orifice of the sac is outside the course of the internal epigastric artery; in the second it is internal to the same vessel. When the protrusion forming an inguinal hernia does not descend below the inguinal canal it is termed a *bubonocoele*; but when it occupies the scrotum or labium it forms an *inguino-scrotal* or *inguino-labial* tumour. The essential difference between the inguinal hernia of youth and of middle age is due to the constitution of the sac which encloses the protrusion. From infancy to early adult life, protruding viscera escape from the abdomen into a serous sheath continuous with the parietal peritoneum, namely, the vaginal process of that membrane which extends into the scrotum or labium. In middle life and afterwards, the parietal peritoneum is thrust through the apertures or weak points in the abdominal walls by the protruding viscus. In this way two distinct kinds of hernial sac are formed: the first being due to a congenital defect; the second, to a mechanical and acquired cause. In practice, it is very important to bear these distinctions in mind. In the first kind a truss is applied to prevent the passage of the viscus into the sheath, in the hope that by this means its walls may unite and its orifice contract—in fact, to assist nature in accomplishing that condition, the failure of which permits the protrusion to take place. But, in the second, a truss is

used to prevent the protruding viscus pushing the peritoneum before it, and so forming for itself a sac. Thus, if the development of the sac be arrested, there can be no hernial tumour.

Inguinal herniæ occupy the inguinal canal, and are therefore in relation with the spermatic cord of the male and the round ligament of the female. They escape from the canal through the external abdominal ring into the scrotum or labium. The neck of the tumour is always therefore above Poupart's ligament, and to the inner side of the external pillar of the external abdominal ring. This anatomical fact constitutes the main distinction between inguinal and femoral hernia.

(b) *Femoral* hernia forms a tumour at the inner and upper part of the thigh, immediately below the pubic attachments of Poupart's ligament. These structures are in immediate relation with the neck of the sac. The protrusion escapes at the femoral aperture, the site of the entrance of the lymphatic vessels of the thigh to the abdominal cavity. The neck of the sac is therefore to the inner side of the sheath of the femoral vessels; although, in proportion to the bulk of the tumour, its body may overlie it, and even extend upwards above Poupart's ligament, and outwards towards the crest of the ilium. The sac of a femoral hernia is always an acquired formation. Hence the importance of wearing a truss after observing the slightest indication of a femoral protrusion. For if the yielding, relaxed, parietal peritoneum be supported at the crural aperture by a well-adjusted pad, a viscerol hernia must be avoided, as there will be no sac into which it can escape. In other words, arrest the development of the sac and there can be no hernia.

To discriminate between a femoral and an inguinal hernia, place the index finger upon the spinous process of the pubes; if the neck of the tumour is to its outer side, and the whole length of Poupart's ligament can be traced above it, a femoral hernia exists. Should precisely the reverse conditions be ascertained, the tumour will depend upon an inguinal protrusion. Another method for diagnosis is the direct and careful examination of the site of the femoral aperture. If it is clearly and distinctly tangible and well-defined, it cannot be occupied by a hernial protrusion.

Femoral hernia is most common in the adult female. It has been developed before ten years of age; is rare between that age and twenty; but is very frequent in persons between twenty and forty years old. Prolific women are more frequently the subjects of this hernia than the single and sterile.

(c) *Obturator* hernia escapes from the pelvis through the thyroid foramen, and traverses the canal normally occupied by the obturator nerve and vessels. It is rarely met with. A

fulness, rather than a tumour, is produced by the protrusion at the inner or pubic region of the thigh, beneath the pectineus muscle, and accompanied by a peculiar numbness and pain, which may be traced to the distribution of the filaments of the obturator nerve. The lives of patients have been lost in consequence of overlooking these herniæ; the cause of death being only ascertained *post mortem*.

(d) *Perineal, pudental, vaginal*, and (e) *ischiatric* hernias are very rarely seen. The name assigned to each indicates the locality in which the tumour is formed; and for a special description of them the reader must be referred to monographs on the subject of this article.

**EFFECTS AND TREATMENT.**—We must next, as briefly as possible, describe generally the various morbid conditions which the hernia itself may undergo, and the means by which serious consequences from such conditions may be averted.

All hernial protrusions are either reducible or irreducible—that is, they can either be restored to their normal situation, or they may be permanently confined to the region in which they are protruded.

The treatment of all reducible herniæ consists in the employment of means to prevent the escape of the protrusion. To effect this object various kinds of bandages or trusses have been devised. Each kind of hernia requires its special form of truss, and every individual should, as far as practicable, obtain a truss well fitted to his or her configuration. The essentials of a good truss consist in the spring having sufficient power to support the hernia and prevent its escape, while it should not be so strong as to injure the structures about the abdominal rings. The pad should be firm, of a shape suitable to the case, and of a size not inconvenient to the wearer.

The irreducibility of a hernia depends on its bulk, adhesions, and special anatomical conditions. Under such circumstances special bandages must be employed.

But other much more important morbid states of the protruded viscus than the above cause impediments to the reduction of a hernia, namely: (1) *constriction* by the tissues around the orifice of the sac; (2) *accumulation* of fecal matter in the protruded viscus; (3) *inflammation* of the hernia; and (4) *strangulation*, when a part of the alimentary canal forms the hernia.

(1) Those herniæ—for example, the inguinal—which pass through openings in the muscular walls of the abdomen, are liable to *constriction* from contraction of the muscular tissue. Inguinal herniæ of long standing, and more than ordinary bulk, are very prone to become irreducible in consequence of muscular contraction. Such cases are well adapted to illustrate the effects of anæsthetics, and their influence on muscular irritability. If the patient be placed under

the full influence of chloroform, the abdominal muscles become relaxed, and the hernia is quickly reduced.

(2) Hernial protrusions formed of large intestine, such as occur at the umbilicus, frequently become irreducible from fecal *accumulations*. In these cases enemata, and even purgative medicines cautiously prescribed, frequently relieve the symptoms.

(3) *Inflammation* excited in an omental protrusion may cause temporary and even permanent irreducibility. Local and constitutional symptoms of a rather severe type sometimes attend such cases. The usual methods adopted to induce resolution must be employed.

(4) A morbid state of the protruded bowel termed *strangulation* has next to be described. A patient the subject of this state remains in the greatest danger to life so long as the exciting cause, the constriction of the bowel, exists. Hour by hour that danger increases; and, although rare instances of recovery might be quoted after the continuance of strangulation for many hours, the majority of patients die because the intestine was not liberated early enough. A hernia is described as strangulated when subject to a constriction which at first impedes, and sooner or later arrests, the circulation of the blood in its capillary vessels. The passage of the intestinal contents is necessarily stopped. The local and constitutional symptoms are strikingly characteristic. Very frequently the first symptom is vomiting, unaccompanied by any alvine evacuation. The vomiting continues, and is excited by ingesta. This state is probably due to mere obstruction of the alimentary canal, but it ought always to excite the anxious solicitude of the medical attendant to ascertain whether the patient has any outward signs of a hernial tumour. He must examine those regions at which protrusions commonly occur, and never rest content with the statements of the sufferer. At first the pulse is not affected in a very marked way, but as vomiting continues the heart beats more rapidly, whilst the pulse becomes weaker and contracted. The surface, especially that of the extremities, becomes cold; the countenance aged and anxious; the visage, lips, and hands shrivelled and bluish; the prostration extreme. The tumour is painful when touched, and it may have increased in size and become tense. All these facts indicate progressive morbid changes in the tissues of the strangulated bowel, as well as in that part of the alimentary canal above the hernia. Besides the mere act of vomiting all ingesta, the characters of the fluid vomited must be carefully noted. Usually, at first, it is the food last swallowed, more or less digested and mingled with bile; in the second stage it becomes yellowish and greenish; and at last it is stercoraceous—that is,

offensive to smell, of a brownish colour and frothy, and often in great quantity. Now the only treatment of these urgent symptoms consists in the liberation of the bowel by surgical means. In the meantime palliatives may be employed, opium administered by the mouth, enemata injected, local applications of ice used, and gentle taxis applied.

JOHN BIRKETT.

**HERNIA CEREBRI.**—See BRAIN, Malformations of; and SKULL, Diseases and Deformities of.

**HERPES** (ἑρπω, to creep).—SYNON.: Fr. *Dartre*; Ger. *Herpes*, *Bläschenflechte*.

The name 'herpes' was formerly applied to a large group of skin affections, which were associated together on account of a supposed similarity in the eruption, although they bore very little real relation to each other. Thus we had herpes iris, herpes circinatus, and herpes gestationis, diseases which are perfectly distinct from any kind of true herpes. At present only three forms of herpes are recognised: *herpes facialis*, *herpes progenerialis*, and *herpes zoster*; all of which resemble each other not only in the character of the eruption, but also in running an acute and definite course, and in having a neurotic origin. As, however, herpes zoster differs considerably from the other two, it is conveniently distinguished by the name *zona*. This nomenclature has the merit of simplicity, and is that of the College of Physicians.

1. **Herpes Facialis.**—SYNON.: Herpes febrilis; Herpes labialis.

Herpes facialis consists of a small group, or groups, of vesicles on a red base. The eruption is associated with some swelling of the skin or mucous membrane, and with a sensation of very slight pain and stiffness of the part affected. It is most commonly met with on the lips, but occasionally on the ala of the nose, the ear, or the cheek. It is also not uncommon on the buccal mucous membrane, and on that of the soft palate and uvula. It is generally, but by no means always, confined to one side, and this is more observable on the face or ear than on the lips, where it often extends to both sides of the middle line. The eruption is frequently associated with catarrh of the air-passages, and, though quite unimportant, is apt to recur. No treatment is required, as the eruption disappears in a few days, but a lotion of carbolic acid may be usefully applied.

2. **Herpes Progenerialis.**—This disease differs but little from herpes facialis except in the part affected; it is, however, very apt to be troublesome, especially when irritated by rubbing or injudicious treatment. The eruption is met with in both sexes, but is more common in the male than in the female. In men it appears on the prepuce or glans penis; in women on the labia or ad-

jacent mucous membrane. When irritated, it is apt to form small ulcers, which are difficult to heal, and which lead to enlargement of the inguinal glands. Under these circumstances herpes has often been mistaken for syphilis.

TREATMENT.—This should be always of the most soothing kind, such as the application of water dressing or a very weak boric acid or lead lotion, with mild aperients.

3. **Zona.**—SYNON.: Herpes zoster; Shingles.

DEFINITION.—An acute inflammatory disease of the skin, running a definite course, consisting of groups of vesicles on an inflamed base, and distributed irregularly along the course of cutaneous nerves. The eruption is usually unilateral, and is apt to produce scars.

SYMPTOMS.—Zona is by far the most important form of herpes. It is met with most commonly on the trunk, where it follows the course of some of the intercostal or lumbar nerves, and often in the gouty diathesis. It is also not very uncommon in the supra-orbital region, and is occasionally met with following the course of some cutaneous nerves down the arm or leg. It is said to be more common on the right than on the left side of the body. The eruption is preceded by a very considerable amount of neuralgic pain and discomfort, which may even last for days before the eruption appears, and has often been mistaken for the pain of pleurisy. The first appearance of the eruption is a bright-red patch or patches, on which are then developed groups of vesicles of rather large size, sometimes even amounting to blebs. The contents of the vesicles become puriform; and they ultimately dry up and leave scabs, which generally fall off without producing permanent scars. Occasionally, however, troublesome ulcers form, leaving behind deep depressed scars. Mild cases of zona are very apt to abort, the eruption hardly developing into true vesicles; this imperfect development may also be seen in the outlying groups of the eruption in well-developed cases. As a rule the eruption of zona runs a definite course of about a fortnight; there are, however, not a few exceptions in which the period of development is prolonged beyond the normal time. The disease is not very apt to recur, and in this respect it differs from herpes labialis and preputialis. When zona occurs in the neighbourhood of lymphatic glands, these almost always become enlarged during the attack.

PROGNOSIS.—There are two points connected with zona that require especial notice. (1) The gravity of the disease, apart from the amount of eruption, is, broadly speaking, in proportion to the age of the patient. In children and young adults, as soon as the sores have healed the patient is generally quite

well, and no trace of the disease is left except perhaps a few small scars and a slight tenderness of the skin. In the middle aged, and still more in elderly people, this is not the case: the eruption is almost always followed by some form of nerve disturbance, generally of a severe neuralgic kind, which may last for months; in other cases, attacks of a kind of vertigo follow the zona. In very old people an attack of zona is a serious matter from which they seldom entirely recover. (2) It must be always borne in mind that supra-orbital zona is very apt to leave deep depressed scars. The patient should invariably be warned of this in the first instance, otherwise the formation of the scars may be attributed to neglect or injudicious treatment. Sometimes the eye itself is involved in the eruption; and cases of blindness are not unknown as the result of a severe attack of zona.

**TREATMENT.**—The treatment of this eruption is very simple. It should be protected from the pressure of the clothes as far as possible. This may be done with a good pad of cotton wool. Lotions should generally be avoided; and simple powders, such as oxide of zinc or starch, should be well dusted on several times daily. The treatment of the neuralgia which follows zona is most difficult. The pain is often of the most violent darting kind, and not amenable to treatment. All the ordinary remedies used in neuralgia may be tried, including hypodermic injections of morphine and quinine. See NEURALGIA.

ROBERT LIVEING.

**HETEROLOGOUS** (*ἕτερος*, other; and *λόγος*, nature).—A word used to characterise any morbid product, whether fluid or solid, which is different in composition or structure from the normal fluids or solids of the body.

**HETEROMORPHOUS** (*ἕτερος*, other; and *μορφή*, form).—Applied to new-formations which are different in form and structure from the normal tissues.

**HETEROTOPOUS** (*ἕτερος*, other; and *τόπος*, a place).—Misplaced. A term applied to the appearance either of a normal tissue in an unnatural situation—for example, of hairs on mucous surfaces; or of morbid growths in unusual places—for instance, of epithelioma in nervous tissue.

**HICCUP or HICCOUGH.**—SYNON.: *Singultus*; Fr. *Hoquet*; Ger. *der Schlucken*.

**DESCRIPTION.**—Hiccup, according to physiologists, is a sudden spasmodic descent of the diaphragm accompanied by a spasmodic closure of the glottis, the characteristic noise being caused by the incoming column of air striking against the partially closed glottis. The assumption of a spasmodic closure of the glottis in hiccup seems scarcely warrantable. Normally the descent of the diaphragm in each respiratory act is accompanied by a

contraction of the posterior crico-arytænoid muscles, which causes an outward rotation of the arytænoid cartilages, and a dilatation of the glottic aperture. The diaphragmatic and the laryngeal acts keep time together, and in health the rhythm of sixteen or eighteen to the minute is maintained. If, however, the diaphragm give a sudden descending jerk irrespective of any respiratory need, as is the case in hiccup, and this jerk occur at a time when the dilators of the glottis are not acting, a noise will be produced by the rush of air through the insufficiently widened glottic aperture. It seems certainly possible to account for the noise of hiccup by the mere fact of the descent of the diaphragm occurring when the glottis is not properly open. The noise is not a constant phenomenon, and during an attack of hiccup it never occurs during ordinary inspiration, or without the spasmodic action of the diaphragm, although the latter phenomenon may occur without the former.

**ÆTIOLOGY AND DIAGNOSIS.**—Hiccup may be produced by any irritation of the phrenic nerve—its origin, its course, or the ultimate twigs which are distributed to the under-surface of the diaphragm. Undue distension of the stomach, by being overfilled with food or drink, or by an accumulation of wind due to faulty digestion, is the most common cause of hiccup. Its occurrence from this cause is far more common in children than in adults. Convulsions and muscular spasms generally are more easily caused in the young, and hiccup in this respect follows the ordinary rule. Hiccup is produced by direct or by reflex irritation. With many persons the introduction of hot spiced or peppery foods into the stomach immediately produces hiccup, and the writer knows one or two persons in whom hiccup is produced by the passage of hot fluids through the pharynx. It is a frequent symptom in peritonitis when the peritoneal covering of the diaphragm becomes affected. It sometimes occurs in cases of cancer of the stomach; occasionally, perhaps, from over-distension of the organ, but more often from an extension of the cancerous disease to the peritoneal surface of the stomach. It is occasionally a troublesome symptom during convalescence in cholera, and is often accompanied by eructations of wind, and sometimes by vomiting. If hiccup occur with any persistency in the course of typhoid fever, it is often an indication of perforation and the onset of general peritonitis. Although most frequently a symptom of gastric or abdominal disturbance, hiccup occasionally occurs as a true neurosis. It may accompany hydrocephalus or meningitis, and is then due probably to an implication of the cerebral origin of the phrenic nerve. Cases of obstinate hiccuping in hysterical subjects have been recorded, and cases of paroxysmal hiccup have been observed by Liveing,

Prichard, and others, which have been regarded as instances of modified epilepsy.

**TREATMENT.**—The treatment of hiccup will depend upon the cause. An emetic to empty the stomach, or a stimulant to increase its natural peristaltic action, will often give relief. If we can manage to produce a forcible action of the diaphragm, we may often succeed in curing it, as it were, of the trick of spasmodic action. Attempts to count a hundred without drawing breath, or to hold the breath for a minute, are familiar remedies for hiccup; and, by producing a feeling of suffocation, and necessitating a violent descent of the diaphragm, they are often successful. Warm applications or counter-irritation applied to the diaphragmatic region or over the cervical spine, may occasionally give relief. Pressure upon the trunk of the phrenic nerve by means of the finger applied over the scalenus anticus muscle, is said also to have given relief occasionally in obstinate cases. Amongst the drugs which have been recommended for the relief of hiccup are chloroform (administered internally), either alone or combined with opium; camphor in the form of a spirit solution, in doses of twenty drops and upwards; valerianate of zinc, belladonna, bromide of potassium, musk, or antacids; and, in very severe cases, morphine administered hypodermically.

G. V. POORE.

**HIPPURIA** (*ἵππος*, a horse; and *ὄρον*, urine).—The condition of the urine in which it contains hippuric acid in excess. See URINE, Morbid Conditions of.

**HISTRIONIC SPASM** (*histrion*, an actor).—A synonym for facial spasm, so called on account of the contortions of the face to which this affection gives rise. See FACIAL SPASM.

**HIVES.**—A popular term for chicken-pox. See CHICKEN-POX.

**HOARSENESS** (Sax., *has*, having a rough voice).—Roughness of the voice, due to disease or disorder connected with the larynx. See VOICE, Disorders of.

**HOBNAIL LIVER.**—A name given to a cirrhotic liver, when it presents small prominences on its surface, somewhat resembling hobnails. See LIVER, Cirrhosis of.

**HODGKIN'S DISEASE.**—A synonym for lymphadenoma or lymphoma. See LYMPHADENOMA.

**HOMBURG, in Germany.**—Common salt waters. See MINERAL WATERS.

**HOMICIDAL INSANITY.**—See CRIME, Irresponsibility for; and INSANITY, Impulsive.

**HOMOLOGOUS** (*ὁμός*, like; and *λόγος*, nature).—In pathology this term is applied to new-growths presenting the same structure as normal tissues, such as fatty or fibrous tumours.

**HOOPING-COUGH.**—See WHOOPING-COUGH.

**HORDEOLUM** (*hordeum*, a barley-corn).—A synonym for sty. See STYE.

**HORN-POX.**—A popular term for a variety of chicken-pox. See CHICKEN-POX.

**HORNS.**—SYNON. : *Cornua*.—DEFINITION.—Horns are epidermic and epithelial formations, consequent on hypertrophy of the horny product of the integument.

**DESCRIPTION.**—Horns generally occur singly. Sometimes they attain a size of several inches in length and in circumference. They have been met with on all parts of the body, more particularly on the scalp, the face, the glans penis, and the glans clitoridis.

**PATHOLOGY.**—When the inspissated product of the follicles of the skin, consisting of laminated epithelium and sebaceous matter, is exposed to the air, it dries, becomes hard and transparent, and is in fact converted into a mass having most of the properties of horn. This is the principal source of the horns of the integument—an accumulation of the contents of a follicle; the protrusion of that substance through the dilated aperture of the follicle, sometimes through a large opening resulting from atrophy or ulceration; its desiccation by the atmosphere; and its growth by continued additions to its base. Being essentially the protrusion of a soft substance through a constricted aperture, the surface of the horn will be modelled in figure by the shape of that aperture; in consequence of desiccation, its shaft will be smaller than its base; and it will be liable to be bent or twisted in the operation of protrusion. Hence these horns are generally curved or twisted, and have been compared to the beak of a bird, or the horn of the goat. A section of the horn affords similar evidence of its manner of formation and growth, it being always laminated in structure.

Another kind of horn is sometimes met with on the glans penis and clitoridis, and is the product of hypertrophy of the papillæ. This form of growth is fibrous in structure, like a wart, which in fact it closely resembles; whilst in the same situation concreted masses are occasionally formed, constituted by a combination of both processes—namely, papillary hypertrophy and accumulation of follicular substance. There is, however, an important difference between the two kinds of horn, the sebaceous and the epidermic—namely, that the former is the mere result of increased

activity of function, whilst the latter is the consequence of hyperæmia or inflammation.

**TREATMENT.**—Horny matter being susceptible of disintegration by moisture, the sebaceous horn may be so thoroughly softened by envelopment in a waterproof covering, or by a poultice, as to be easily broken away at its base. The follicular bed from which it has been removed may then be cleared by a small scoop, when the sac will contract and close. Sometimes it may be thought desirable to sponge the surface with a solution of chloride of zinc or sulphate of copper; but operation by the knife seems quite uncalled for. In the instances of epidermic and epithelial horn, however, it will be necessary to have recourse to caustics, especially potassa fusa; and when the case evinces great obstinacy, or where an epitheliomatous degeneration is suspected, the use of the knife becomes essential.

ERASMUS WILSON.

**HORRIPILATION** (*horreo*, I bristle up; and *pilus*, a hair).—A sensation of chilliness and creeping, the hairs of the general surface of the skin appearing to stand on end. See RIGOR.

**HOSPITALISM.**—The term 'Hospitalism' was introduced into medical literature by Sir James Simpson (*Edinburgh Medical Journal*, March 1869), but, as far as the writer can see, no exact definition of it was given by its author. It, however, was evidently intended to signify 'the hygienic evils which the system of huge and colossal hospital edifices has hitherto been made to involve'—to use Sir James Simpson's own words. These evils appeared to him so evident, and so necessarily connected with the size of the hospital, that he taught (and in fact the sole object of his papers was to teach) that our system ought to be revolutionised—'hospitals changed from being crowded palaces, with a layer of sick on each flat, into villages or cottages, with one, or at most two, patients in each room—the village constructed of iron instead of brick or stone, and taken down and rebuilt every few years.'

Mr. Erichsen has, to some extent, accepted the teaching of Sir James Simpson, though he allows that some of it is very questionable. His tract on the subject of Hospitalism has the advantage of being written in a more sober style than Sir James Simpson's, and also of putting the question in a clearer light.

By the term, he says, 'is meant a general morbid condition of the building, or of its atmosphere, productive of disease. . . . Doubtless,' says Mr. Erichsen, 'all the septic diseases that are met with in hospitals may be encountered in the practice of surgeons out of these institutions, but they are unquestionably infinitely more common in hospital than in private practice, and their

causes are certainly different.'<sup>1</sup> The writer believes, on the contrary, that, if a hospital be properly managed, there is no general morbid condition of the building—that there is no reason for thinking that septic diseases are more common relatively in hospitals than out of them—and that their causes are identical wherever they occur.

And if the term 'Hospitalism' is to be taken in the sense in which Sir James Simpson evidently intended—that is, as meaning to convey the idea that there is an inevitable tendency to the generation of septic disease in large hospitals, that that tendency becomes greater as the size of the hospital is increased, and that it increases as the hospital grows older—the writer has no hesitation in saying that there is no such thing as Hospitalism. No doubt the aggregation of the sick and wounded in hospitals is a cause of danger, and much care and vigilance are required to keep hospitals healthy. But the dangers are in no sense peculiar to hospitals. The surgical affections which spring up in hospitals—erysipelas, phagedæna, pyæmia, and allied affections—all of them prevail in private practice, and, as far as has been shown, prevail equally.<sup>2</sup> Further, although the perfect publicity of our hospital practice enables us to obtain tolerably accurate data for a comparison of the experience of the smaller and larger hospitals of this and other towns, no one has ever seen the least reason for believing that the smaller are in any respect healthier than the larger; while several of the hospitals that have been longest built are renowned for their healthy condition, and in many large hospitals, parts of which are ancient and other parts modern, the former, if equally well or better constructed, are (under similar conditions of cases and management) as healthy or more healthy than the parts more recently built.

The subject is not one which can be passed over as dealing with an insignificant question, or one of verbal interest only. The doctrines which Sir James Simpson taught led him to deprecate altogether the construction of any hospital of considerable size, and to advocate the extravagant proposal above quoted for substituting small detached temporary sheds for our present permanent hospitals. The same views led Mr. Erichsen to say that when a hospital had become, as he

<sup>1</sup> *On Hospitalism*, p. 37.

<sup>2</sup> It is not, of course, meant that pyæmia, for instance, is seen as often in private as in hospital practice, because its causes are less often met with in the former than the latter; but, if due allowance be made for this obvious consideration, there is much reason to acquiesce in the conclusion to which Sir James Paget's ample experience has led him, that pyæmia is just as frequent in private as in hospital practice (*Clin. Soc. Trans.*, vol. vii. p. lvi.) Erysipelas seems to be more frequent and more fatal in private practice than in hospitals.

phrased it, 'pyæmia-stricken,' it ought to be destroyed,<sup>1</sup> while similar ideas have led others, such as the late Dr. Farr and Miss Nightingale, to question whether hospitals had not destroyed more lives than they had saved. Such doctrines should not be passed over in silence, since they exercise a great influence on the public, on whose co-operation the efficiency of our hospital system is to a great extent based. And certainly a theory which has received the support, however qualified, of so eminent a hospital surgeon as Mr. Erichsen, cannot be considered as of no importance.

It is therefore necessary to point out to the reader that the theory, as so stated (and stated quite correctly after Sir James Simpson's writings), is utterly disproved by the experience of all well-managed hospitals, both before and after the introduction of the antiseptic method of dressing wounds. It must be noticed, in the first place, that the basis of the theory was entirely what is called 'statistics'—that is, a hasty inference from figures, showing the results of a large number of cases on either side. Now nothing is more dangerous than to draw conclusions from such figures, which are quite unsupported by any histories of the cases on which they are founded.<sup>2</sup> The success of a surgical operation depends more on the antecedents of the operation than on its consequents, and the healthiness of hospitals depends far more on other circumstances than on their construction, size, or age. Nay, more, the success of surgical operations does not necessarily vary with the healthiness of the hospital. In the healthiest hospital a careless surgeon, house-surgeon, or nurse may make havoc of the major operations while all is going on well with the general run of patients.

For all these reasons, any one of which would be sufficient, the conclusions of Sir James Simpson are to be repudiated, and to be considered the more mischievous because, while they allege imaginary causes of danger, they thereby conceal those which are real and certain, and necessarily induce surgeons and managers of hospitals to overlook details, attention to which is always followed by success in the treatment of grave surgical cases. With such attention, a hospital may be brought into a condition not inferior to the best circumstances under which private practice is carried on.

The writer must not be misunderstood, as if he thought the details of hospital-construction unimportant. The principles of construction which are now accepted for the building of a hospital will be found at page 879; but it has been shown to

demonstration that, provided wards be well, but not excessively, ventilated, and kept perfectly clean, and provided also that the beds are far enough apart, the precise ground plan of the hospital matters little—that the doctrines so much insisted on by the French writers on hospitals as to the superiority of the 'pavilion plan,' as to the unhealthiness of upper storeys and so on, and which have been adopted as if they were unquestionable truths by many writers on the subject, have led to much waste of money on buildings too scattered for hospital service, which have turned out to be no healthier than the more compact and convenient structures which they superseded. But we ought not, in reaction from these exaggerations, to undervalue the importance of good ventilation, good aëration, proper isolation of beds, and, above all, scrupulous cleanliness in hospital wards. These essentials being secured, the writer is persuaded that a hospital may be just as healthy with thirty wards as with three, with twenty patients in each ward as with two, and with five storeys as with one. Far more important, and far too little thought of, till within the last few years, is the amount of attention given to the personal care of the patients. This is particularly the case in the treatment of open wounds. Everyone who has been much in hospitals must have often seen, and especially abroad, surgeons, dressers, and nurses hurry from one patient to another, hardly washing or wiping their instruments, still less their hands, and using the same dressing materials for one case after another. Surgical practice cannot be safely carried on in this way, however healthy in itself the hospital may be. The first care of a surgeon in charge of hospital-wards ought to be to impress upon all his assistants, and never to forget in his own person, that the success of surgical practice depends more upon minute care in the dressing of cases than on all other matters put together. Our surgical wards have become far more healthy since the introduction of antiseptic surgery; and that this must be largely due to the increased care in the minutæ of surgical treatment which has followed on the discussion of this method is proved by the fact that the improvement is as conspicuous in the practice of some of those who reject, as those who follow, Sir Joseph Lister's teaching.<sup>1</sup>

If it were not true that the septic diseases, or erysipelatous diseases, which interfere so much with the success of operations in our hospitals, depend in a very large proportion of cases on the method of dressing the wound and not on matters conveyed by the atmosphere, how could the success of the so-called

<sup>1</sup> *On Hospitalism*, p. 98.

<sup>2</sup> The writer lays less stress on the total absence of any guarantee for the accuracy of Sir James Simpson's table of cases in private practice, and is willing, for the sake of argument, to assume that the figures are correct.

<sup>1</sup> The late Mr. Callender stated it as his deliberate opinion that the great surgical operations were ten times more successful in hospitals than they used to be in the past generation. This was before the general use of Lister's method.

'open method' of dressing wounds be explained? In this method the wound is left freely exposed to that hospital air which is, we are told, charged with deadly miasma. But care is taken to see that the wound is well drained of all putrefiable discharges, and kept perfectly clean. If Sir James Simpson's theory were true, we ought to have an increased mortality following on the freer exposure to this deadly atmosphere. On the contrary, the perfect drainage of the wound, and the care taken to keep it free from all putrefying matters, are followed by results which can hardly be surpassed. Thus Dr. James Wood, of the Bellevue Hospital, New York, relates, that in wards which had been recently vacated on account of an outbreak of puerperal fever, he treated fourteen successive cases of unselected amputation of the limbs successfully, by merely leaving the flaps ununited, the raw surfaces exposed to the air, but carefully drained, and all putrescible matters continually removed;<sup>1</sup> and this is only one of many proofs which have recently been given of the fact that there are many other plans of treating wounds besides that which is specially designated 'antiseptic,' under which, conjoined with proper construction and general management, a hospital may be as healthy as a private house.

This statement, which is made after long experience, and with a conscientious conviction of its truth, by no means asserts that it is *as easy* to keep a hospital healthy as a private sick-room; or, in other words, that the aggregation of the sick and wounded involves no dangers. Such a doctrine would be absurd; but the dangers are the same in kind, and the precautions required are the same, with a single exception. Hospitals, like private houses, must be kept well-aired, well-drained, scrupulously clean, properly, but not excessively, lighted,<sup>2</sup> and so forth. The great difference in the precautions required to ensure the salubrity of hospitals and private houses is, in one word, to guard against direct infection, and this may occur in two ways. Surgically, infection is carried directly by careless dressing—and every hospital surgeon must have remarked that as he himself is more watchful and careful, and as he has the good fortune to be surrounded by more careful assistants, his cases do better; and in the present healthy condition of most of our large London hospitals, such precautions of themselves suffice to raise the success of surgical practice to a level probably higher than it reaches in private houses.

<sup>1</sup> *New York Med. Journ.*, Jan. 1876.

<sup>2</sup> Most of our hospitals are too light, and too destitute of the means of excluding the light. No doubt a flood of light in the ward is very useful in detecting dirt, but it sadly interferes with the repose which many medical and surgical cases require.

The second way in which infection may be carried is by direct proximity or contact. This is more important in medical cases, and the obvious danger of the spread of infectious fevers has led the managers of most of our general hospitals to exclude such diseases from their wards as small-pox, scarlet fever, and typhus, while other affections are admitted, which, though contagious, are so in a less degree, as typhoid fever, erysipelas, diphtheria, &c. Enough, or more than enough, has been done in this direction—that is, the public safety might be as well consulted if typhus and scarlet fever cases were still admitted (as before the institution of special hospitals for such cases they used to be) into the wards of our general hospitals in small numbers, and under strict precautions. Anyhow, it appears that there is little if any evidence of spread of disease from such cases reputed to be infectious as are still to be found in our general hospitals. Hearing that some distinguished surgeons teach confidently that pyæmia and erysipelas are usually propagated by contagion, the writer has often watched the progress of such cases when originating in hospital or admitted from without, but has never been able to verify any spread of the disease from them, though he does not deny the possibility of such an occurrence.

To sum up the whole matter—the writer would define the term 'Hospitalism' as expressing the danger which exists in hospitals of contamination from the aggregation of patients—and would add that the extent to which such contamination prevails has been greatly exaggerated by theoretical writers; that, as far as the general atmosphere of the ward is concerned, the danger may be, and appears in all well-managed hospitals really to be, obviated by ventilation and cleanliness; and the more immediate danger from the contiguity of patients cannot be shown to produce any appreciable effect, while the danger of contamination of wounds by putrefying materials demands constant vigilance to counteract it; but with such vigilance seems to be so far counteracted that hospital practice is probably more successful than private practice in similar cases. In support of this assertion the following facts are submitted.

Phagedæna, pyæmia, and erysipelas, originating in the hospital, are the affections universally admitted as tests of the healthiness of the wards, or (to use Sir James Simpson's words) of the existence of Hospitalism. In reviewing, a short time ago, his own career as assistant-surgeon and surgeon to St. George's Hospital, the writer had occasion to examine the past and present records of its practice—records, he may say, more perfect and more available than exist, as far as he knows, at any other hospital; for detailed notes are preserved of every case, and are so

tabulated as to be immediately accessible. First, with regard to phagedæna, whether in the milder form, to which the name is often restricted, or in the more formidable sloughing variety spoken of as 'hospital gangrene,' the affection is now unknown; only three cases having been admitted with venereal phagedæna, and none having broken out in the wards, during a period of five years. Next, with regard to pyæmia, there had been fifteen cases in the wards during three years, eight of which were admitted with the disease. Of the seven cases occurring in the hospital, one was puerperal in a patient admitted for another cause; three after suppurative affections—viz. carbuncular abscess, acute periostitis, and diffuse cellulitis—i.e. after various forms of blood-poisoning which had preceded admission; and three only after surgical operations of all kinds, and in one of these (who was under the writer's own care) the diagnosis was doubtful—the man recovered. One of the other cases was an operation for stricture and perineal abscess, where the wound could not be kept aseptic. The last was after an amputation in which the surgeon (who was again the present writer) had probably amputated too near the suppurating tissues. As to erysipelas, a large number of cases of phlegmonous erysipelas were admitted; but there was no record of the outbreak of this disease in the hospital. Cutaneous erysipelas was freely admitted into the ordinary wards, and a few sporadic cases originated in the wards, but almost always of a very mild type. During the two last complete years included in this survey, there had been thirty-one cases on the whole—twenty-three admitted and eight originating in the wards. Only one death occurred out of the whole thirty-one, in a drunkard admitted with scalp-wound. Delirium tremens and erysipelas supervened, and he died in a few days, whether of the delirium or the erysipelas could not be determined.

There is no reason to believe that other hospitals are in any respect inferior to St. George's, so that there seems fair cause for saying that 'Hospitalism' has no existence in the institutions of the present day in this country, if we are to understand by that term any condition of the hospital which exposes a patient to greater risk after injury or operation than he would run if treated at home.

T. HOLMES.

**HOSPITALS, Administration of.**—The administration of a hospital should be so framed as to enforce the necessary economy consistent with the due supply of the requirements for the sick.

*Governing Body.*—The form of government depends to some extent upon the authority under which the hospital is placed; that is to say, whether the hospital is a

foundation or subscription hospital; a hospital established by a municipality or corporation; or a military or naval hospital under the Government.

But there are certain general principles which must in all cases be observed, without which proper discipline throughout the several branches of the administration will not be secured.

The government may be by means of a governor, or by a committee, on which the professional element should be represented. It provides for the general supervision and discipline of the establishment, and for the financial arrangements. It controls the appointment and removal of all members of the staff of the hospital. It makes all general regulations, after consultation with the professional department, as to internal economy, admission and discharge of patients, distribution of beds, dietary, and other matters. It regulates the expenditure. In hospitals supported by subscription, it raises the funds for supporting the institution.

*Governor or Treasurer.*—In large foundation or subscription hospitals, and in military and naval hospitals, there is a resident representative of the governing body, generally termed Governor or Treasurer, who exercises a general supervision over the structure and the discipline of the establishment.

*Secretary or Steward.*—The chief executive officer immediately under the governing body is called the Steward, or sometimes the Secretary. He has the control of all servants not included in the nursing staff—such as porters, ambulance-men, engineer, bath-assistants, and other male attendants. He sees that all structural appliances are in good order, and that cleanliness and discipline are maintained throughout the building. He controls the issue of all orders for the supply of goods, provisions, fuel, &c., and watches that they are used with a due regard to economy. He countersigns all orders for payment after they are passed by the treasurer, and is responsible for the hospital accounts. He sees that the records of admission, discharge, and death are duly kept by the professional staff. He is responsible for the safe custody of clothing, money, and property brought in by the patients, till their discharge. He has charge of the correspondence.

*Clerk or Assistant Steward.*—The secretary or steward is assisted by an assistant or clerk. This latter receives all provisions and stores, sees that they are correctly delivered, and is responsible for their safe custody until distributed to an authorised person. He sees that the diet-tables are prepared from the prescription-papers, and that the articles of food supplied to patients are strictly in accordance with the diet-tables, or else specially ordered by the physician or surgeon.

*Professional Staff.*—The professional staff

directs the proceedings to be taken for the well-being and cure of the patients. It consists of the consulting and visiting physicians and surgeons; the assistant physicians and surgeons; and the resident and house physicians and surgeons, and assistants continually present in the hospital.

*Medical Committee.*—The professional staff forms a medical committee with a specified quorum. This committee is consultative only, and should be consulted by the governing body on all matters concerning the medical and surgical departments of the hospital, the admission and discharge of patients, the distribution of beds, the dispensary, the in-patients, the out-patients' department, and the students. The medical committee, moreover, puts forward recommendations for the purchase of instruments, apparatus, and medicines. The committee provides for a descriptive record of cases admitted into the hospital, and for the efficient instruction of students.

*Physicians and Surgeons.*—The physicians, surgeons, assistant physicians, and assistant surgeons undertake the charge of the wards and out-patients' departments, and attend at the hospital at fixed times. The physicians and surgeons order the diet of the patients; and no article of diet which does not appear in the diet-tables is supplied unless specially ordered by them.

*Resident Medical Officers.*—The resident medical officers control the treatment of patients in the absence of the physicians and surgeons; and have a disciplinary control over the dressers and clinical clerks. They decide on the admissibility or otherwise of applicants for relief, when not admitted directly by the physicians and surgeons, as well as on the wards in which the in-patients are to be placed, and are responsible for their care until seen by the physicians and surgeons. They visit the wards and dispensary, according to the regulations, to see that the patients are duly attended to. They superintend the conduct of the assistants of the medical officers, and of the dispenser and his assistants, of pupils (if any), and of patients; give notice of any misconduct of the nurses and servants to the matron or lady superintendent; and inform the governor or secretary, the physician, surgeon, and governing body, of any matter requiring their attention. They are responsible that the records of cases are properly made out. In most cases, however, this duty is now performed by *registrars*.

*Dispenser.*—The dispenser acts under the resident medical officer, subject to regulations laid down by the governing body.

*Nursing Department.*—The nursing department is under a trained matron, who should be lady superintendent of the training school, and head of all the women employed in the hospital.

*Matron or Lady Superintendent.*—The whole responsibility for nursing, internal management, care of linen and housekeeping, and the discipline and training of nurses is vested in the trained female head of the nursing staff, by whatever title she be called. To the governing body of the hospital she is responsible for the conduct, discipline, and duties of her nurses. To the governing body and the physicians and surgeons in charge of wards she is responsible for the care and cleanliness of the wards, for the care and cleanliness of the sick, and for the linen. She is responsible to the medical officers that their orders about the treatment of the sick are strictly carried out. To fulfil these responsibilities, she has the power of engaging, appointing, and dismissing all nurses, female servants, and probationers, subject, of course, to the general control of the governing body. The nursing establishment cannot be made responsible on the side of discipline to the medical officers, or the governor of a hospital. Simplicity of rules, placing the nurses in all matters regarding management of the sick absolutely under the orders of the medical staff, and in all disciplinary matters absolutely under the lady superintendent or matron, to whom the medical officers should refer all cases of neglect, is essential to due discipline and efficiency. Any remissness or neglect of duty is as much a breach of discipline as drunkenness or other bad conduct, and can only be dealt with to any good purpose by report to the matron. But neither the medical officer nor any other male head should ever have power to punish for disobedience. His duty should end with reporting the case to the female head, who is responsible to the governing authority of the hospital, as all her nurses and servants, in the performance of their duties, are immediately responsible to the matron only. If the matron or lady superintendent does not exercise the authority entrusted to her with judgment and discretion, it is then the legitimate province of the governing body to interfere and to remove her.

The matron resides in the hospital where her nurses and probationers are at work.

In a hospital of, say, above 300 beds, and with a training school of, say, above twenty probationers (which all such hospitals ought to include), the trained matron should have three trained representatives or assistants—one in the training school as mistress of probationers or *home-sister*; and two in the hospital—one by day as *assistant matron* (or superintendent), and one by night as *night superintendent of nurses*; and of these two the night representative is the more important. Besides the trained assistant matron (superintendent), who should have such inspection of the wards as the matron may commit to her, the matron will require one *linen-assistant and housekeeper*, who might

also have the charge and inspection of the nurses' rooms, if the trained assistant matron has not time. She should 'mother' the ward-maids, and have some 'gathering' for them.

*Nurses and Servants.*—Under the matron there should be distinct grades of nurses, and distinct duties for each grade: 1. Trained chief nurses (ward-sisters). 2. Trained nurses (day). 3. Trained nurses (night), at least equal in pay and status to the day-nurses. 4. Probationers in training. 5. Ward-maids, and nursemaids for children's wards. 6. Dormitory and stairs women. 7. Female cook, and her assistants under the house-keeper. The hospital cook in a large hospital would probably be a man, and under the steward. The sisters, nurses, and probationers would require a female cook, under the matron. All women employed in the hospital should reside in the institution.

*Night Superintendent.*—The (trained) night superintendent of nurses should be in charge of the night-nursing, in communication with the ward-sisters, as well as of the night-nurses, and should see that the ventilation and temperature of the wards is maintained, as directed by the medical staff. She must be one qualified not only to have charge of nurses and have some 'gathering' for night-nurses by day, but to train probationers told off to accompany her at night, to their own benefit and hers.

*Assistant Matron.*—The assistant matron is to have special charge over the nurses' rooms, to see that the nurses rise in time to wash themselves, strip their beds, empty their slops, and have breakfast, before going on duty; that later they make their beds and put their rooms in order; that they never wash their own clothes in their own rooms (for all nurses' washing should be done for them); that they go quietly to bed at night, and lights be put out at a certain hour; that their rooms are always clean, in order, wholesome, and cheerful. Without this constant supervision, what is necessary for the nurses' health is not done; the same for night-nurses is yet more important. Nurses' meals should always be presided over by some such authority.

The hospital night-nurses should have two hot meals in the common dining-room—it might be in the probationers' home—say, at 9.30 A.M. and 9.30 P.M., ready and prepared for them. And sisters, staff-nurses, and ward-maids should have, though at different hours—as all cannot be absent from the wards at once—dinner and supper, each set together in a common dining-room, away from the ward air. No nurse should have to prepare her meals for herself.

*Laundry.*—In small and moderate-sized hospitals, when there is a laundry attached to the hospital, the supervision should be under the lady superintendent or matron. In large hospitals, it would be advisable for

the laundry, which should in all cases be in a building entirely detached, to be worked independently, under the general supervision of the governor or secretary.

*Chaplain.*—The religious care of the patients is generally provided for by the appointment of one or more permanent chaplains, or by visits and religious services of other ministers whom the patients may desire to attend them; subject, however, to the opinion of the medical attendant, as to how far the state of health of the patient will admit of such visits, and to the visits being made at such times as do not interfere with the discipline of the hospital.

DOUGLAS GALTON.

**HOSPITALS, Construction of.**<sup>1</sup>—A hospital or infirmary is a building intended for the reception and treatment of sick and injured persons, under conditions favourable for their recovery.

A hospital must be so constructed and arranged as to enable a limited staff of medical men, nurses, and assistants to minister to the necessities of a large number of sick, and to promote their speedy restoration. The conditions essential for such objects are as follows:—

(1) *Pure air.* There should be no appreciable difference in purity between the air inside the wards and that outside the building.

(2) The air supplied should be capable of being warmed to any required extent.

(3) *Pure water* should be supplied for internal use, and sufficient also to ensure the removal of impurities to a distance from the hospital.

(4) The most perfect *cleanliness* within and around the building should be enforced.

These conditions depend on—

(1) The *site* of the proposed hospital.

(2) The *form* of the rooms or wards in which the sick are to be placed, so as to ensure purity of air and convenience of nursing; these rooms forming the principal *units of hospital-construction*.

(3) The *distribution of these units*, and of the other accessories which, combined, constitute the hospital.

**1. Sites of Hospitals.**—The local climate should be healthy, and there should be a free circulation of air over the district. Town sites should be avoided as far as possible. When necessarily placed in a town, a space free from buildings should be reserved on all sides. There should be no nuisances, damp ravines, muddy creeks, undrained or marshy ground, near the site, or in such a position that the prevailing winds would blow effluvia arising from them over the hospital.

<sup>1</sup> This article has reference more especially to permanent general hospitals. The principles apply equally to special temporary field or other institutions for 'the cure of the sick.'

The site selected for a hospital should not receive the drainage of higher ground, and the natural drainage outlets should be sufficient. There should, if possible, be no buildings near a hospital except those connected with its object. The number of sick to be allowed per acre will depend practically upon the arrangement of the buildings in which they are lodged.

**2. Form and Distribution of the Parts of a Hospital.**—The structural arrangements of a hospital should be such as to secure free circulation of air.

**THE WARD.**—The basis upon which the structural arrangements rest is the *ward*. The administration, means of access, and discipline must be made subsidiary to the question as to how the sick are to get well in the shortest possible time, and at the least expense; and this, so far as the structure is concerned, is mainly determined by the form of the wards.

**Size.**—The size of a ward depends upon the number of patients who are to occupy it, and upon the cubic space and floor space which should be allotted to each patient. The disciplinary and economical dispositions in a hospital require that each head-nurse should have the patients allotted to her placed under her immediate eye. Economy of labour requires that the hospital should be so laid out as to enable the largest number of patients to be nursed by a given number of nurses. The *number* to be placed in a ward therefore depends upon the number which can be efficiently nursed; and the form of the ward must be as much calculated to facilitate nursing, as to ensure free circulation and change of air. From twenty to thirty-two beds have been taken as the unit for ward-construction. In hospitals where cases of more than ordinary severity are likely to be received, it would be necessary to diminish the size of the wards on grounds of health. Small wards containing one or more beds are also required for isolating certain cases or for various necessary objects.

**Conditions regulating ward-construction.** The general form of ward-construction is mainly governed by the question of the renewal of air. The air within an inhabited space, enclosed on all sides, is vitiated by the emanations proceeding from the bodies of those who inhabit it, and especially by the effect on it of their respiration. In all sickness, and all surgical cases, wounds with discharge, or sores, these emanations are greater in quantity, and more poisonous in quality, than from persons in health; whilst, at the same time, most cases—medical and surgical—are more susceptible to these emanations. Stagnation in the movement of the air leads to rapid decomposition of these emanations. If they diffused themselves uniformly throughout the space, which in fact they do not, ventilation would be com-

paratively simple, and, whatever the cubic space, the air would attain a permanent degree of purity, or rather impurity, theoretically dependent upon the rate at which emanations are produced, and the rate at which fresh air is admitted. Hence the same supply of air would equally well ventilate any space; but the larger the cubic space, the longer it will be before the air in it attains its permanent condition of impurity, and the more easily will the supply of fresh air be brought in without altering the temperature, and causing injurious draughts. The amount of air which should be removed, and its place supplied with fresh air, is at least 3,000 cubic feet per patient per hour; but this must depend to some extent upon the emanations of the patients, which vary with the diseases or injuries they are suffering from. The ventilation of each ward should be kept independent of other wards or rooms.

**Means of ventilation.**—The change of air may be effected in various ways. For instance, the air may be drawn out by a fan; or it may be removed by a shaft, whose action depends on the difference of the temperature of the air in the shaft and that in the outer atmosphere. Of this the ordinary fireplace is one example; a caldron of water kept boiling for the use of the hospital by a steam pipe is another; a sunlight is a further example; and a heated shaft connected with flues led from holes in the wall near the patients' beds, through each of which air is drawn into the shaft, is another method. Theoretically it is thus quite possible so to arrange the ventilation mechanically that a specified quantity of air at a fixed temperature shall be brought into the ward by day and by night. Practically, however, hospitals dependent upon such means alone for ventilation require an attention which they can rarely or never receive, and, except under favourable circumstances, are not healthy.

The emanations from the body do not uniformly diffuse themselves; they hang about as the smoke of tobacco may be said to do. In wards into which a fixed quantity of air is forced, there is not even a uniform degree of impurity; consequently it is necessary, in order to ensure the purity of the air of a ward, that means should exist for absolutely sweeping out at intervals all the impure air from it, and starting afresh with pure air. This is best effected by the direct action of currents of fresh air brought in by open windows placed on opposite sides of the wards. The distance between windows for this purpose must not be too great to prevent their efficient action in moving the air. Twenty-four feet is a good width, but opposite windows for such an object should in no case be more than from thirty to thirty-five feet apart. The space between the windows should not be obstructed by

walls or partitions. The number of patients—that is to say, the sources of impure emanations—placed between opposite windows, should be limited to two rows. In the daytime, and when the weather admits of open windows, a ward with windows opening on both sides can easily be kept fresh; but for other seasons it is necessary to provide openings for the escape of impure air, and for the admission of fresh air which shall not cause draughts.

For the purpose of removal of air, shafts carried up from near the ceiling-level to above the roof are convenient, the lower ends being louvered to prevent patients feeling down-draughts which may occasionally prevail. The most powerful engine of ventilation for drawing out the air is an open fireplace.

In order to prevent the temperature of the ward from being lowered by the extraction of air, that is, to maintain an equable temperature and to prevent draughts, air warmed to a moderate degree should replace that removed by the fireplace or by other openings. Means for the admission of air in an upward current should be provided direct from the open air, independently of the windows and doors; for this purpose tubes with bends, which favour the collection of dirt, are objectionable. Sherringham's ventilators, which are easily cleaned, placed between the windows near the ceiling, answer well; they admit the air without perceptible draught, and also frequently act as outlets when open on the leeward side of a ward. The external air may be warmed as it enters by being made to pass over hot-water or steam tubes. Openings, if placed close to the floor under the beds, should be capable of being easily and securely closed. All openings for the admission of fresh air should be so placed as to be easily examined and cleaned throughout their whole length, and this should be done at least once a year.

*Superficial area.*—The next most important element in the question of ward-construction is the superficial area to be allotted to the patients, which is even more important than cubic space, for on this depends the distance of the sick from each other, the facility of moving round the sick, shifting beds, cleanliness, and other points of nursing. In connexion with this it is convenient to allude to the question of circular wards. The circle affords a maximum of area in proportion to the perimeter, whilst the rectangular form affords a large perimeter in proportion to the area. Therefore, with a given floor-space the rectangle affords a large wall-space for the beds, whereas the circular form affords a small wall-space, and the beds must consequently be placed close together, whilst the large part of the area enclosed is in the centre of the ward, away from the beds, which is a disadvantage. If there be a medi-

cal school attached to the hospital, the question of area has to be considered with reference to affording the largest amount of accommodation practicable for the teacher and his pupils, without their breathing up the patients' air. A ward with windows improperly placed, so as not to give sufficient light, or where the beds are so placed that the nurse must necessarily obstruct the light in attending to her patients, will require a large floor-space, because the bed-space must be so arranged, and of such dimensions, as to allow of sufficient light falling on the beds. In well-constructed wards with opposite windows, the greatest economy of surface-area can be effected, because the area can be best allotted with reference both to light and to room for work. In a ward 24 feet in width, with a window for every bed or every two beds, a 7 ft. 6 in. bed-space along the walls would probably be sufficient for nursing purposes. This would give 90 square feet per bed, and there should be as little reduction as possible below this amount for average cases of sickness; but this space is much too small for surgical, fever, or lying-in wards, which should also be for a much smaller number. The Herbert Hospital, without a medical school, has 99 superficial feet per bed; St. Thomas' Hospital, with a medical school, has 120 superficial feet per bed. The bed-space should be larger if the locality is less healthy.

The area allotted to patients practically settles the cubic space, because wards should not be less than 12 feet high, though as a rule they need not exceed 14 feet in height.

In a good situation, and for ordinary cases of disease, those spaces which are enough for nursing and for ward-administration would, with good ventilation, be sufficient for all sanitary purposes; but for such cases as typhus or other infectious diseases, operations, and bad surgical diseases, a larger space and area would be required. When severe cases of this class come into an ordinary hospital, the simplest plan is to leave the bed vacant adjacent to that occupied by the patient.

Lying-in wards should never contain more than from one to four beds. In recent French hospitals these wards contain one bed each, and the only access to them is from an open verandah and through a small ante-room. Ovariectomy, and other operations of the like risk, should be performed in a one-bed ward. When space permits, a room may be provided for the use of patients when convalescent, and able to be temporarily absent from the wards. The same observation applies to grounds for outdoor exercise. But separate convalescent hospitals or homes are better, both for health and discipline. If hospitals are provided for special infectious diseases, every patient should be placed in a separate hut-ward. Cleanliness and

abundance of fresh air is the best safeguard against infection.

*Materials.*—With a view to economise heat in winter, and to keep the rooms cool in summer, the walls should be hollow, and all wards should be ceiled, unless the roof is constructed of a good non-conducting material. The best lining for a hospital-ward is an impervious polished surface, which on being washed with soap and water, and dried, would be made quite clean. Plaster, wood, paint, and varnish all absorb the organic impurities given off by the body. Parian cement polished is the best material at present known for walls, but it is costly, and it can only be applied on brick or stone walls, and not on woodwork, because, being inelastic, it is liable to crack. Cracks in a hospital ward are inadmissible, as they get filled with impurities, and harbour insects. The numerous joints required for glazed bricks, or tiles, render the use of these questionable for wards. In default of Parian cement, the safest arrangement is plaster lime-whited or painted, which should be periodically scraped, and be then again lime-whited or painted.

The floor should be as non-absorbent as possible, and for the sake of warmth to the feet it must in this country be of wood. Oak, or other close hard wood, with close joints, oiled and beeswaxed, and rubbed to a polish, makes a very good floor, and absorbs little moisture. The joints should be like those of the best parqueterie, affording no inlet for dirt. There should be no sawdust, or other organic matter subject to decay, under the floor. When a ward is placed over another, it is essential that the floor should be non-conducting of sound, and that it should be so formed as to prevent emanations from patients in the lower ward passing into the upper wards. Practically, with care, a well-laid oak floor, with a good beeswaxed surface, can always be kept clean by rubbing.

There should be as little woodwork as possible in a ward, besides the floor; and what woodwork there is should be varnished, so as to admit of easy washing and drying. The cleanest and most durable material is varnished light-coloured wainscot oak or teak.

The form of the windows must be considered, in their aspect of affording light as a necessary means of promoting health, of affording ventilation, of facilitating nursing, and of enabling the patients to read in bed. Light can always be modified for individual patients. In order to give cheerfulness to the wards, and to renew the air easily, the windows should extend from within 2 feet or 2 feet 6 inches from the floor—so that the patients can see out—to within 1 foot from the ceiling. An end window to a long ward is an element of cheerfulness, and materially assists the renewal of the air at night. It is essential to cleanliness that every part of the

ward should be light. One superficial foot of window-space to from 50 to 55 cubic feet of space, will afford a light and cheerful room, but this depends much on aspect and upon the walls being light-coloured. To economise heat in wards, it is desirable to make the windows of plate glass; double windows of ordinary glass would secure the same object and facilitate ventilation, but they are troublesome to clean, and give a gloomy appearance to a room. The best form of sash for ventilation in this climate is the ordinary sash, opening at top and bottom; but windows made in three or four sections, each of which falls inwards from an axis at the bottom of the section possess many advantages for the hospitals.

*Ward-offices.*—The ward-offices are of two kinds:—

(1) Those which are necessary for facilitating the nursing and administration of the wards, as the *head-nurse's room and ward-scellery*.

(2) Those which are required for the direct use of the sick, so as to prevent any unnecessary processes of the patients taking place in the ward; as, for instance, the *ablution-room*, the bath-room, the water-closets, urinals, and sinks for emptying slops. There ought to be, in addition to the bath-room here mentioned, a general bathing-establishment attached to every hospital, with hot, cold, vapour, sulphur, medicated, shower, and douche baths. Hot and cold water should be laid on to all ward-offices in which the use of either is required, because of the economy of labour in the working of the hospital. For the same reason, when the wards are on two or more floors, lifts should be provided to carry up coals, trays, and patients. The ward-offices should have ample window-space. There should be no dark corners or closed spaces under sinks. Nothing should be kept in these offices which is not in constant use, and everything in use should be open to inspection. All fittings should be light-coloured, so as to show dirt.

(1) The *head-nurse or sister's room* should be sufficiently large to contain her bed and to be also her sitting-room. It should be light, airy, and well-ventilated. It is necessary to discipline that it should be close to the ward-door, and that it should when practicable have a window looking into the ward. If the head-nurse has two wards to supervise, her room should be placed between the two, with a window opening into each.

There should be a *ward-scellery* to each ward, and so situated as to be under the head-nurse's eye. The scellery should be supplied with efficient simple apparatus for its purposes: there should be a small range for ward-cooking, so that the nurse can warm the drinks, prepare fomentations, &c. Shelves or racks should be provided. There should also be a small larder, with free cir-

ulation of air, for temporarily keeping provisions fresh. In the scullery, or adjacent to it, a hot closet should be provided for airing clean towels and sheets. Foul linen should not be retained near the wards, or in the hospital building. It should all be placed in baskets, on wheels, and conveyed as soon as possible to the laundry. Ward sweepings and refuse should similarly be placed in movable receptacles, and taken out of the building with as little delay as possible; structural provision for the retention of these in or near the hospital is undesirable.

(2) The *ablution-room, water-closets, &c.*, ought to be as near as possible to the ward, but cut off from it by a lobby, with windows on each side, having its ventilation and warming distinct from that of the ward and that of the ward-offices, so as to prevent the possibility of foul air passing from the ward-offices into the wards. The ward-offices will be most conveniently placed at the end of the ward farthest from the entrance and nurse's room; and distributed at each side, so as to enable the ward to have an end window. In this arrangement the ablution-room would be on one side of the ward, and should contain a small bath-room with a fixed bath of copper, supplied with hot and cold water. A lavatory-table of impervious material, such as slate or common marble, with a row of sunk porcelain basins with outlet tubes and plugs, each basin supplied with hot and cold water, should be placed in the same compartment as the bath, but separated from it by a partition and door. There should also be room for a portable bath for each ward; and there should be a sink on the floor-level for running off the water from the bath after it has been used.

On the other side of the end window of the ward would be placed the *water-closets*. These should never be against the inner wall, but always against the outer wall of the compartment in which they are placed. A pan of a hemispherical shape, never of a conical shape, with a syphon, abundantly supplied with water to flush it out all round with a large forcible stream, is the best contrivance for the water-closet of a hospital. The urinal should be of a shape to be easily cleaned. The sink for slops, bed-pans, expectoration-cups, &c., which should have a compartment of its own adjoining the water-closets, should be a large, deep, round, pierced basin of earthenware, with a cock extending far enough over the sink for the stream of water to fall directly into the vessel to be cleaned, and with an ample supply of water in a full stream; this sink should be so arranged as to be flushed out like a water-closet pan, and it should be so placed as to make it unnecessary for male patients to pass the nurse on their way to the w.c. There should be a special receptacle near the sink, ventilated to the outer air, for keeping cham-

ber utensils, &c., for the examination of the medical officer.

Walls of ablution-rooms and water-closets should be covered with white glazed tile, slate (enamelled or plain), or Parian cement; plaster is not a good covering for them on account of their liability to be splashed, and of the necessity for their being frequently washed down. There should be separate private water-closets for the nurses; and also water-closets for the patients when in day-rooms or recreation-grounds. The ablution-room and water-closets should be supplied with plenty of fresh air, warmed when necessary, and abundance of light, so as to ensure there being no dark corners. Three water-closets per ward will suffice for a ward of thirty-two beds, but two at least will be required for even a twelve-bed ward.

The water-supply should provide, in addition to pure water for general consumption, a service of distilled water for dressings and such-like purposes.

**3. Aggregation of Ward-Units in the Construction of a Hospital.**—The principles upon which these units of hospital-construction, or, as they are generally termed, pavilions, when so constructed, should be aggregated, are as follows:—

(1) There should be free circulation of air between the pavilions.

(2) The space between them should be exposed to sunshine, and the sunshine should fall on the windows, for which purpose it is desirable that the pavilions should be placed on a north and south line.

(3) The distance between adjacent pavilions should not be less than twice the height of the pavilion reckoned from the floors of the ground-floor ward. This is the smallest width between pavilions which will prevent the lower wards from being gloomy in this climate; where there is not a free movement of air round the buildings, this distance should be increased.

(4) The arrangement of the pavilions should be such as to allow of convenient covered communication between the wards, without interfering with the light and ventilation, and therefore the top of the covered corridor uniting the ends of pavilions should not be carried above the ceiling of the ground-floor wards. Each block of wards—that is, each pavilion—should have its own staircase.

(5) No ward should be so placed as to form a passage-room to other wards.

(6) As a general rule, there should not be more than two floors of wards in a pavilion. If there are three floors or more, the distances between the pavilions become very considerable because of the rule already mentioned, which ought to be absolutely observed, which regulates the distance at which the pavilions must be kept apart. Besides, when two wards open into a common staircase, there is, with every care, to some extent a community of

ventilation; this will prevail even if the staircase be furnished with permanent openings to the outer air. When there are as many as four wards one over the other, the staircase becomes a powerful shaft for drawing up to its upper part, and thence into the ward, the impure air of the lower wards. Similarly, heated impure air from the windows of the lower wards has occasionally a tendency to pass into the windows of the wards above. For the same reason the upper floor over the wards should not be used as a dormitory for nurses. Similarly, if there is a basement under sick wards, it should not be used for any purpose, such as cooking, or for the reception of perishable stores, from which smells could penetrate into the wards; and it is best not to continue the staircase into the basement, for with every care the basement will always form a receptacle for ground air, which should be kept out of the hospital if possible.

There is a limit to the numbers which should be congregated under one roof. This limit will depend very much on the nature of the cases. In town hospitals, a double pavilion should not contain above 80 to 100 beds.

The size of any given hospital ought not to be determined by increasing the number of beds in any one building, but by increasing the number of units or wards. So far as the sick are concerned, they would be better placed in single detached wards; or, for convenience of administration, in wards all on one floor—opening out of a common corridor. But on town sites the cost of land makes it absolutely essential to build hospitals as compactly as possible; moreover, economy in the current expenses will be best secured by a building with wards on two or more floors, provided with lifts and other labour-saving appliances.

In addition to the larger wards, it is necessary, as already stated, to have a few wards of one or two beds each for special cases.

*Corridors* should connect the pavilions on the level of the lower floor of wards only. They should be kept as low as possible, so as not to impede the circulation of air between the pavilions; they should be lighted by windows on both sides, opening widely, or removable in warm weather; and they should be provided with ample means of ventilation, and with fresh warmed air in cold weather, so as to prevent draughts.

The *staircases* should be treated similarly as to light and ventilation, and it is desirable to cut off the connecting corridors from adjacent staircases by swing-doors. These arrangements prevent draughts, and cause the passages and staircases effectually to cut off the ventilation of one pavilion from that of another.

The staircases for patients should be broad and easy; the rise of each step should

not exceed four inches in height, and the tread should be at least one foot in width; there should be a handrail on each side, and a landing after every six or eight steps provided with seats. The patients in their beds may be wheeled on to the roof of the corridors between the pavilions, or on to a broad balcony outside the end window of the ward, in fine weather.

**4. Administrative Buildings.**—The necessary subsidiary accommodation may be briefly described.

*Operating-rooms, dispensary, and school.* These should include examining-room, surgery, drug-store, and operating-theatre; the latter having roof-light from the north, and being of easy access to the surgical wards; there being one theatre for male, and one for female patients. Special wards for such cases as ovariectomy should contain but one bed. A dead-house and *post-mortem* room, with ample means of cleanliness, should be provided, quite outside, and detached from the hospital. Where a school is to be established, the necessary lecture-rooms, laboratories, dissecting-rooms, &c., should be kept entirely separate from the building for the sick.

All rooms should be plain, and without projections or ornaments, which form a resting-place for dust.

*Officers' quarters.*—Apartments for the resident physicians and surgeons, matron, nurses, and servants should not, if practicable, be placed under the same roof with the sick. All the rooms should be light and airy. The resident physicians and surgeons should have each a bedroom and sitting-room, with proper convenience adjacent; and a dining-room for joint use.

The dispenser, if resident, requires a bedroom and sitting-room. An office is required for the steward (purveyor or financial officer), and a room for meetings of the governing body. Each ordinary nurse should have a separate bedroom—neither bare, tarnished, nor dismal; where she may obtain pure air while she sleeps—the night nurses' bedrooms being apart, where they may not be disturbed by noise. There should be bath and closet accommodation near; but each woman should be allowed a washstand and foot-bath in her own room. The head-nurse or 'sister' sleeps in her own room, overlooking her ward; but if there is no common-room, she would have better health if a small sitting-room, as well as bedroom, could be given her off her ward. The head-nurses or 'sisters' should have a dining-room, and also a comfortable, well-furnished sitting-room. They will work better in their wards if they themselves are made comfortable. For sisters and nurses nowadays are, or ought to be, educated women. It is most undesirable that they should seek necessary amusement all out of doors. Nurses should dine

in the sisters' dining-room, but at a different hour; and in a large hospital there would probably be required an additional dining-room for ward-assistants. In hospitals with an establishment for training nurses, which every large hospital ought to have, the probationers or pupil-nurses (in a proportion not exceeding one to every ten or twelve patients) would live in a 'Home' under the hospital roof, and under the direction of the hospital matron.

The 'Home' should consist of—(1) Classroom and nurses' library, large, airy, and convenient. (2) One or two dining-rooms, in which sisters and nurses might also dine, and pantry adjoining. (3) Two rooms and an office for the 'home sister' (class mistress). (4) One separate bedroom for each probationer, sufficient to contain press, table, chair, bookshelf, washstand, bedstead, and arm-chair.

Each floor should have a bath-room and other conveniences. Bedrooms for probationers on night duty should be cut off from the noise of the 'Home.' There should also be provided—one sick room; one visitors' room; and servants' offices and bedrooms for cook and other necessary servants.

*Stores for bedding and linen, kitchen and provision stores.*—The kitchen and all those stores between which and the wards there is a constant movement, should be as central as possible, so as to save labour; but the kitchen should be carefully cut off from the corridor connecting the pavilions. Attached to the kitchen should be a good scullery, larder, and serving conveniences. There should be a large, well-aired, well-lighted, well-warmed, well-arranged linenry and mending-room. The hospital laundry should be detached from the hospital. Special care should be taken to make the laundry buildings airy and very light, with ample means of ventilation for removing the steam, which is heavily charged with organic impurity, and with ample space for the washers. They should have separate drying and ironing rooms.

*Out-patients' department.*—Those hospitals which afford outdoor relief require a dispensary for outdoor sick. This should always have an entrance separate from the hospital, and should never be under or near a ward, or in the space between adjacent wards. It is placed in proximity to the hospital, for the convenience of the medical men, and medical school (if any), and in order to be near the drug-store and apothecary's shop; but it should be entirely detached, with a free air-space between, and no direct communication be possible.

The out-patients' department requires a large airy waiting-room, with separation of sexes, and separate closet accommodation for males and females, which, without great care, may (even when detached) become a

nuisance to the sick-wards; a consulting-room for each of the physicians and surgeons, to which should be attached a small lavatory, and all necessary convenience for the complete examination of patients. The out-patients' department of children's hospitals requires more care, and more ample space in waiting-rooms, because each patient is brought by its nurse. The entrance and exit should be through different doors.

In every large hospital should be provided a well-ventilated chapel, capable of being well warmed.

The position and general construction of the administrative buildings should be made quite subservient to the accommodation for the sick, and to the broad general principle that these buildings should not interfere with the circulation of the air around, or the light of, the wards. DOUGLAS GALTON.

**HOT SPRINGS**, in Bath County, Virginia, U.S.A.—Thermal waters. See MINERAL WATERS.

**HOT SPRINGS**, in Garland County, Arkansas, U.S.A.—Thermal waters. See MINERAL WATERS.

**HOWICK**, in Natal.—See AFRICA, SOUTH.

**HUM, VENOUS.**—A continuous murmur heard in the veins, generally observed in cases of anæmia. See ANÆMIA; and PHYSICAL EXAMINATION.

**HUMID** (*humidus*, moist).—Moist: in contradistinction to *dry*. Applied to *râles* (see PHYSICAL EXAMINATION); to characterise a variety of asthma attended with expectoration (see ASTHMA); and also to particular climates. See CLIMATE, The Treatment of Disease by.

**HUMORAL** (*humor*, a humour).—Of or relating to the humours. Chiefly used as a term for a pathological doctrine—the *humoral pathology*—which associates all diseases with morbid states of the fluids of the body (see BLOOD-DISEASE). The word is also sometimes employed as synonymous with *humid*, when applied to asthma.

**HUNYADI JANOS**, in Hungary. Sulphated waters. See MINERAL WATERS.

**HYDATIDS.**—See ENTOZOA.

**HYDATIDS, UTERINE.**—See MOLE, MOLAR PREGNANCY.

**HYDRÆMIA** (ὑδρωρ, water; and αἷμα, the blood).—A watery condition of the blood. See ANÆMIA; and BLOOD, Morbid Conditions of.

**HYDRAGOGUES** (ὑδρωρ, water; and ἄγω, I drive).—SYNON.: Fr. *Hydragogues*; Ger. *Wasserreibende Mittel*.

DEFINITION.—Purgative medicines which cause a copious watery discharge.

ENUMERATION.—Hydragogue purgatives include:—Bitartrate of Potassium, Buckthorn, Colechicum, Colocynth, Croton Oil, Elaterin, Gamboge, Hellebore, Jalap, Podophyllin, and Scammony. The two most important are Compound Jalap Powder and Elaterin.

ACTION.—It has been supposed by some writers that the action of the drugs included in the present class is due only to the increased peristaltic action which they produce. This, however, is not the case, as certainly some, if not all, of those just enumerated induce a free secretion of fluid by the intestinal mucous membrane, while at the same time they stimulate the peristaltic action of the bowel, and cause the evacuation of this watery fluid.

USES.—Hydragogues are chiefly employed for the removal of fluid from the body, in cases either of general anasarca or of dropsical effusion in serous cavities. They may be employed to assist the action of the kidneys when this is insufficient; and it has been observed not infrequently, when the secretion of urine has previously been deficient, that it becomes greatly increased after a free discharge of fluid has taken place from the bowels, in consequence of the administration of a hydragogue cathartic. See PURGATIVES. T. LAUDER BRUNTON.

**HYDRARGYRIASIS** (*hydrargyrum*, mercury).—The state produced by the introduction of mercury into the system. See MERCURY, Poisoning by.

**HYDRARTHROSIS** (*ὑδάρ*, water; and *ἄρθρον*, a joint).—Effusion of a serous fluid into a joint. See JOINTS, Diseases of.

**HYDROA**.—SYNON.: Fr. *Hydroa bulleux*; *Pemphigus pruriginosus*; *Herpes gestationis*.

The term 'hydroa' was applied by Bazin to three different eruptions which he called respectively, *vesicular hydroa*, *bullous hydroa*, and *vacciniform hydroa*; the last of these has not been recognised by any other writer. Vesicular hydroa is now known to be identical with erythema iris, in which vesicles or small blebs are developed (see ERYTHEMA). Therefore hydroa is at present limited to the hydroa bulleux of Bazin.

SYMPTOMS.—The disease is a severe and rare one; it is attended with much eruption, great itching, and more or less constitutional disturbance. Pregnancy seems in some way to favour its development—hence the name, *herpes gestationis*. The eruption consists of blebs, mixed with pruriginous papules, somewhat resembling those of prurigo. The blebs resemble those of pemphigus, but their average size is smaller and more variable than in the latter disease; the resemblance, however, induced Hardy to name the disease *pemphigus pruriginosus*.

DIFFERENTIAL DIAGNOSIS.—Hydroa should be distinguished from pemphigus, prurigo, and erythema iris. From pemphigus it is distinguished (1) by the relatively small and unequal size of the blebs; (2) by the presence of pruriginous papules, and the intense pruritus which is often present; (3) by the constitutional symptoms, which, though sometimes severe, are less than in cases of pemphigus attended with equal amount of eruption; (4) by the disease not being amenable to arsenical treatment to anything like the same extent as pemphigus.

It is only when the blebs are absent or but little developed that hydroa could be mistaken for prurigo. The relatively acute nature of the disease, and the history of the case would greatly aid the differential diagnosis.

Erythema iris with blebs is probably the disease which is most nearly allied to hydroa; but in the latter the blebs are a chief and characteristic feature, whereas in erythema iris they are, so to speak, accidental and ephemeral. The pruriginous papules and intense pruritus are, moreover, not present in erythema. Altogether, hydroa is a much more severe disease.

TREATMENT.—Alterative aperient remedies may be required; and attention should be given to diet. Arsenic has not been found generally useful, though cases are occasionally met with in which it appears to do some good. Quinine is certainly a more useful tonic. Various local remedies, such as linimentum calcis made with almond oil, or carbolised oil and zinc ointment, may be tried to relieve the irritation of the skin.

ROBERT LIVEING.

**HYDROCARDIA** (*ὑδάρ*, water; and *καρδία*, the heart).—An effusion of serous fluid into the pericardial sac. See PERICARDIUM, Diseases of.

**HYDROCELE** (*ὑδάρ*, water; and *κήλη*, a tumour).—SYNON.: Fr. *Hydrocèle*; Ger. *Wasserbruch*.

DEFINITION.—A swelling produced by a collection of fluid in connexion with (A) the testicle, or (B) the spermatic cord.

A. HYDROCELE OF THE TESTICLE.—The principal forms of hydrocele of the testicle are (1) the *vaginal*, (2) the *congenital*, and (3) the *encysted*; (4) *chylous*, (5) *bilobular*, and (6) *diverticular* hydrocele will also be noticed.

1. **Vaginal Hydrocele**.—DESCRIPTION. This is a chronic dropsical effusion into the sac of the tunica vaginalis. The fluid is a pale-yellowish serum, which in old cases is often loaded with cholesterine. The quantity varies, but seldom exceeds twenty ounces. The writer (T. B. C.) has removed as many as forty-eight ounces. The testicle is usually situated at the back part and rather below the centre of the sac; but its position may be altered by adhesions; and, in cases of con-

genital inversion, the testicle is in front of the sac. In old hydroceles the sac is often greatly thickened by deposition of lymph on the tunica vaginalis, and its conversion into fibrous tissue, which is sometimes the seat of calcareous deposits. It is generally single, but often occurs on both sides. Vaginal hydrocele forms an oval or pyriform swelling, which fluctuates; has a smooth, even surface; and commences at the lower part of the scrotum, very gradually and without pain. When examined by transmitted light it is found to be translucent, except at the back part, where the testicle is situated. Owing to the tunica vaginalis remaining unobliterated for some distance along the cord, the swelling occasionally assumes an elongated form, and extends up towards the inguinal canal. A hydrocele sometimes varies in size, becoming larger and more tense during the day than when the patient first rises in the morning.

**ETIOLOGY.**—Hydrocele is a common disease, especially in warm climates; and occurs at all periods of life, but is most common in middle age.

**DIAGNOSIS.**—The circumstances—that the swelling commences below; that the spermatic cord can be detected above the tumour; that the testicle cannot be felt; and that the tumour receives no impulse on coughing, and does not vary in size on pressure—are signs distinguishing a hydrocele from an inguinal rupture. When the sac is much thickened, so as to obscure fluctuation, and prevent the passage of rays of light, the tumour may be mistaken for a hæmatocele, or disease of the testicle, and the diagnosis is difficult.

**TREATMENT.**—In *infants*, vaginal hydrocele usually disappears under the application of weak tincture of iodine or, as this may irritate the skin, of some evaporating lotion. Acupuncture, causing the fluid to escape into the areolar tissue of the scrotum, is the only operation that is required.

In the *adult* external remedies are of no use. It is usual to resort at once to operative treatment, *palliative* or *radical*. The *palliative* operation consists in puncturing the tumour with a trocar, and evacuating the fluid accumulated in the tunica vaginalis. The hydrocele usually returns in the course of two or three months, and then the operation must be repeated, or the patient must undergo *radical* treatment. This may be effected by incision or excision of the sac; by the passage of a seton; or by injection of the sac with a stimulating fluid. The latter is the plan commonly resorted to; and the fluid almost invariably employed is the tincture of iodine. The only apparatus required, in addition to a medium-sized trocar, is a glass syringe. The surgeon generally injects about a drachm of strong tincture, which he leaves in the sac. The Pharmacopœial preparation is not sufficiently strong. Half a drachm of

iodide of potassium, and 40 grains of iodine, dissolved in one fluid ounce of rectified spirit, as an injection, is generally successful. It causes considerable pain, extending to the loins; but all suffering may be prevented by ether or chloroform inhalation. The iodine acts as a stimulant, stirring up mild inflammation, and causing a rapid return of swelling, which gradually subsides until the patient is cured. Instead of this solution, 30 to 60 minims of pure carbolic acid liquefied with 10 per cent. of glycerine have been injected with a successful result, but a permanent cure has not been obtained in all cases where this agent has been employed. Compression with strapping helps the absorption. In double hydrocele, injection may be performed on both sides at the same time. Injection rarely fails to cure the hydrocele. If it does not succeed, the injection may be repeated, and very often proves effectual, but if not, recourse can be had to the seton; or, in cases of thickened sac, to incision or excision.

**2. Congenital Hydrocele.**—In children, the original communication between the cavities of the peritoneum and of the tunica vaginalis sometimes fails to be obliterated; and fluid accumulated in the sac constitutes the variety termed congenital hydrocele. The communication is usually small in size.

**DIAGNOSIS.**—Congenital hydrocele is easily distinguished from ordinary hydrocele by the absence of a defined boundary on the upper part of the tumour; by the impulse received on coughing; and by pressure causing the disappearance of the swelling, and rendering the testicle perceptible. It is distinguished from a reducible hernia by the fluctuation and transparency of the swelling; by the absence of gurgling accompanying its disappearance on pressure; and by the slow return of the swelling on the patient assuming the erect posture.

**TREATMENT.**—Congenital hydrocele is readily cured by the gentle pressure of a truss on the inguinal canal, so as to occasion obliteration of the neck of the sac, after which the fluid usually becomes absorbed.

**3. Encysted Hydrocele.**—In this form of hydrocele fluid is effused into an adventitious cyst distinct from the vaginal sac, developed in the areolar tissue beneath the visceral portion of the tunica vaginalis investing the head of the epididymis. There may be two or even more such cysts. As the cyst enlarges, the epididymis becomes flattened and displaced to one side, whilst the testicle is found either in front or at the bottom of the cyst. The fluid contained in the sac differs from that of vaginal hydrocele in being less in quantity, perfectly limpid and colourless, and nearly free from albumen. The fluid sometimes contains spermatozoa in great abundance, rendering it opaque and milky-looking. Their presence may be owing to the

rupture of one of the tubes of the epididymis, and the escape of semen into the sac of the hydrocele. More probably, the spermatozoa-containing cysts are due to dilatation of one or more of the vasa efferentia in consequence of stricture; and those that contain limpid fluid may be developed from the organ of Giraldès.

**DIAGNOSIS.**—An encysted hydrocele is distinguished from vaginal hydrocele by the position of the testicle at the bottom of the tumour; and generally by the colourless character of the fluid evacuated.

**TREATMENT.**—When large in size, so as to be inconvenient, encysted hydrocele may be treated in the same way as vaginal hydrocele, and injection is attended with the same success.

**4. Chylous Hydrocele.**—**SYNON.** : Chylocele; Galactocoele; Liparocoele; Fr. *Hydrocèle laiteuse ou graisseuse*.

**DESCRIPTION.**—Chylous hydrocele may be bilateral or unilateral. It is seldom met with in temperate climates. The writer (J. McC.) has seen only one case. It was unilateral, and the patient had lived for many years in India. It occurs more frequently in tropical countries, where it is sometimes associated with elephantiasis of the scrotum. It is not translucent, but it otherwise resembles an ordinary hydrocele. When it is tapped, a fluid resembling milk is evacuated, which, examined microscopically, proved in the writer's (J. McC.) case to be a very fine emulsion without any formed elements. In other cases a few blood-corpuscles, coloured and colourless, have also been seen. The fluid is neutral, and sometimes coagulates spontaneously.

The occasional association of chylous hydrocele with elephantiasis of the scrotum, and the discovery in many cases of the *filaria sanguinis hominis* in the fluid, indicate that it is due to this parasite. Blocking of the lymphatic glands and vessels connected with the testicle by this worm is easily conceivable, but no demonstration would appear to have yet been made of a communication with the chyloferous vessels. See *FILARIA SANGUINIS HOMINIS*.

Two of the recorded cases of chylous hydrocele appear to have resulted from injury, but in the great majority of cases there has been no history of traumatism, and the fluid most probably transudes as in ordinary hydrocele.

**TREATMENT.**—Tapping is merely palliative, as the fluid soon re-accumulates. In one case, at the third tapping the fluid was of the ordinary serous nature, but this result appears to be unique. Injection with solution of iodine is usually successful. If it fail, incision, with drainage, or excision of the sac may be performed.

**5. Bilobular Hydrocele.**—**SYNON.** : *Hydrocèle en bissac* (French surgeons).

**DESCRIPTION.**—This disease may be alto-

gether outside, or partly inside and partly outside, the abdomen. The formation of the former variety is easily explicable. The *processus vaginalis* may be closed completely at the internal abdominal ring, and partially at its connexion with the *tunica vaginalis propria*, the intervening part remaining open. If in such a condition a hydrocele develop, it will form two sacs, continuous internally, and marked at their junction externally by a constriction, the depth of which will vary inversely with the size of the opening between the sacs. It will be a combination of a *funicular* and a *vaginal* hydrocele, with a communication between the sacs. The diagnosis cannot present any difficulty, and the symptoms, possible results, and treatment will not differ from those of an ordinary hydrocele of the *tunica vaginalis propria*.

For the formation of the other variety, where one sac is inside and the other outside the abdomen, two hypotheses are possible. It may result from some congenital malformation of the *processus vaginalis*, or this process may be closed at the internal abdominal ring, and, if a hydrocele subsequently develop, a diverticulum may be formed at the upper part of the process, and experience less resistance to its extension in the sub-peritoneal tissue than in the inguinal canal or the *tunica vaginalis communis*.

This form of bilobular hydrocele has been observed at birth, and the great majority of recorded cases have been in patients under forty years of age; but it may develop or become manifest at a later age. Some cases have been complicated with retention of the testicle within the abdomen or in the inguinal canal, but in others the gland has been in its normal position. The intra-abdominal sac may vary greatly in size. In one case it was not larger than a hazel-nut; in another it extended above the level of the umbilicus and beyond the median line of the abdomen. It is usually situated between the peritoneum and the iliac fascia, but in one case it was between the peritoneum and the *fascia transversalis*. The external sac may also vary in size. Sometimes it is small, and limited to the *processus vaginalis*; but when the *tunica vaginalis propria* is also involved, the sac may be very large. When the contents of the sac are serous, the patient does not experience any greater inconvenience than with an ordinary hydrocele. But there appears to be in these cases a great tendency to the formation of a hæmatocele, and the intimate relation of the internal sac with the peritoneum may then become very important.

**DIAGNOSIS.**—When the intra-abdominal sac is large and causes a projection of the abdominal wall, attention cannot fail to be directed to it, and pressure over the abdominal swelling will produce increased tension

of the external sac, and *vice versa*. But if the intra-abdominal sac be small, or lodged deeply in the iliac fossa, or extend into the pelvis, it may escape notice; but the reducibility, partial or complete, of the contents of the external sac and the *immediate* re-appearance of the swelling when the pressure has been removed, ought to suggest the possibility of an intra-abdominal sac. Careful palpation, when the patient has been anæsthetised, will detect some fulness in the iliac fossa; and if the sac has extended into the pelvis, digital exploration through the rectum will aid the diagnosis.

**TREATMENT.**—The treatment will depend upon the size of the communication between the sacs, the nature of their contents, and their walls. If the communication be free, the contents serous, and the walls thin, tapping the external sac will empty the internal sac also, and injection of iodine solution or carbolic acid in glycerine will probably effect a cure. But if the communication be very narrow, tortuous or valvular, if extravasation of blood has occurred, or if the walls of the sacs be thick, operative treatment varying with the nature of the case will be requisite.

**6. Diverticular Hydrocele.**—This variety of hydrocele is also bilobular, but is altogether scrotal. It was first described by Béraud in 1856, who showed by dissection that the *tunica vaginalis propria* sometimes has very small diverticula. If a hydrocele form, one of these diverticula may become larger and protrude through some accidental rupture or inherently weak part of the *tunica vaginalis communis*. This protrusion might subsequently become so distended as to form the chief part of the swelling.

The *diagnosis* of this condition during life must be very uncertain. The form might be unusual, and the coverings would be very thin, so that translucency would be very marked. If the hydrocele be not tense, it is said that pressure will convey to the hand of the observer a sensation of fluid passing from the diverticulum into the parent sac. It is practically unimportant, for the *treatment* is similar to that of ordinary hydrocele.

**B. HYDROCELE OF THE CORD.**—Hydrocele occurs in the spermatic cord in two forms—*diffused* and *encysted*.

The *diffused*, which is very rare, is simply an œdema of the areolar tissue of the cord.

**Encysted hydrocele** of the cord arises from a collection of fluid in the unobliterated funicular process of peritoneum, which is carried down in the natural transition of the testicle. It produces a small swelling in the cord, of an oval form, above and distinct from the testicle, more or less transparent, and quite movable. The swelling, when small, is of no importance; and it seldom requires treatment. See TESTES, Diseases of.

T. B. CURLING. JEREMIAH MCCARTHY.

## HYDROCEPHALOID DISEASE.—

This term is one which has been applied to a set of symptoms, ill-defined in their mode of grouping, occasionally met with in delicate children soon after they have been weaned. The symptoms may in some cases have been due to reflex disturbance of the nervous system induced by improper food; they may appear as the sequence of some exhausting disease, such as a prolonged diarrhœa; or they may be concomitants of a pneumonia in its initial stages. In each of these, as well as in other allied conditions, there may be a febrile elevation of temperature; combined, in the first place, with undue irritability and extreme restlessness, whilst later on signs of mental sluggishness and stupor may supervene. Such symptoms are apt to be met with where, in addition to a depression of the vital powers, there is a reflex disturbance of brain-functions. The condition was specially described by Marshall Hall, by whom the above name was given. It may be well, therefore, to quote from his own description:—

‘This affection may be divided into two stages—the first that of irritability; the second that of torpor. In the former there appears to be a feeble attempt at reaction; in the latter the powers appear to be more prostrate. These two stages resemble in many of their symptoms the first and second stages of [acute] hydrocephalus respectively.

‘In the first stage the infant becomes irritable, restless, and feverish; the face flushed, the surface hot, and the pulse frequent; there is an undue sensitiveness of the nerves of feeling, and the little patient starts on being touched or on hearing any sudden noise; there are sighing and moaning during sleep, and screaming; the bowels are flatulent and loose, and the evacuations are mucous and disordered.

‘If, through an erroneous notion as to the nature of this affection, nourishment and cordials be not given, or if the diarrhœa continue, either spontaneously or from the administration of medicine, the exhaustion which ensues is apt to lead to a very different train of symptoms. The countenance becomes pale, and the cheeks cool or cold; the eyelids are half-closed, the eyes are unfixed and unattracted by any object placed before them, the pupils unmoved on the approach of light; the breathing, from being quick, becomes irregular and effected by sighs; the voice becomes husky, and there is sometimes a husky, teasing cough; and eventually, if the strength of the little patient continue to decline, there is a crepitus or rattling in the breathing. The evacuations are usually green; the feet are apt to be cold.’

But in such groups of symptoms there is nothing distinctive; and the term ‘hydrocephaloid,’ which is now seldom heard, might be discarded. Its use can only be justified

on the ground that the symptoms met with under such conditions are apt to be occasionally confounded with those which usher in the commencement of acute hydrocephalus. We do not diminish the difficulties — already sufficiently pronounced — besetting the early diagnosis of this latter affection, by endeavouring to group together under one name a set of symptoms which, on different occasions, have no other common bond than that they are apt to occur in delicate children, the tone of whose nervous system has been lowered, and thereby rendered more irritable than natural. We should be really better without such a name, especially now that bleeding and lowering treatment are no longer in vogue for the amelioration of the grave organic affection with which the states in question may occasionally be confounded.

H. CHARLTON BASTIAN.

**HYDROCEPHALUS, Acute** (*ὄδωρ*, water; and *κεφαλή*, the head).—A synonym for tubercular meningitis. Tuberculosis *plus* a meningeal inflammation is the generating condition; and acute hydrocephalus is only the occasional, though very frequent, concomitant. See MENINGES, CEREBRAL, Diseases of.

**HYDROCEPHALUS, Chronic.**—SYNON.: Water on the Brain; *Hydrops Capitis*; Fr. *Hydrocéphale*; *Hydropisie du Cerveau*; Ger. *Der Wasserkopf*; *Hirnwassersucht*.

**DEFINITION.**—A gradual accumulation of serous fluid within the lateral and third ventricles of the brain, causing them to become more or less distended, and the head enlarged; occurring principally in infants or very young children; and leading to restlessness, irritability, or convulsions, followed by dulness, drowsiness, motor weakness or actual paralysis, together with failure of mind and of the special senses.

The essential condition in this malady is the intra-ventricular effusion. The cases in which the fluid has been found outside the brain and within the arachnoid sac are, in all probability, merely examples of the disease in which, intra-ventricular effusion having previously been well-marked, the distended corpus callosum, or, it may be, the floor of the third ventricle, has given way, and allowed the fluid to pass beneath the arachnoid. The so-called extra-ventricular form of the disease is therefore, in the majority of cases, merely a secondary and altogether accidental condition.

As a sequence of a large arachnoid hæmorrhage, serous fluid may also be found within the arachnoid cavity; this, however, is a condition which has no real title to be mentioned under the head of chronic hydrocephalus, as some writers have done. And the same remark applies to those accumulations of serous fluid which take place beneath the arachnoid,

as a sequence of wasting or atrophy of the cerebral hemispheres, one or both. The collection of fluid in such cases is to be regarded as a simple sequence of the atrophy, and is of itself unproductive of morbid symptoms.

**ÆTIOLGY.**—Two principal groups of causes are appealed to as productive of chronic hydrocephalus. In certain cases the affection is believed to be *idiopathic*, due to an 'essential dropsy,' occasioned by an inflammatory affection of the lining membrane of the ventricles. In other patients, however, this affection is distinctly secondary or *symptomatic*, and then may be caused by one or other of two principal sets of conditions. Thus (1) it is often occasioned, as writers of the last century pointed out, by the pressure of scrofulous or other tumours upon the 'straight sinus,' producing mechanical congestion of the great veins of Galen as well as of their radicles on the walls of the lateral ventricles, and, as a consequence, an increasing dropsical condition of the ventricles themselves. (2) Much more rarely it is said to be produced as a sequence of an attack of acute hydrocephalus. This latter mode of origin is admitted by some authors, and denied by others. It is at least a possible mode of origin, although one which is difficult to be established with certainty. By far the largest percentage of cases probably belong to the first set of the 'symptomatic' category.

The disease is sometimes congenital, and may be so far developed during uterine life as to cause great difficulties in parturition—frequently necessitating the sacrifice of the life of the child. At other times the enlargement of the head begins to show itself soon after birth, or at some period before the end of the first or second year. Or it may reveal itself later still in childhood; much more rarely during adolescence; and more rarely still in adult life.

Congenital 'microcephalism' must not be confounded with hydrocephalus. It is true that in certain small-headed infants, having the cranium malformed and the sutures ossified, an excess of fluid may be found within the head; but then the fluid in these cases is situated outside the atrophied brain, and not within the ventricles. The two conditions are, in fact, totally opposite in nature.

**ANATOMICAL CHARACTERS.**—Three different states, in regard to size of head, have been described as existing in this affection—(1) where the head is smaller than natural; (2) where the head is of natural size; and (3) where the head is more or less considerably enlarged.

Those of the first category ought not to be included at all. They are the cases of 'microcephalism' above referred to. Those of the second category could never be diagnosed with any degree of positiveness during life; and it may, indeed, be questioned whether such cases exist to any large extent, except as

more or less transitory stages of instances of the disease pertaining to the third of the above categories.

Even the cases in which the head is distinctly enlarged differ amongst themselves, since in some of them (a) both sutures and fontanelles are widely open; while in others (b) the sutures, and perhaps the fontanelles, are completely closed. It seems probable that the latter may represent conditions into which some of the former pass, when the disease lapses into a chronic and stationary condition.

Owing to the separation of the cranial bones in young infants, this disease, when it occurs in them, soon becomes associated with an actual enlargement of the head, which increases rapidly. In consequence of the distending pressure from within, caused by the increasing size and fulness of the ventricles, the bones entering into the formation of the vault of the cranium become separated from one another, though the bones of the face remain unaltered. The frontal, parietal, the superior part of the occipital, and a small part of the squamous portion of the temporal bones become expanded and thinner than natural, at the same time that they are separated from one another—especially in the regions of the anterior and posterior fontanelles, and of the sagittal suture.

The cranial bones are often very thin, but occasionally they may be unusually thick throughout—even in young children.

The circumference of the head, though that of a young child, may in hydrocephalus easily reach 24 to 30 inches, or more. The size attained by the head in certain cases has been comparatively enormous; thus, in an altogether exceptional case, recorded by Cruikshank, it is said to have measured, in a child sixteen months old, no less than 52 inches in circumference, and the amount of fluid contained within the cranium was found to weigh as much as twenty-seven pounds.

The fluid is generally slightly albuminous; possesses some saline constituents; and has a specific gravity ranging from 1006 to 1014. Its composition agrees pretty closely with that of dropsical fluids generally.

In the great majority of cases, as already stated, the fluid is contained within the more or less distended lateral and third ventricles of the brain. The upper and lateral parts of the cerebral hemispheres, as well as the corpus callosum, become thinned and distended, so as to resemble a mere bag, the walls of which are represented externally by pale unfolded and much flattened convolitional matter, and internally—next the fluid itself—by the lining membrane of the ventricles. This latter has become much thicker and tougher than natural; it may also be more or less granular on the surface; and often shows an increased number of distended vessels. These appearances are, however,

not to be taken as an indication of the inflammatory origin of the malady, as some observers seem to suppose. They may be found, as the writer has seen, well-marked, in cases where the effusion and distension have been the result of a mere mechanical congestion, produced by pressure upon the commencement of the straight sinus, owing to a tumour in the middle lobe of the cerebellum. On the other hand, some years ago the writer examined the head of a hydrocephalic child who died in University College Hospital, in whom the most careful search revealed nothing that could have produced mechanical congestion, and in which there was, moreover, no sign of anything like an inflammatory condition of the walls of the greatly distended lateral ventricles. To fall back upon the hypothesis of an 'essential dropsy' was felt to be far from satisfactory. Such a phrase cannot indeed be regarded as conveying any real explanation of the condition in question.

The shape of the ventricles and of the compressed ganglia about the base is, of course, greatly altered. The foramen of Munro may be half an inch or more in diameter. The optic and olfactory tracts and lobes are also often much altered by pressure.

The brain-substance may be even tougher than natural, because the long-continued mechanical congestion, which exists in so many cases, favours the overgrowth of the neuroglia; and, indeed, it would appear probable that in some instances the overgrowth of this mere connective substance is well-marked. For, notwithstanding all the pressure upon and distension of the brain-substance, this rarely shows signs of atrophy. It is rather the reverse. The mere brain alone of a hydrocephalic child, after the fluid has been evacuated, commonly weighs more than the brain of a healthy child of the same age. Thus, in an instance that came under the writer's notice, the brain of a child five years of age weighed rather more than 52 oz.

In those cases in which during life the fluid has escaped from the ventricles through a rupture in the corpus callosum, the brain has been found more or less flattened and collapsed in the lower part of the enlarged cranium, whilst the escaped fluid occupies the arachnoid cavity above it.

**SYMPTOMS.**—Chronic hydrocephalus begins to manifest itself in various ways, and also, as above stated, at various ages. The great bulk of the cases are either congenital, or commence before the fifth month. But, in certain rare instances, the head may begin to enlarge long after the union of the sutures, in early adult life, or even beyond middle age.

As to modes of commencement, at least four, pretty distinct from one another, may be encountered. (1) The disease may be

ushered in by a period of fretfulness and irritability, with or without the occurrence of convulsions and strabismus, before any enlargement of the head is detected. Or (2) slow enlargement of the head may be noticed as the first event. In some cases, this enlargement not only sets in, but may continue for months, till notable increase in size has taken place, and yet the child may exhibit no morbid symptom whatever. The writer has seen a well-marked instance of this in a child whose head had been enlarging for eighteen months (the process beginning when it was a year old), and in whom, though the head was twenty-four inches in circumference, no other morbid signs or symptoms presented themselves.

(3) Chronic hydrocephalus may supervene in a child after a fall, through the intermediation of cerebellar disease. The writer had under his care a little girl four years old, who, after falling from a table and striking the occiput severely, suffered for from twelve to eighteen months from symptoms indicative of cerebellar disease, after which the head began to enlarge, and hydrocephalus became the apparently dominant condition. Complete blindness ensued, then convulsions set in, and in the midst of one of these the patient died. A tumour of the middle lobe of the cerebellum was found, *plus* all the signs of a well-marked hydrocephalus. (4) The disease may occur as a sequence of an attack resembling acute hydrocephalus (tubercular meningitis); that is to say, a child appears to suffer for a time from what is regarded as tubercular meningitis; the symptoms then undergo some mitigation; they become more or less chronic; and ultimately the head begins to enlarge, as it does in chronic hydrocephalus. There is some doubt about the real nature of the starting-point in this mode of origin. The initial symptoms may not in reality have been those of tubercular meningitis. The chronic disease and its symptoms may occasionally be initiated in an acute manner.

It may be easily imagined that the subsequent course of the symptoms in persons suffering from chronic hydrocephalus, beginning in these various ways, may also be subject to great variations.

As the head enlarges, or as the pressure within increases, sensations of weight or pain may be experienced. The child may show increased fretfulness and irritability; or its manner may become more dull and heavy than natural. At other times there is no noticeable change in these respects.

In the 'symptomatic' forms associated with tumours, there is apt to be vomiting of a very obstinate and paroxysmal character, together with continuous pain, marked by exacerbations. Convulsions, either unilateral or general, may also occur, as well as paralysis of one or other of the ocular muscles.

In such cases, too, in comparatively early stages, ophthalmoscopic examination will frequently reveal optic neuritis, which has a tendency to go on to white atrophy, with the production of more or less complete blindness.

In later stages of the disease mental action becomes increasingly impaired; there is loss of memory, dulness, and a great tendency to sleep during the day. There may be marked weakness or actual paralysis of limbs. Children affected to this extent often keep to the recumbent position, having at last no power of sitting up, or even of raising their head from the pillow. Where the enlargement becomes extreme the weight of the head is so great that it cannot be maintained in the upright position. It has to be supported by the hand or some artificial prop; or else the child does not attempt to rise from the recumbent position. A sense of fluctuation is often recognisable. The forehead becomes prominent and overhanging, while the eyeballs are depressed; and as the face remains unaltered or even becomes emaciated, it seems altogether unnaturally small, and thus contributes to produce a most characteristic appearance ('*facies hydrocephalica*'), which is often intensified by the old-looking, and more or less blank, expressionless aspect of the face. The appetite sometimes remains good; at other times it becomes much impaired, and a gradual emaciation ensues. Blindness, more rarely deafness, loss of smell, and impairment of other senses tend gradually to reveal themselves after a time.

COMPLICATIONS.—In all cases where the hydrocephalus is itself symptomatic of some primary intracranial disease, interfering with the proper return of blood from the ventricles and central portions of the brain, the symptoms resulting from this latter state of things are necessarily complicated with others immediately produced by the original morbid condition. Hence the very great variations encountered in the grouping of symptoms in different cases.

DIAGNOSIS.—Where the head becomes distinctly enlarged, with widely separated sutures and open fontanelles, there can be scarcely any room for doubt about the diagnosis. But before the head is distinctly enlarged, the diagnosis of chronic hydrocephalus with any degree of certainty is impossible. In many cases also where the head is only slightly enlarged, and the sutures are not opened, it may be very difficult, for a time, to pronounce an opinion as to whether or not an infant or young child is hydrocephalic. Natural variations in the size of the head are considerable; and it may also be enlarged from rickets, or from that very rare condition known as 'hypertrophy of the brain.' Even great thickenings of the bones of the head have occasionally given rise to uncertainties in regard to diagnosis. But in

all these cases, in order to enable the practitioner to arrive at a trustworthy opinion, the particular form of the head has to be considered, together with the sum-total of the various symptoms which may have preceded or accompanied its increase.

Whether we have to do with an instance of 'idiopathic' or of 'symptomatic' hydrocephalus in any particular case often cannot be settled; but in others it can be decided by the existence of symptoms distinctly pointing to the presence of an intracranial new-growth.

**COURSE AND TERMINATIONS.**—Hydrocephalus often proves fatal in the course of a few months; or it may be less rapid, entailing death only after a year or two. Its progress is variable, however. Remissions and stationary conditions are apt to occur, with occasionally distinct exacerbations of all the symptoms.

Occasionally one of these stationary conditions becomes prolonged, and the individual may live for years. Some hydrocephalic subjects have subsequently lived on to the age of twenty, thirty, or even forty years. In a few exceptional cases a cure seems to have been effected, either naturally or under the influence of remedial agencies.

Death may take place in convulsions; from slow exhaustion with emaciation; or from intercurrent pneumonia or some other acute disease.

**TREATMENT.**—Very little, unfortunately, can be done, in the majority of cases, to produce decided or lasting improvement. This is especially so in those instances—only too numerous—in which the hydrocephalus is due to some scrofulous or other tumour interfering with the return of blood from the ventricles.

Blistering the scalp, with mercurial inunctions, formerly much lauded, may do a great deal more harm than good; and the same may be said in reference to pressure of the enlarged head by strapping or bandages. This latter is a barbarously coarse method of treatment, which has happily fallen into disuse. Blistering may do good in some cases, but it should be cautiously employed.

The general health of the child must be maintained as much as possible, by the aid, if necessary, of tonics and cod-liver oil. Purgation and diuretics may also be had recourse to. Iodide of potassium may be given internally in gradually increasing doses, as even young children bear this remedy well. Bromide of potassium will also help, perhaps, to mitigate vomiting and convulsions, when these are urgent symptoms.

It may be worth while in suitable cases to try the effect of greatly diminishing the amount of fluids taken, so as to reduce the fulness of the vascular system. The writer has had reason to believe that this method is well worthy of being tried, where other

means have failed, and where there is any chance of being able to carry it out.

Puncture of the head has been much lauded, and practised by many, but with an amount of failure and fatality that has caused the method to have almost fallen into disuse. If in any given case we could be reasonably certain that the hydrocephalus belonged to the 'idiopathic' variety (if there really are such cases), the method might be had recourse to with much more chance of success than if it were occasioned by some mechanical pressure, which persists, and prevents the return of blood from the central parts of the brain. Puncture of the head can scarcely be compared with puncture of the chest, because (even apart from the greater risks attaching to the former operation) a puncture of the chest in a case of pleurisy has a fair chance of being actually curative, while puncture of the head in hydrocephalus, in the majority of cases and for the reason above indicated, could only be palliative. Still the cases of this disease are so grave that where the sutures are opened, where the patient's condition is rapidly getting worse, and death seems otherwise inevitable, the question of performing the operation, at least once, ought to be entertained as a possible means of affording relief.

H. CHARLTON BASTIAN.

**HYDROMETRA** (ὑδωρ, water; and μήτρα, the womb).—Dropsy of the womb. See WOMB, Diseases of.

**HYDRONEPHROSIS** (ὑδωρ, water; and νεφρός, the kidney).—SYNON.: Dropsy of the Kidney; Fr. *Hydronephrose*; Ger. *Hydronephrose*.

**DEFINITION.**—A chronic disease of the kidney, caused by obstruction of the ureter; which leads to dilatation of the pelvis, and commonly of the ureter, with more or less extensive atrophy of the substance of the organ; usually affecting one kidney, sometimes both; characterised clinically by the presence of a soft fluctuating tumour in the renal region, but most distinctively by sudden discharge of urine with collapse of the tumour; and resulting, if not relieved, in complete destruction of the kidney.

**ÆTIOLOGY.**—Among the most common causes of hydronephrosis are obstruction of the ureter at its lowest part, in consequence of pressure by new formations, such as carcinoma of the uterus or tumour of the ovary in females, and the impaction of calculi of various kinds. More rarely it is induced by new formations in the bladder; stricture of the urethra; the pressure of the pregnant, prolapsed, or retroflexed uterus. Exceptionally it is met with without apparent cause; in such cases it is to be referred to some contraction, due to inflammation or other cause, which has disappeared. The condition is also sometimes congenital, being

due to malformations, such as impermeable ureter, or valve-like obstruction to the passage of the urine downwards. From whatever cause the obstruction springs, accumulation of urine takes place behind it, leading to gradual distension of the organ. See URETERS, Diseases of.

**ANATOMICAL CHARACTERS.**—In the earlier stage of hydronephrosis there is simple dilatation of the pelvis of the kidney. As the disease advances, the dilatation increases, the organ becomes more and more distended, and the ureter often becomes so dilated or elongated as to present the appearance of a bluish-white tube, as large as or even larger than the inferior vena cava. Coincidentally with this distension and pressure the substance of the kidney atrophies. At first the apices of the cones become flattened and wasted, but gradually the renal substance becomes more involved, until at length, in old-standing cases, scarcely any trace of it remains, and the kidney is represented by a large lobulated bag, the fibrous walls of which are distended by clear fluid. Commonly one kidney only is affected, especially when extreme dilatation exists, but in some instances both kidneys are involved. There is a case on record where the whole abdominal cavity was occupied by an enormous sac, containing sixty pounds of fluid.

**SYMPTOMS.**—In the slighter cases of hydronephrosis there are no symptoms of such a kind as to attract attention. This condition is often an unimportant complication of other serious diseases. In the more severe cases there are no constitutional symptoms, but the local changes are well-marked. There is a tumour situated in the lumbar region, extending upwards, downwards into adjacent regions, or forwards towards the anterior abdominal wall. The colon is usually in front of the tumour, and always displaced and compressed, so that constipation frequently coexists with hydronephrosis. The mass is often lobulated, always undulating in character; and frequently fluctuation may be detected. The most conclusive evidence of the condition is afforded by the discharge of a large quantity of urine, generally of low specific gravity, and often containing mucus, coincidentally with the disappearance or diminution of the tumour. It sometimes happens that the obstruction is permanently removed, and dilatation alone remains as evidence of the old obstruction. Hydronephrosis, when double, sooner or later proves fatal by suppression of urine or uræmia. When only existing on one side, it has proved fatal by pressure upon neighbouring parts; by the supervention of impaction of a stone in the kidney of the opposite side; or from other causes.<sup>1</sup>

<sup>1</sup> In one case, which occurred some years ago, the cyst, in collapsing after being tapped, carried with it

**DIAGNOSIS.**—The distinction of hydronephrosis from ascites may be sometimes difficult when the disease affects both sides. The diagnosis is partly to be made by observing the effects of change of posture, hydronephrosis being much less influenced thereby than ascites. The history and mode of origin of the affection also afford indications. From hydatids of the kidney it is sometimes impossible to discriminate hydronephrosis, but the history of the case and the characters of the urine often afford a clue. If there be tumour on both sides, it is extremely unlikely to be hydatid. From ovarian tumour the diagnosis is to be made by the history of the case, the position of the mass, and its relations to the colon; and by vaginal and rectal examination. From perinephric abscess hydronephrosis is distinguished by its being less hard, and by the absence of pain and fever.

**PROGNOSIS.**—The prognosis is always serious; but if one of the kidneys be sound, it becomes enlarged, and does double work; and so long as this condition continues, the patient may suffer little inconvenience.

**TREATMENT.**—Careful manipulation of the tumour is often useful in extreme conditions. Tapping with the aspirator may be employed, and the further aid of the surgeon may be required. T. GRAINGER STEWART.

**HYDROPATHY** (ὑδωρ, water; and πάθος, a disease).—A synonym for hydrotherapeutics. See HYDROTHERAPEUTICS.

**HYDROPERICARDIUM** (ὑδωρ, water; περί, about; and καρδία, the heart).—An accumulation of serum in the pericardium, either dropsical or inflammatory. See PERICARDIUM, Diseases of.

**HYDROPHOBIA** (ὑδωρ, water; and φόβος, fear).—SYNON.: Rabies; Dog-madness; Fr. *Lyssa*; *la Rage*; Ger. *Hundswuth*; *Wasserscheu*.

**DEFINITION.**—An acute disease, produced by the inoculation of a specific animal poison; manifesting itself by symptoms due to disturbance of the central nervous system; and almost invariably proving fatal. The name 'hydrophobia' is really that of a single symptom, which, though very common, is not invariable.

**ÆTIOLGY.**—Never spontaneous in man, the disease is always due to inoculation with the poison from a rabid animal, almost invariably with the saliva, and commonly by a bite. In about nine-tenths of the cases, the malady is contracted from dogs; in most of the remainder from cats; in very few cases from foxes or wolves, but a larger proportion is due to wolf-bites in countries where these animals are numerous. It has also been acquired through a wound received during the sigmoid flexure, which being thus turned on itself caused fatal obstruction.—EDITOR.

the dissection of a rabid animal. Inoculation with the blood of a rabid animal will probably (but not certainly) produce the disease. The poison is not known to be present in any other secretion than the saliva, but it is abundant, after death, in the central nervous system. Commencing decomposition is said to destroy its activity, but it sometimes resists this for many days. The dried saliva retains its virulence for some days, but in the dried nerve-centres the virus gradually loses its activity, and becomes inert in about ten days.

Inoculation by a bite takes place more certainly if this is on an uncovered part of the body, as on the hands and face, than when inflicted through the clothes, by which the teeth may be cleansed. But it may occur without an actual bite, as by a lick upon an abrasion. It has followed the scratch of a cat, probably by the animal's saliva being thus inoculated. A healthy dog has communicated the disease by a bite given immediately after it had been fighting with a rabid animal, the saliva of which, no doubt, was hanging about its jaws. The disease has resulted from the teeth being used to loosen a knot upon a rope with which a rabid dog had been tied.

It has been asserted that the disease may arise from the bite of a healthy dog, but this is most improbable. Cases are on record, however, in which the disease has followed the bite of a dog, which did not at the time, or for several weeks afterwards, present the recognised symptoms of the disorder. It is probable that, in such cases, rabies has occurred as a mild and insignificant malady, since it is certain that a dog may recover even after communicating fatal rabies.

When no preventive measures are adopted, at least half, perhaps two-thirds, of persons bitten escape. The immunity may be due partly to the bites being inflicted through clothes, but probably a fallacy underlies the estimate, owing to the inclusion of many bites from dogs not really rabid. A certain diagnosis can only be made by inoculation, but the preventive treatment of Pasteur has, fortunately, made it now impossible to obtain statistics on the point.

More males than females suffer, the proportion being three to one. The largest number of cases occur in the middle period of life, doubtless from greater exposure to the cause. Children, however, are prone to suffer, being often badly bitten on the face. Most cases are contracted from straying dogs, or from pet dogs that have been bitten in some street fight.

The *period of incubation* is longer than that of any other acute specific disease, and is singularly variable. It is rarely less than a month, the shortest on record having been about twelve days. The average period is six or seven weeks. In about half the cases

it is between one and three months. In some cases it is longer, reaching six, nine, or twelve months. Cases have been recorded in which two, three, five, and even ten years intervened. Most authorities believe that such cases were either not true hydrophobia, or were due to a second unknown infection. Although it is as hard to explain an incubation period of one year as of five years, it is certain that the lick of a strange dog on a scratched hand is more likely to be forgotten than remembered, and an unnoted later infection is, on the whole, the most probable explanation of these cases.

**ANATOMICAL CHARACTERS.**—General fluidity of the blood, such as is met with after death from acute septic diseases; redness of the throat and pharynx, and occasionally of the salivary glands; together with, in some cases, evidence of congestion of the brain and spinal cord, constitute the chief morbid appearances visible to the naked eye. The microscope has shown that there is evidence of inflammation (congestion and leucocytal infiltration) in the salivary glands (Coats); and that minute changes in the nerve-centres are almost constantly to be found (Clifford Allbutt, Hammond, Benedikt, Coats, and the writer). Of nine cases examined by the writer, the microscopic changes in seven were distinct, and in character and position so far characteristic, that, given the fact of an *acute* disease, a *post-mortem* diagnosis might, in this proportion of cases, be made with certainty by the microscope. The essential change consists in the accumulation of leucocytes around the vessels, and their infiltration into the adjacent tissue; this change having a special distribution, being either confined to, or most intense in, the region of the medulla which is contiguous to the lower part of the fourth ventricle, that is, the neighbourhood of the respiratory centre. Here we have also the centre for deglutition. The change is most intense in the hypoglossal, glosso-pharyngeal, and vagal nuclei and their neighbourhood. There is little or no change in the upper part of the medulla, corpora quadrigemina, cerebellum, or basal ganglia. In the convolutions a similar but slighter alteration is present in some cases. It is, however, often very marked in the spinal cord. In the most affected regions, traces of *ante-mortem* clots and even of inflammation of the walls of the minute vessels may be found in some cases. Perivascular areas of disintegration are common; but such frequently occur apart from hydrophobia or any other disease. Minute extravasations are common, partly mechanical. The only change in the nerve-elements themselves consists in a granular degeneration of the ganglion-cells of the regions chiefly diseased. In the dog the changes are quite similar in characters and distribution.

**SYMPTOMS.**—During the period of incuba-

tion of hydrophobia there are commonly no symptoms. Vesicles under the tongue were formerly described, but their occurrence has not been confirmed. Occasionally pain and discomfort have been felt in the seat of the wound, explicable, in part, by the attention directed to it, but sometimes too pronounced to be thus explained, especially during the week or two preceding the onset. Mental depression has been noted, also probably due to anxiety regarding the possible consequence of the bite.

The onset of the acute symptoms is commonly attended by no local disturbance, although sometimes by pain, rarely by actual inflammation, in the wound. The first evidence of the impending disorder is usually malaise, mental depression, disturbed sleep, and some discomfort about the throat, with a difficulty in swallowing, especially liquids. The attempt occasions some spasm in the throat, which soon, if not at first, involves the muscles of respiration, causing a short, quick inspiration, a 'catch in the breath,' resembling that due to an affusion of cold water. In a few hours this increases to a strong inspiratory effort, in which the extraordinary muscles of respiration take more part than the diaphragm; the shoulders are raised, and the angles of the mouth are drawn outwards. The saliva, which is abundant and viscid, cannot be swallowed. It hangs about the mouth, and the patient is annoyed by his efforts to get rid of it. As the intensity of the spasm increases, so does the readiness with which it is excited. The mere contact of water with the lips, or cutaneous impressions, as a draught of air, will bring on a paroxysm, owing to the intense reflex excitability. The distress thus occasioned leads to a mental state, which is due to disturbance of the cortex, but increases the readiness with which the spasm is produced. The intense excitability of the sensory centres may be such that mere sight of water, or the sound of dropping water, will cause the spasm, and even analogous visual impressions, as a sudden light or the reflection from a looking-glass. Thus the respiratory spasm excited by swallowing liquids, which is, as it were, the keynote of the disease, extends on the one hand to widely spread muscular spasm, and on the other to mental disturbance. In each of these directions the symptoms develop. The early reflex excess involves the spinal cord, the knee-jerk is increased, and reflex action in the legs may be great. While this over-action increases, the spasm, from being limited to the muscles of respiration, may become general and convulsive in character, though still excited by the same causes. The mental distress passes into disturbance, in which the balance of reason is lost, continuously or during the paroxysms. In the frenzy, the horror of the distress is transferred to the attendants by whom

any discomfort may have been occasioned, and during the paroxysms the patient may attempt to bite them, and even others. Conscientiousness may so far remain that in the intervals he may beg those whom he regards to keep away. The saliva is ejected with force, and the patient hawks it up with a noise 'like a dog.' The sight of a dog has been known greatly to intensify the disturbance; and this, strangely enough, in cases in which the sufferer had no suspicion of the nature of his affection. The delirium may, in some cases, be continuous and violent. As the mental disturbance increases, the respiratory spasm and convulsion may lessen, or the latter may persist to the end.

Vomiting is common, and is often an early symptom, a greenish-brown liquid being ejected. Priapism or nymphomania occasionally occurs. The temperature is usually raised two or three degrees. Albumen is often present in the urine; and sometimes sugar is found.

**DURATION AND TERMINATIONS.**—The duration of hydrophobia is usually from one to four days; sometimes it lasts six, eight, or ten days. In the rare cases which have recovered, the duration of the acute affection has been from four to ten days, although slight spasmodic symptoms have lasted for a longer time, as does the spasm of whooping-cough.

The common cause of death is exhaustion due to the attacks of fury and convulsion, often aided by manifest cardiac failure, which may occur early, and be out of proportion to the general asthenia. Sometimes the patient has died asphyxiated in a paroxysm of respiratory spasm; partly, perhaps, from spasm of the glottis.

**VARIETIES.**—The relative degree of the above-described symptoms varies in different cases. The mental disturbance, or the general muscular spasm, may, respectively, predominate over the respiratory throat-spasm, even in the early stages, and may impress a special character on the attack, so that it resembles in the one case a primary mental affection, in the other a general convulsive affection, as tetanus.

**PATHOLOGY.**—We cannot doubt that the virus of hydrophobia is a micro-organism, although this has not yet been clearly detected. It is supposed that the organisms or their germs, frequently at least, remain in the tissues near the wound, and only become dislodged at a later period, to reach the nerve-centres and there develop (Pasteur). The symptoms indicate a primary influence on the nerve-centres, especially on the respiratory region of the medulla, spreading more widely in its ultimate action, both in the medulla and to the brain and cord. The vascular changes, from their variability and occasional absence, are probably secondary effects of the

disturbed action of the nerve-structures, produced by the poison carried by the blood. The virus has been thought to ascend by the nerves, but this is not likely, although germs may simultaneously enter the peripheral nerves and develop there, since the virus has been found to be abundant in them (Dowdeswell).

The first effect of the poison is probably to lessen the 'resistance' of the medullary centres. Their action becomes spasmodic, and is excited with undue readiness. The excessive action of the medulla and spinal cord finds its counterpart in the cortex and the delirium which results. At a later stage this gives place to depressed activity, which may end in paralysis and coma, when general, but is sometimes unequal in its distribution. The secondary vascular changes may have their own effects. They are, as in other functional diseases, somewhat random in distribution within the affected area. By the infiltration of leucocytes, the tissue may be broken up, and what are practically minute points of suppuration may result. If the part damaged is important, grave consequences may ensue. The nucleus of the pneumogastric is often so damaged, and from this, or the late depression, we can understand the occurrence of cardiac failure. The changes in the convolutions and the spinal cord are probably proportioned to the mental or tetanoid (and paralytic) symptoms respectively. The mental excitement no doubt acts upon and increases the irritability of the medulla (Putnam). Conversely, the disturbance of the latter may help to determine the character of the mental disturbance due to the poison of hydrophobia.

**DIAGNOSIS.**—The symptom of greatest diagnostic value is the respiratory spasm excited by attempts to swallow, increasing until it resembles a convulsive action, and accompanied after a time by mental disturbance. The diagnosis is generally aided by the history of a suspicious bite, but it must be remembered that such a history sometimes misleads, especially when the mind of the patient has dwelt much on the possible consequences, and he has made himself acquainted with the symptoms of the disease. In certain diseases of the throat and chest, especially œsophagitis and pericarditis, a reflex throat-spasm may occur, but in such cases there are commonly pain and other obtrusive signs of the local affection. When the mental disturbance occurs early, the affection may be confounded with acute mania: the association with slight respiratory spasm is still the most important diagnostic indication. In cases (if such occur) in which this symptom is absent, the diagnosis is a matter of great difficulty, and can only be made by the history of the antecedent bite, by the rapid course of the disease, and by its association with other convulsive phenomena

and with salivation. From tetanus, hydrophobia is distinguished by the late period after the bite at which the symptoms develop; by the absence of trismus and of continuous spasm; and by the presence of paroxysmal respiratory spasm, of aversion to liquids, and of mental disturbance. Too much weight must not be given to the general character of the convulsive symptoms, if other signs of the disease are present, since there is probably a tetanoid form of hydrophobia; but in this the spasm is excited by attempts at deglutition, and there is no trismus. Organic brain-diseases accompanied by delirium and convulsions, occurring after a bite, have been mistaken for hydrophobia, as in one case in which the nature of the disease was only discovered when, after exhumation, meningeal hæmorrhage was found. Here also the respiratory spasm was absent.

Mere mental excitement, directed to the symptoms of the disease, may determine dysphagia simulating that of the genuine disease—'spurious hydrophobia' as it has been termed, often accompanied by delirious excitement. The spasm, however, is not of the respiratory character of true hydrophobia. Recovery commonly ensues on the mind being tranquillised, or by the application of some remedy in which the patient has confidence. It must be remembered that in some cases of genuine hydrophobia the influence of the patient's mental state has been very clearly traceable even in the early symptoms, and it is probable that many true cases have been regarded as spurious, and even published as such. It is doubtful whether death has ever resulted except from the genuine disease.

**PROGNOSIS.**—Hydrophobia is practically fatal, but not certainly so. Recovery has certainly occurred in the dog and probably in man. Every patient should unquestionably receive the 'benefit of the doubt.' Cases differ in the intensity and rapidity of their course; and the less rapidly the symptoms are evolved, the greater is the hope, slight though it still is, that an exception to the common fatality may be obtained. The prognosis is better the longer the spasm remains limited; it is worse if there are general convulsions, mental disturbance, and signs of exhaustion or of disproportionate heart-failure.

**TREATMENT.**—Adequate measures against the spread of rabies would undoubtedly lessen, perhaps entirely prevent, the occurrence of hydrophobia in man; but the discussion of these is beyond the scope of this article (*see RABIES*). When a person has been bitten by a suspicious animal, the circulation in the part should, if possible, be at once arrested by a tight ligature above the place; the wound should be washed; and then it should be allowed to bleed freely. It may probably be sucked with impunity if the mouth is

rinsed with water or, better still, with vinegar and water, after each act, especially if there are no abrasions in the mucous membrane. The act has been supposed to be dangerous; but all experience is opposed to this. Poisons have to remain for some minutes in contact with a mucous membrane before they are absorbed, and during the act of sucking there is a flow from the mucous membrane into the mouth, which must be opposed to absorption. The wound should be cauterised as soon as possible, and before circulation through the part is again permitted. Of chemical caustics, nitrate of silver, freely applied at once, is probably effectual. If any time have elapsed, nitric acid or liquid carbolic acid is preferable. The actual cautery, applied deeply and freely, is an efficient and ready means. If practicable, free excision of the bitten part is wise, and should not be neglected, even though the cautery has before been used, especially if there is any doubt as to the thoroughness of the application. Without arrest of the circulation, cauterisation after ten minutes has often failed.

Next in time, but first in importance, is the prophylactic treatment of Pasteur, employed as soon as possible after the bite. The dried spinal cord of a rabbit contains less and less of the virus, up to the tenth day of drying, when it fails to produce the disease. If an emulsion of such a cord is first injected, then, on successive days, that of cords dried for a shorter time may be employed, until a strength of cord-virus is injected that would otherwise cause the disease. Not only does this fail to do so, but it prevents the occurrence of rabies from the virus inoculated by the preceding bite, and thus confers immunity even against the past inoculation. It is believed that the organisms of the virus produce a material, probably chemical, destructive to them, incompatible with their development, and that this is not destroyed by the drying in the same degree as the virus. Being injected in larger and larger quantities from cords dried for a shorter time, it effectually protects both from the virus injected with it and from that which was already in the system. Facts show that this result, though not absolutely certain, generally follows, and that the mortality from bites by certainly rabid animals is reduced to about 1 per cent. The most careful estimate shows that, during the last five years Pasteur has thus saved at least 1,000 lives. (For a detailed account of the treatment, see Ruffer, *Brit. Med. Journ.* 1889.)

The methods adopted for the treatment of the developed disease have been numerous, but, for the most part, quite ineffectual. All so-called 'specifics' have been proved to be useless. An attempt has been made to neutralise the poison by administering large doses of mercury, and to eliminate it by dia-

phoresis. The two have been combined in the mercurial vapour-bath. Two or three cases are on record in which mercury has appeared successful; many in which it has been powerless. Of late it has been but little tried; nevertheless, there are facts which suggest that the apparent influence of mercury has been real, and that in it we have probably a more promising means of counteracting the virus than in any other agent. The vapour-bath treatment has been abundantly proved to be entirely useless, although its supposed value is, from time to time, reaffirmed by lay advocates whose energy is proportioned to their ignorance.

Other agents employed have been designed to counteract, not the virus, but its consequences, and so to prevent or postpone death, possibly until the malady has run its course. One of these is curara, first recommended half a century ago by an Englishman (Sewell); it was tried in small doses and failed. Niemeyer suggested larger doses, and in a case by Offenburg it was apparently successful; and since then another case, in America, has recovered under its use. In many cases it has failed—in all cases in this country up to the present time. It is recommended that it should be used in injections of from  $\frac{1}{15}$  to  $\frac{1}{2}$  of a grain, repeated every quarter or half an hour, until the severity of the paroxysms is lessened. This point may not, however, be reached until general muscular paralysis is imminent or produced, and then artificial respiration may be necessary until the effect has passed away. As often as this is the case, and the spasms recur, another injection must be given. In hydrophobia there is remarkable tolerance of the drug, poisonous doses (one grain repeated) of active curara having in one case been without any effect (Curtis). This is, perhaps, a hopeful fact, as it indicates that curara has an action to which the changes in the central nervous system are opposed.

Ordinary sedatives have been the remedies commonly employed, and of these the best are chloral and morphine. One case (probably genuine), in which morphine and Calabar bean were used, recovered (Nicholls); and one in which chloral was employed lived for ten days (Sansou). The effect of the two on the respiratory centre in animals suggests their joint use. The morphine should be given by hypodermic, the chloral by rectal injection. Chloroform is useful in moderating the paroxysms, but appears somewhat inferior to chloral. Other sedatives—Indian hemp, &c.—have appeared of inferior value.

Cold affusions to the cervical spine and head were used in India in two cases which recovered, the throat and spine being blistered with nitrate of silver, and chloroform administered. Ice to the spine has been tried without effect. Tracheotomy was recommended by Marshall Hall in one case. Death

from laryngeal spasm is, however, too rare to justify the measure.

In all cases tranquillity is of the greatest importance. Every excitant of spasm should be avoided; the patient being kept in a dim still room, and friends as much as possible excluded. Next in importance is nourishment, which should be given by the rectum, if spasm is excited by the attempt to swallow. Restraint, which may be necessary, should be as little as possible, but it should be effectual.

The saliva of persons suffering from hydrophobia has been proved to be capable of communicating the disease to animals. Hence the attendants should be cautioned to have no uncovered abrasion on the hands, and to wash from the eyes or face any saliva which may have been spit on them; and if they are bitten by the patient, the wound should be treated as if it had been inflicted by a rabid animal. These precautions remove all danger; and any anxiety the subjects may feel may be relieved by the assurance that, of the thousands of persons who have attended on patients with hydrophobia, no authentic instance has ever been recorded in which the disease was contracted, either by attendance during life, or inspection after death.

W. R. GOWERS.

**HYDROPS** (ὑδρωρ, water).—A synonym for dropsy. See DROPSY.

**HYDRORHACHIS** (ὑδρωρ, water; and ῥάχης, the spine).—A collection of fluid in the spinal canal. The term is commonly used as a synonym for spina bifida. See SPINA BIFIDA.

**HYDROTHERAPEUTICS** (ὑδρωρ, water; and θεραπεύω, I treat).—SYNON.: Water-cure; Hydropathy; Fr. *Hydrothérapie*; Ger. *Wasserheilkunde*.

It would be out of place were we to enter here into a description of the sources and the composition of the numerous varieties of water used for hygienic and dietetic purposes; but we may refer to Dr. Parkes' instructive *Manual of Practical Hygiene*. We intend to divide this article into (1) a short sketch of the history of the water-cure or hydrotherapeutics; (2) notes on the internal use of water, and on the more common hydrotherapeutic procedures; and (3) a consideration of the morbid conditions suitable for hydrotherapeutic treatment.

**History of the Water-cure.**—Although the old Greek and Roman physicians occasionally employed water internally and externally in the treatment of disease, the systematic water-treatment seems to have gained ground for the first time in the fifteenth and in the beginning of the sixteenth century in Italy and France, and again after a period of oblivion in the seventeenth century, especially in England (Floyer, T. Smith), and in

the beginning of the eighteenth century in Germany (F. Hoffmann). The next important scientific application we owe to J. G. and J. S. Hahn, who, towards the middle of the eighteenth century, treated febrile diseases with cold sponging, and were so convinced of the beneficial result, that one of them when attacked with typhoid fever subjected himself to this treatment. It fell, however, again into neglect, until towards the latter part of the eighteenth century, when Wm. Wright, James Currie, W. Jackson, and others resuscitated the cold-water treatment in fevers, and strengthened their reasoning by thermometric observations. In spite of the results obtained, not only in England but also in Germany, amongst others by Reuss, Frohlich, Brandis, and Horn, the method was again falling into disuse when, soon after 1820, a small farmer, Vincent Priessnitz, of Graefenberg, in Silesia, began to treat every kind of ailment, chronic as well as acute, with various hydrotherapeutic procedures, and added to the external applications the abundant internal use of water, combined with active exercise, and a very simple diet; prohibiting at the same time all alcoholic beverages, and also tea and coffee. Priessnitz gradually made considerable changes in his method of treatment. For the original packing during several hours in dry woollen blankets covered with featherbeds, and followed by cold affusions, he substituted packing in wet linen sheets during several hours, followed by a full bath or a douche; and at a still later period he frequently employed cold wet packing of only fifteen or twenty minutes' duration, repeated several times on the same day; he introduced also the method of rubbing the whole body with a cold wet or dripping sheet instead of the full bath, and made extensive use of partial baths, as hip or sitz baths, baths for the hands, the arms, the feet, wet abdominal belts, and wet compresses over different parts of the body. Priessnitz seemed to search for a universal method applicable to all cases. One of the guiding ideas was that disease of the most different nature was caused by an acrid humour in the blood, and that the skin was the organ through which this humour was to be removed.

Though the success of such treatment, combined with active exercise in a healthy mountainous country, and simple diet, was considerable in many cases, the indiscriminate, too energetic, and protracted use often led to unfavourable results, and the system was beginning to be regarded as a species of quackery, when, about fifty-five years ago, some establishments were placed under the superintendence of regularly educated physicians, who studied the physiological effects of the different forms of bathing, and modified them with regard to duration, temperature, &c., according to the requirements of

individual cases, combining pharmaceutical remedies with hydrotherapeutic procedures when required. Thus a more or less modified water-cure has now become a branch of rational medicine, at least in France and Germany; and a new impulse has been given to it during the last thirty years, by the employment of various forms of baths in the treatment of fevers. In this country there is as yet very little systematised relation between the special hydrotherapeutic and the general medical treatment; and the experience gained at hydrotherapeutic establishments is not communicated and discussed in our medical societies, and scarcely ever in the general medical journals. This is much to be regretted, for there can be no doubt that hydrotherapeutic measures might be more widely introduced with great advantage into our hospitals, as well as into our private practice; but this is not likely to be the case so long as the medical profession has not fuller opportunities for studying the effects of water-treatment. The fault may lie to some degree in the nature of most of the establishments for the water-cure; but this might be remedied if more establishments were to be erected under the guidance of superior members of the profession—establishments where the usual medical, dietetic, and hygienic treatment, as well as massage and gymnastics, would in suitable cases go hand in hand with hydrotherapeutic management adapted to the individuality of each case.

**Internal Therapeutic Use of Water, and the more Common External Hydrotherapeutic Procedures.**—The dietetic necessity of water is well known; life cannot exist without it; all our tissues contain an indispensable proportion of water; we constantly lose a large amount by respiration, and by all excretions; all the internal functions of tissue-change are dependent on a certain quantity of water. This want is supplied by the solid and fluid food which we take, water included; while temporary excess of supply leads to increased discharge by the excretions, and temporary deficiency to a diminution of the water of the excretions. An increased ingestion of water further leads, for a time at least, to an increased removal of the products of retrogressive tissue-change; the tissues and the blood itself are, so to speak, washed out by it; and, as the consequence of the increased removal of the used-up material, the body is enabled to take in a larger amount of new substance, and hence we observe not rarely increase of weight as the effect of plentiful water-drinking, if not carried to excess as regards quantity and time; the secretions of the urine, bile, saliva, and pancreatic juice appear to be increased by the abundant internal use of water, as well as the perspiration; though the latter to some degree requires the concomitant in-

fluence of high external temperature or bodily exercise.

Water has also an important share in all internal courses of mineral waters. Used by itself, it can exercise some good influence in cases of gout and gravel, in hæmorrhoidal complaints, imperfect secretion of bile, and constipation from sluggish peristaltic action. As, however, excessive water-drinking, according to Priessnitz's original plan, is apt to cause dyspeptic troubles, water is now, in general, used internally only either for dietetic purposes, or to assist in other courses of treatment.

The *external use* of cold water admits of a very great variety of applications, and a corresponding variety of effects on the body. The two main effects of the different forms of cold baths are *abstraction of heat*, with its further influences on the functions of the body; and *stimulation of the cutaneous nerves*, and through these of the nerve-centres. The two effects are usually combined, but in some forms of bath the stimulation or the *exciting effect* preponderates; in others the abstraction of heat, with its *calming* or *depressing influence*. Hence the different forms of baths, or rather hydrotherapeutic procedures, may be divided into *stimulating* and *calming*, but it is to be borne in mind that there is no strict line of distinction. With this limitation we may regard as *stimulating*—the *full* cold bath of short duration, the stimulating action of which is increased by motion of the water, be it natural or artificially imparted; the rapid wash-down, either by means of a large sponge, or a wet sheet, with or without friction; the spouting of the back, and the pail-douche; the needle-bath or circular shower-bath; the different forms of the rain-bath, and the usual shower-bath; the great variety of other douches; and the running or flowing sitz-bath. The immediate effects of these stimulating forms, in a constitution endowed with a certain amount of reactive power, are exhilaration, increased activity of circulation and muscular force, and improved appetite and digestive power. By altering the duration of the bath and the temperature of the water, the effects may be considerably modified, and thus adapted to different conditions. In adult, as well as young persons, most beneficial effects are often produced by the combination of warm baths with cold douches on the spine.

The more *calming* forms are—the wet sheet-envelope, entire or partial; the impermeable wet compresses; the full cold bath of long duration and without motion; the sitz, the shallow, and foot baths without motion; and the full bath of higher temperature. Depression through abstraction of heat exceeds the stimulation in these forms: diminution of nervous irritability, of sensation and mental activity, and of the frequency

of the pulse and energy of circulation; a feeling of lassitude; and a tendency to sleep, are the principal effects. These forms can, however, be modified, and the effects vary in proportion. Thus the wet sheet-envelope allows ample variation by using warm or cold water, by using the sheet dripping or wrung out, by making it fit tightly round the neck, by moving it to and fro, by frequently changing it, &c. The calming and stimulating form may be further combined by using, first, the wet sheet-envelope, or the woollen blanket-envelope, for a sufficient period to produce perspiration; and then a more or less cold bath or shower-bath of short duration. The physician has, indeed, infinite varieties of application at his disposal, to be used according to necessity.

Powerful and most important hydrotherapeutic helps are the different vapour and hot air-baths (Russian, Roman, Turkish baths), combined with douches and baths of various temperatures. These kinds of baths are, however, treated of in another article. See **BATHS**.

A plain nourishing diet, without or with only a limited amount of stimulants; outdoor exercise in proportion to the strength of the individual; and in some cases massage, in others active or passive gymnastics, are likewise to be regarded as valuable adjuvants in the hydrotherapeutic treatment of chronic diseases. For massage and muscular exercise mean not only increased action, oxidation, excretion, and development of muscle, but also increased general circulation and respiration, increased inhalation of oxygen, and increased production of heat, so necessary in the cold water-cure. There is also no reason whatever why suitable pharmaceutical remedies should not be combined with the water-cure treatment—a method which, as already mentioned, is frequently adopted in the best establishments.

**Therapeutic Effects, and Morbid Conditions Suitable for Hydrotherapeutic Treatment.**—The principal results of well-adapted courses of cold-water treatment are: improved nutrition and action of the skin; increased tone of the nerve-centres; regulation of the circulation; amelioration of the sanguification and nutrition; and acceleration of the retrogressive tissue-changes. It is essential for such successful results that the organism be able to stand a certain amount of abstraction of heat; that it be capable of more or less energetic reaction; and that the digestive and assimilative organs be able to take up a fair amount of nourishing material, which is required by the increased demand on the body.

*Acute febrile diseases.*—Amongst the oldest therapeutic uses of the cold bath, though it has only lately been more extensively revived, is the employment of different forms of cold

baths in acute febrile diseases, attended with a high degree of pyrexia. The moderately cold or the cooled-down bath, as proposed by Dr. von Ziemssen of Munich, is the form principally employed; but cold affusions, the shower-bath, the wet envelope frequently changed, cold compresses, the application of ice in substance, washing with iced water, and iced enemas, are likewise applicable; and the liberal internal allowance of cold water forms an important part of the dietetic management of this class of diseases.

*Typhoid fever.*—Typhoid fever is the disease in which this treatment, with numerous modifications, has been most generally adopted. As soon as the temperature of the patient reaches  $102.2^{\circ}$  to  $103^{\circ}$  F., he is placed in a bath of about  $90^{\circ}$  F., and the temperature is gradually cooled down, by the addition of cold water, to  $80^{\circ}$  or  $60^{\circ}$  F., according to the patient's power of reaction. The patient is kept in the bath generally from 10 to 15 or 20 minutes, when slight shivering often manifests itself. The patient's temperature, measured in the rectum, is usually reduced by this procedure about  $1\frac{1}{2}^{\circ}$  to  $5^{\circ}$  F., not immediately, but within the first hour after the bath. As often as the temperature may again reach  $102.2^{\circ}$  to  $103^{\circ}$ , the patient is again placed in the bath. Thus, during the height of the pyrexia three to five baths may be required in twenty-four hours, while later on about two are usually sufficient, and often only one. Instead of the bath gradually cooled down, a bath of a temperature between  $60^{\circ}$  and  $90^{\circ}$  F., may be given, according to the condition of the individual patient. The frequent and careful use of the thermometer is an essential element in this method of treatment, which may be, as it often is, advantageously combined with the administration of alcohol, quinine, and other remedies. The earlier the baths are commenced, the greater seems to be their influence in mitigating the severity of the disease and its sequelæ, and in shortening its duration. In the numerous accounts of German physicians (Brand, Ziemssen, Zimmermann, Jürgensen, Liebermeister, &c.) it is claimed that the mortality is considerably less with this than with the expectant or any of the other usual modes of treatment.

*Hyperpyrexia.*—A still bolder use may be made of the cold-water treatment in those rarer cases of hyperpyrexia occasionally occurring in the course of rheumatic fever, when the temperature rises to  $108^{\circ}$  F. and more; and where very cold and prolonged baths, the application of ice-bags, &c., appear to be the only means of saving life. The same treatment is likely to be useful in some forms of sunstroke; and also in some cases of hyperpyrexia arising from other acute disease than rheumatic fever. (See the late Dr. Wilson Fox's *Treatment*

of *Hyperpyrexia*; the writer's case in the *Clinical Society's Transactions*, vol. v.; and several other papers in the *Clinical Society's Transactions*.)

*Scarlet fever*.—In scarlet fever the writer has found warmer baths (80° to 98° F.) more generally applicable than quite cold or cooled-down baths, though in cases attended by a high degree of pyrexia and brain-symptoms these are preferable.

*Hectic fever*.—In hectic fever, connected with various chronic diseases, the effect of hydrotherapeutic treatment is less decided, and not yet sufficiently tested.

*Digestive derangements* of the most different kind, associated with *sluggish venous circulation* in the abdominal organs—conditions which may be grouped together under the term *abdominal venosity*, tendency to hæmorrhoids, to hypochondriasis, &c., are often the objects of the water-cure, which may be useful by stimulating the physical and psychical energy of the nervous system, as well as the nutrition and tissue-change, by invigorating the skin. Habitual constipation from this cause is often relieved by the hydrotherapeutic belt. In this class of cases the common salt waters, and the alkaline sulphated waters are more frequently used, and are often preferable; they may, however, be advantageously combined with judicious hydrotherapeutic treatment.

*Chronic metallic poisoning* may be treated in some cases with equal benefit, if there is sufficient reactive power, at cold-water establishments, as at the thermal sulphur and simple thermal spas. The external hydrotherapeutic procedures, aiming at increased perspiration and tissue-change, are in this class aided by the abundant internal use of water, in order to wash out the tissues, and especially the liver.

*Skin-weakness or atony of the skin* is often the cause of frequently recurring attacks of diarrhœa with neuralgic pains, of tendency to catarrh of the respiratory mucous membrane, and of rheumatism. Gently stimulating hydrotherapeutic appliances, with gradually increasing energy, are here mostly useful, unless, as in impeded convalescence, the reactive power is so reduced that the gaseous thermal salt-baths and mountain-air are preferable, while in other cases sea-air and sea-baths are successful competitors of the water-cure.

*Hysteria*.—In hysteria and hysterical affections the water-cure has obtained many good results, not by the internal use of water, but by the milder forms of baths. Functional hyperæsthesia and anæsthesia, hemispheres, spinal irritation, intercostal neuralgia, and other forms of neuralgia depending on imperfect nutrition and tissue-change, are likewise often benefited.

*Organic diseases of the nerve-centres* are not suitable for treatment in cold-water

establishments, excepting occasionally for palliative purposes.

*Rheumatism and gout*.—In *muscular rheumatism* the original supporters of the water-cure considered their plan as infallible, but this is by no means the case. The diaphoretic methods, namely, the woollen blanket-pack and the wet sheet-envelope, often prove useful; but we know also of many failures in even good establishments. The exposure to all weathers during the cure ought certainly not to be imitated by such invalids, and the access of cold air to the wet body is to be more carefully avoided than it often is. The course must not be prolonged too much at one time, but may be repeated after an interval of months, which may be spent with advantage at sheltered seaside localities, at moderate elevations, with the help of pine-leaf baths, or at one of the gaseous thermal saline spas.

*Rheumatic and gouty swellings* of joints require great care in their management. The enfeebled invalid is rarely a fit object for the ordinary water-cure; but the stimulating local compress, more or less impermeable, is a useful element in the treatment of such cases.

Milder cases of *gout* may expect benefit from the usual hydrotherapeutic treatment, in so far as it aims at increased retrogressive tissue-change, and invigoration of the nervous system, especially if this treatment is associated with great moderation in the use of stimulants, and also of food in general; but local packing not rarely causes fits of gout. The more serious forms of gout are too much complicated with various defects of constitution to encourage us in recommending cold-water treatment.

*Chronic affections of the skin*.—In some diseases of this kind, such as prurigo, urticaria, eczema, and local perspirations, a more or less modified hydrotherapeutic treatment is an important adjuvant.

*Syphilis*.—The favourable results obtained in syphilis, or rather in the often complicated conditions of *lues*, have greatly contributed to the reputation of the water-cure; but the latter is only an excellent adjuvant to medicinal treatment in these cases, in a similar way as the sulphur waters are; and many of the cures of so-called *lues* may be regarded as cures of *mercurialism*.

*Catamenial irregularities* are not rarely treated at hydrotherapeutic establishments. Profuse menstruation is often checked by the regular use of the cold hip-bath of short duration, namely, three to five minutes; in insufficient menses, on the other hand, warm hip-baths of ten to fifteen minutes' duration are frequently useful, combined in some cases with the wet sheet-envelope; and dysmenorrhœa is likewise occasionally treated with advantage by the partial wet sheet-envelope.

This list of morbid conditions which may be more or less benefited might easily be increased. This is not astonishing if it is considered that hydrotherapeutic treatment can be infinitely modified and adapted to the powers of the constitution; and that it may be assisted by varying hygienic, climatic, dietetic, and medicinal influences. For we must reiterate that there does not exist any antagonism between hydrotherapeutic and other rational treatment, the former being, in fact, only part of the latter. Hence, however, it is also evident that the treatment in well-arranged hydrotherapeutic establishments ought to be under the guidance of the most intelligent physicians, just as is the case at all the best spas. Indeed the physician to such an establishment ought to be of a high class, possessing in a more than usual degree the gift of recognising all the individual peculiarities of the constitution, especially the amount of reacting power, adapting the principal remedy to every individual case, and combining other elements of treatment with hydrotherapeutic management wherever this is necessary. In the same way as we demand in suitable cases the administration of other remedies together with water-treatment in hydrotherapeutic establishments, so we must also express a wish that, apart from such establishments, hydrotherapeutic elements should be more generally combined with the usual medical treatment. For this purpose it is to be desired that well-conducted establishments should be in or near large towns, in order that persons following their usual occupations might undergo certain kinds of treatment at such establishments, or that attendants from such establishments might be sent to the houses of invalids.

HERMANN WEBER.

**HYDROTHORAX** (*ὑδωρ*, water; and *θώραξ*, the chest).—Dropsy of the pleura. See PLEURA, Diseases of.

**HYDRURIA** (*ὑδωρ*, water; and *οὔρον*, urine).—A profuse flow of watery urine. See URINE, Morbid Conditions of.

**HYÈRES, France**.—Town three miles from the sea. Mean winter temperature, 55° F. Well sheltered; little rainfall. See CLIMATE, Treatment of Disease by.

**HYGIENE** (*ὑγίεια*, health).—The science and art which relate to the preservation of health. See PERSONAL HEALTH; and PUBLIC HEALTH.

**HYPÆMIA** (*ὑπό*, under; and *αἷμα*, blood).—Deficiency of blood in a part; a synonym for local anæmia. See CIRCULATION, Disorders of.

**HYPÆSTHESIA** (*ὑπό*, under; and *αἴσθησις*, sensation).—Diminished sensibility of a part. See SENSATION, Disorders of.

**HYPERÆMIA** (*ὑπέρ*, over or excessive; and *αἷμα*, blood).—Excess of blood in a part. See CIRCULATION, Disorders of.

**HYPERÆSTHESIA** (*ὑπέρ*, over; and *αἴσθησις*, sensation).—Increased sensibility of a part. See SENSATION, Disorders of.

**HYPERALGESIA** (*ὑπέρ*, over; and *ἄλγος*, pain).—Undue sensibility of a part to painful impressions. See SENSATION, Disorders of.

**HYPERIDROSIS** (*ὑπέρ*, excessive; and *ἰδρῶς*, sweat).—Excessive perspiration; also termed *idrosis*, *ephidrosis*, and *sudatoria*. See PERSPIRATION, Disorders of.

**HYPERINOSIS** (*ὑπέρ*, over; and *ἴς*, *ἰνός*, flesh).—Excess of fibrin in the blood. See BLOOD, Morbid Conditions of.

**HYPERMETROPIA** (*ὑπέροτροπος*, beyond measure; and *ὄψ*, sight).—A congenital or acquired error of refraction of the eye, in which, owing to low refractive power of the dioptric media, or too little convexity of the refracting surfaces, or unnatural shortness of the antero-posterior axis of the eyeball, parallel rays of light do not, while the accommodation is in repose, converge to a focus on the layer of rods and cones of the retina, as in the normal or emmetropic eye, but to an imaginary point somewhere behind. It is the opposite of myopia, and is sometimes called hyperopia or hyperpresbyopia. See VISION, Disorders of.

**HYPEROPIA** (*ὑπέρ*, above; and *ὄψ*, sight).—See HYPERMETROPIA.

**HYPERPLASIA** (*ὑπέρ*, over; and *πλάσσω*, I mould or form).—An excessive growth of normal tissue-elements, which may lead to hypertrophy, or to the formation of distinct tumours. See HYPERTROPHY; and TUMOURS.

**HYPERPRESBYOPIA** (*ὑπέρ*, above; *πρέσβυς*, old; and *ὄψ*, sight).—See HYPERMETROPIA.

**HYPERPYREXIA** (*ὑπέρ*, excessive; and *πυρεξία*, fever).—Excessive pyrexia. See FEVER; and TEMPERATURE.

**HYPERTROPHY** (*ὑπέρ*, over; and *τροφή*, nourishment).

DEFINITION.—The word 'hypertrophy' signifies excessive nourishment, but is in practice used to designate the result of excessive nourishment—that is, excessive growth. It must be carefully distinguished from mere enlargement, and only spoken of where there is a real increase in a part, or, at least, in a tissue, without alteration of quality. Hypertrophy may be *general* or *partial*.

I. General Hypertrophy. — General

hypertrophy, though a remarkable condition, is of little practical importance. It is known only in those individuals of enormous size who are called 'giants.' The production of giants depends on causes entirely unknown, since it is noticeable that this condition commonly affects only one in a family, and is in its most conspicuous forms not hereditary. Giants are usually of feeble constitution, and deficient in procreative power. The name *macrosomatia* has been given to a condition equally unexplained, in which the whole body becomes enlarged in a monstrous degree. This condition has been observed to be in some instances congenital, or, at least, to begin in very early life. True general hypertrophy does not appear to be capable of being produced by any artificial means, since excessive feeding either produces hypertrophy almost confined to one tissue—namely, fat—or else fails to produce any enlargement at all.

**II. Partial Hypertrophy.**—By this is meant (a) excessive increase of any part of the body during the period of natural growth, either in intra-uterine or extra-uterine life; or (b) increase of a part already completely formed. According to this distinction hypertrophy may be classified as either (1) *congenital* or (2) *acquired*.

**1. CONGENITAL HYPERTROPHY.**—Congenital hypertrophy is that condition in which some part of the body begins from the first to grow so rapidly as to attain a size far beyond the normal. This condition has been seen to affect one side of the body, or one limb only, which thus becomes much larger than its fellow on the other side. Such a condition might be in theory difficult to distinguish from atrophy of the opposite side, or of the other limb—that is, from hemiatrophy (*see* ATROPHY); but in general the hypertrophic side is so far beyond the normal size as to prevent ambiguity. One remarkable case is on record in which one leg and arm assumed the proportions of those of a giant, whilst the other remained unaltered. Sometimes a congenital hypertrophy occurs without this unilateral character, as in the case reported by Mr. Curling of a girl aged fifteen, who had several fingers of both hands enlarged in an extraordinary degree without any assignable cause, the equality of the two sides being nevertheless preserved. Such instances, although unexplained, must, it would seem, be put into the same class as the gigantic growth of the whole of the body. Hypertrophy of special tissues is also sometimes congenital, as of the skin in ichthyosis. Hypertrophies similar to those here called congenital may occur, though rarely, in adult life. The enlarged part is found to be highly vascular, to have an increased temperature, and to preserve its normal proportions. The cause is in these cases equally unknown.

**2. ACQUIRED HYPERTROPHY.**—Acquired hypertrophies may be classified as follows:—

(a) *Recuperative*, consequent on increased work.

(b) *Defensive*, arising from pressure or inflammation.

(c) *Nutritive*, from increased supplies of food.

(d) *Apparently spontaneous* or of unknown cause.

(a) *Recuperative.*—Some organs and tissues are so constructed that increased functional activity causes hypertrophy. This is especially the case with the muscles. It is a matter of familiar observation that voluntary muscles increase in size when much employed, as is seen in the often-mentioned arm of the blacksmith or the leg of the ballet-dancer. In order to produce this increase, the exercise must be of a certain degree of intensity, but not excessive. It must be frequently repeated, with intervals of rest; and at the same time the nutrition of the whole body must be good. In the absence of these conditions, exercise is more likely to produce wasting. The explanation of this familiar process is still obscure.

Hypertrophy of the heart occurs in cases where that organ is made to work at a higher tension than the normal, and this higher tension can only result from increased resistance to the flow of blood, either at one of the orifices of the heart, or in the peripheral vessels. Hence the conditions most commonly giving rise to it are valvular disease, especially stenosis; and obstruction of the arteries, either by the thickening of their walls, or by contraction of their muscular coats. The right side of the heart will also become hypertrophied when any condition whatever hinders the passage of blood through the lungs. Disease of the kidneys is a frequent cause of enlargement of the left ventricle of the heart, though in what way is still a matter of discussion. It is only quite clear that the kidney-disease in some way increases the resistance in the smaller arteries and capillaries of the body generally.

Hypertrophy of the smooth or involuntary muscular fibres also occurs whenever that tissue has to contract for a long period under a higher tension than the normal. Thus the walls of the bladder become thickened in cases where, from obstruction of the passages, the evacuation of urine is effected with more difficulty, and under a higher pressure, than usual. In the same way the walls of the stomach, the œsophagus, and the intestines become hypertrophied in cases of obstruction to the passage of food through the alimentary canal.

The explanation commonly given of these cases of hypertrophy in the contractile organs is, that in consequence of obstruction the organ has to contract with greater force than usual, and thus becomes hypertrophied in

the same manner as a voluntary muscle which is frequently exercised. It is, however, clear that this so-called explanation does not account for the connexion between obstruction and more powerful contraction. The only explanation that can be given is that in these cases pressure or tension on the organ itself is the stimulus to contraction, and that the force of contraction appears to depend roughly upon the strength of the tension which produces it.

There is less evidence that nervous or glandular structures undergo hypertrophy in consequence of their increased use; but if one kidney be destroyed the other is generally found enlarged; and some authorities believe in an increase in the size of the brain from mental activity.

(b) *Defensive hypertrophy* is the natural reaction of certain tissues against slight injury or irritation, whereby they become increased and serve the purpose of protection. But if the injury be excessive, atrophy or destruction of tissue results.

*Pressure*, which is a mild form of irritation, produces, when moderate and intermittent, hypertrophy of the external integument; but some regions of the skin are much more affected by this cause than others. Besides the epidermis, the coverings of certain internal organs sometimes show the same phenomenon, as the capsule of the liver from pressure of a belt, and certain parts of the pericardium where friction occurs. The defensive character of these processes is obvious.

*Inflammation* does not cause hypertrophy of organs generally, but only of tissues, and among these only of the connective tissues. In most tissues, e.g. muscular, nervous, glandular, the only result of inflammation is atrophy or destruction, with loss of functional power. But it seems to be a general law that moderate inflammation, chronic or intermittent, of connective tissue causes it to increase. Purely fibrous structures, such as serous membranes, become thickened by chronic or repeated inflammation of moderate intensity, especially if suppuration be not produced, but this increase is not always permanent. In peritonitis the formation of new bony tissue causes the increase to be permanent, and thus hypertrophy of bone is a frequent consequence of inflammation. The increase of connective tissue from irritation is obviously a conservative or defensive process, placing a barrier to the extension of injurious agents; and forming a capsule round foreign bodies.

Increase of connective tissue is also constantly present in chronic interstitial inflammation of solid organs, where indeed it is often impossible to draw the line between inflammation and fibrous overgrowth. These changes, being produced by some irritation or injury, illustrate the law of the depend-

ence of increase of connective tissue upon such causes; but since there is generally simultaneous or consequent atrophy of the other tissues of the organ, there is likely to be rather a diminution than an increase in the size of the organ or part generally.

(c) *Nutritive hypertrophy*.—It must be regarded as doubtful whether increased nourishment alone is capable of producing enlargement of any part of the body. It certainly does not necessarily do so, as is shown by the case of experimental hyperæmia. When this condition is produced, for instance, by section of the cervical sympathetic nerve, in one side of the face and head of an animal, hypertrophy is only a rare and occasional consequence. When, however, the increased supply of nutrition in the form of blood is combined with some irritation or functional stimulus, we often find hypertrophy result. Thus, for instance, reflex hyperæmia of the skin of the face, or blushing, which is produced by numerous internal causes, such as gastric or uterine derangement, may subsist for years and reach a very high degree without altering the nutrition of the part. But if there should be in addition some disturbance or inflammation of the glands of the skin, we have the conditions called *acne rosacea*, &c., in which hypertrophy is an important element. Almost the only instance that can be quoted of hypertrophy from increase of blood-supply alone is that of the corpus luteum during pregnancy, when the ovary participates in the functional hyperæmia of the uterus. The well-known experiment of Hunter should also not be forgotten, in which he transplanted the spur of a cock from its foot to its head, and found it to increase in size.

It is, however, important to remember that hypertrophy, however produced, is always accompanied by an increased supply of blood, and enlargement of the vessels.

*Special nutrition*.—It is well known that abundant supplies of amylaceous and fatty food, especially with a full allowance of water, cause hypertrophy of the adipose tissue. It is also said that small doses of phosphorus favour the growth of bone in young animals. But there is no other instance in which it is known that any special food causes hypertrophy of any special tissue.

(d) *Apparently spontaneous or physiological hypertrophy*.—Physiological hypertrophies form an important class. One of the best instances is that of the enlargement of the uterus during pregnancy. This enlargement is clearly not the consequence of hyperæmia alone, nor of increase of the functional activity in the muscular walls, though both these conditions are present; but must proceed from some direct physiological stimulus like that which determines the growth of the embryo itself. The hypertrophy affects all parts of the organ—its

mucous and serous coats, as well as the muscular walls. Enlargement of the mamma appears to arise from similar causes; and it is even probable that swelling of the thyroid may, through some obscure connexion with the sexual organs, be caused in the same way. Some instances of hypertrophy we cannot in any way explain, such as the apparently spontaneous enlargements of the tonsils, spleen, and thymus gland which are sometimes observed. These changes have been explained by a supposed derangement of the so-called *trophic nerves*, but this explanation only puts the difficulty a little further back.

**Process of Hypertrophy.**—It has been a question whether hypertrophy depends upon the increase in the size of the minute elements of an organ, or only on increase of their number. There can be no doubt that the former change often occurs. Thus, in the pregnant uterus the muscular fibres have been found from seven to eleven times as long as natural, and from twice to seven times as wide. In a remarkable case of enlargement of the nerves, described by the late Dr. Moxon, the nerve-fibres were found to be on an average three times and some of them even forty times as large as normal. When enlargement of one kidney takes place as a consequence of destruction of the other, the tubules and Malpighian tufts are found greatly increased in size. In hypertrophy of the heart, the muscular bundles are found to be thickened, though the fibrillæ are unchanged; but in most cases multiplication of the tissue-elements is the chief cause of the increase in size. To this latter process Virchow gives the name of *hyperplasia*, and it is important to remember that, though constantly occurring in hypertrophy of organs, it does not necessarily lead to the latter change.

**'False' Hypertrophy.**—The term *false hypertrophy* is sometimes used for a process in which an organ becomes outwardly increased in size, owing to the deposition within it of some foreign material, or to mere distension. Thus in a fatty liver the real liver-tissue is wasted, but is replaced by fat. An emphysematous lung appears to be increased in size, but has actually suffered atrophy. The substance of the brain in hydrocephalus may be greatly reduced in volume, though the head appears of enormous size. The very remarkable disease called 'Duchenne's paralysis' is an instance of apparent hypertrophy of the muscles, through deposition of fat between the fibres, but the term is hardly needed, except as a caution.

**TREATMENT.**—It is obvious that no general rules can be laid down for the treatment of hypertrophy. When it is connected with increased functional activity, it is usually a favourable rather than a hurtful condition, though in some cases it may appear that the hypertrophy more than compensates the de-

ficiency or irregularity by which it is produced. But even if this be so, the cure of excessive hypertrophy is not within our powers. The utmost that can be done is to endeavour, if possible, to check the process by which the hypertrophy is produced. It is, however, well to point out that when functional hypertrophy has resulted from some obstacle or undue resistance, it may entirely subside when that resistance is removed. Thus if the uterus have enlarged around a fibrous tumour, it may regain its normal bulk when the tumour is removed; and we sometimes see a sensible diminution in the size of a hypertrophied heart when the derangements which produce it no longer act. Moreover, hypertrophy may be completely reduced by a general lowering of the nutrition of the body. Thus, in early stages of pulmonary phthisis, the heart may be hypertrophied; but when death occurs in a late period of the disease, the organ is rarely found enlarged, and is even wasted; though, according to Dr. Peacock's tables, less so than in other wasting diseases.

J. F. PAYNE.

**HYPINOSIS** (*ὑπός*, under; and *ἴς*, *ἰνός*, flesh).—Deficiency of fibrin in the blood. See BLOOD, Morbid Conditions of.

**HYPNOTICS** (*ὑπνος*, sleep).—Measures or agents employed to induce sleep. See NARCOTICS.

**HYPNOTISM** (*ὑπνος*, sleep).—A synonym for Braidism. See BRAIDISM; and MESMERISM.

**HYPOCHONDRIAC REGION** (*ὑπό*, under; and *χόνδρος*, a cartilage).—This region is double, right and left, occupying the upper part of the abdomen on either side of the epigastrium, and partly corresponding to the lower regions of the chest, being almost, and in some cases entirely, covered in by the ribs and their cartilages. Each hypochondrium is bounded below by a horizontal line at the level of the ninth costal cartilage; and internally by a vertical line from the eighth cartilage downwards. The organs situated in the right hypochondrium are the liver and gall-bladder mainly; with, more deeply, the pyloric end of the stomach, part of the duodenum, and the hepatic flexure of the colon. In the corresponding left region lie the spleen, a small portion of the left lobe of the liver, the fundus of the stomach, the tail of the pancreas, and the splenic flexure of the colon. The gastro-splenic fold of peritoneum, with its vessels, passes from the stomach to the spleen.

**CLINICAL INVESTIGATION.**—It must be borne in mind that morbid conditions within the chest not uncommonly originate clinical phenomena, both subjective and objective, in connexion with one or other hypochondriac region, and these must always be taken into consideration when investigating any parti-

cular case. In making a diagnosis, no reliance whatever can be placed on mere sensations referred to these regions, but physical examination is in every instance required, in order to determine the conditions present, and especially palpation and percussion. Moreover, it must be remembered that disease of an important and serious nature may arise without the occurrence of any unusual feelings. Pain of various kinds is often complained of, and when referred to the right hypochondrium is usually supposed to be connected with the liver; but it may depend upon affections of the superficial structures; peritonitis; right pleurisy or pneumonia; or conditions associated with the gall-bladder, pylorus, duodenum, or colon. A characteristic pain starting from this region is that of hepatic colic, usually due to the passage of a gall-stone along the ducts. Persons who are hypochondriacal not uncommonly refer some abnormal sensation to their right hypochondrium, for which there is really no obvious cause. In the left hypochondrium pain may also be due to affections of the walls, or of structures within the thorax; of the cardiac end of the stomach; of the colon; or, in exceptional cases, of the spleen.

The morbid conditions which are capable of originating abnormal physical signs in the hypochondriac region may also be either thoracic or abdominal. Of the former, pleuritic effusion is the most frequent; and on the right side this condition may cause marked depression of the liver, as also may pneumothorax. In exceptional instances the heart is so enlarged as to reach the left hypochondrium; and the writer has met with a case in which a large thoracic aneurysm presented in this region. As regards abdominal diseases occupying the right hypochondrium, abnormal physical signs are usually associated with the liver, which is altered in position, shape, size, or physical characters. Occasionally they are connected with the abdominal walls; the gall-bladder; or the stomach, duodenum, or colon. On the left side enlargement of the spleen is the main condition discovered by physical examination; but the stomach may also give signs of distension in this direction, or of organic disease of its walls. Exceptionally the colon may present abnormal physical signs. Growths may originate here in connexion with the peritoneum; and the writer has met with a case in which a growth started from the left hypochondrium, probably of a malignant nature, and involving more than one structure, but it was impossible to say where it commenced. Of course the hypochondria are involved along with other regions in general enlargement of the abdomen; and organs from other regions may so increase in size in certain diseases as to extend into one or both of these regions.

FREDERICK T. ROBERTS.

**HYPOCHONDRIASIS** (*ὑπό*, under; and *χόνδρος*, a cartilage).—**SYNON.**: Fr. *Hypochondrie*; Ger. *Hypochondrie*.

**DESCRIPTION.**—The term 'hypochondriasis' is derived from an ancient hypothesis that the symptoms of this disorder were due to perturbations of natural force generated in the liver and pylorus, to which idea the frequent prevalence of flatulence in the disorder conduced. The condition thus called is really a disease of the nervous system. It is a form of mental unsoundness closely allied to melancholia, of which, indeed, it often forms the initiatory stage. It is characterised by a morbid anxiety, either without any, or having only very slight, foundations, relative to the state of physical health. The patient thinks about his health unduly, observing himself with restless care, examining especially the characteristics of his secretions, translating into evidence of progressive organic mischief every trivial departure from perfect action of his organs, and becoming more and more absorbed in precautions against the malady with which he believes himself affected. Nothing that happens tends to the side of reassurance. If his sleep be disturbed, the symptom may be portentous, he thinks, of brain-softening; if it be sound, the patient, instead of being comforted, fears apoplexy. Constipation of the bowels signifies obstruction; a slight diarrhœa implies coming exhaustion. Everything which he reads or hears in reference to disease, the patient applies to his own case, examining himself on every point thus presented to his mind, and rarely failing to find something which dovetails with symptoms of his own. For he recognises only the points of resemblance; the features of difference are unconsciously ignored. In some cases the patient is constant in referring his troubles to one particular organ. Year after year his story is the same: it is his stomach, liver, brain, or some other organ, which is in fault; but always the same. In other instances there is a vacillation quite as remarkable. Routed, perhaps, by the convincing arguments of his adviser, he is forced to yield the position which he had assumed, but only to take an equally strong one in reference to some other part of his frame. These diversities strongly recall the fixed and shifting delusions of insanity. The patient is prone to wander from one doctor to another, often carrying with him a bundle of prescriptions and a long written list of questions, which must receive categorical answers. Apparently satisfied at the time, he speedily recollects some point upon which he has not received assurance, and thus he conceives vitiates the whole of the explanation and advice which have been given to him, and he is plunged again into his previous state of anxiety and doubt. Where circumstances do not involve forced labour for existence, the patient passes his time in chasing his health,

which is always contriving to elude his grasp. If he holds an appointment, he will resign it in order to have full opportunity for studying himself; and his occupation once gone, he finds too late that it was his best friend, and he then ascribes to his forced idleness all the ills which had induced him to seek retirement.

In many cases the most careful examination can discover no signs of disease, and the patient wears the aspect of health; or there may be a worn, anxious look. In others there may be, especially in the digestive organs, slight deviations from perfect integrity, which explain some of the symptoms, but not the exaggerated apprehensions to which they give rise.

Hypochondriasis is a chronic disorder. It may continue, and this most frequently, as a harmless peculiarity attached to a life which is not perceptibly shortened in length, though often sadly diminished in utility and happiness, by its symptoms. Or there may be an improvement practically amounting to a cure, which will endure for a longer or shorter period. In the decline of life, however, there is very apt to be a return of symptoms. Or hypochondriasis may pass into true melancholia, and then the bodily health, previously the constant object of solicitude, improves wonderfully. Indeed, nothing more is heard about it.

**ETIOLOGY.**—Hypochondriasis is very much more common in the male than in the female sex. The period of life most prone to it is from 20 to 40 years of age. It is apt to occur in those who inherit a tendency to insanity, and the disease in its own peculiar form is often hereditary. Excesses of various kinds, especially on the side of the sexual system, will precipitate the appearance and intensify the symptoms of the affection, but it is doubtful whether they can altogether originate it. The same may be said of gout, which is apt to be associated with the condition and to complicate its symptoms. Depressing moral circumstances also are not without influence in determining an onset of hypochondriasis in those predisposed to it. This is especially true of mental strain. The frequent occurrence of some deviation from healthy condition in the liver, stomach, or bowels, which is noted in these cases, would suggest that, probably through an interruption to the perfect nutrition of the body, diseases of these viscera bear their part in the causation of hypochondriasis. The intercurrent of internal hæmorrhoids with bleeding is very common, and this would manifestly tend to keep up, if it did not originate, the disease. Stricture and chronic ulcer of the intestines are occasionally associations which probably also influence the appearance of hypochondriasis.

**ANATOMICAL CHARACTERS.**—There are no anatomical characters peculiar to the disease.

**DIAGNOSIS.**—When careful examination, which must never be omitted, has disproved the existence of organic disease tending to produce the symptoms described by the patient, it sometimes becomes a question whether the case is one of hypochondriasis or of melancholia.

In the former there is no tendency to suicide; on the contrary, a strong desire to live pervades the sufferer's mind, and impels him to endless search for the cure of his ailments. He delights in consulting medical men and entering into the minutest details which he thinks can aid them in helping him. Up to a certain point his story is frequently characterised by a logical accuracy, which fails him, however, in some point of great importance, by which the conclusions are invalidated. The melancholic patient, on the other hand, is often suicidal and always despairs of any relief to his condition, the description of which, as given by him, is confused, frequently incoherent, and unintelligible.

**PROGNOSIS.**—Early and marked hypochondriasis occurring in a person with a strong hereditary taint of insanity, without any definite cause of mental depression, is of ill omen. Such a case very often drifts into melancholia.

The prognosis is more favourable, perhaps, the less strongly marked the hereditary predisposition and the more evident and adequate the immediate causes to which the patient has been exposed, the most potent of which are sexual or alcoholic excesses, mental strain or shock, or the sudden change from a life of activity to one of forced and, as the sequel shows, uncongenial leisure.

**TREATMENT.**—Moral treatment is alone of any influence in a large majority of cases. Where there is, however, manifest anæmia, a history of syphilis, evidences of gouty mal-assimilation, accumulation of fæces, catarrh of the intestinal canal, or hæmorrhoids, the therapeutics proper to these conditions should be employed. Alcoholic stimulants should be avoided. Travel, especially under judicious companionship, and the encouragement of regular, definite, and useful employment for the attention and the physical powers, are the most potent means of treatment, by which the disease may be often much ameliorated, and sometimes cured. Ridicule of the patient's sufferings will rarely or never be of service, but at the same time a habit of prescribing for all the symptoms as they arise must be avoided.

T. BUZZARD.

**HYPODERMIC MEDICATION** (*ὑπό*, under; and *δέγμα*, the skin).—Fr. *La Méthode Hypodermique* or *Sous-cutanéë*; Ger. *Subcutane Arzneiapplication*; *Hypodermatische Injection der Arzneimittell*.

The introduction, by means of a sharp-pointed hollow needle, of alkaloids and

other agents into the subcutaneous cellular tissue.

**HISTORY.**—Hypodermic injections were first employed by physiologists in experimental researches. John Hunter appears to have used them in this way, and they were certainly so employed as far back as 1819 by Magendie. Valleix made a crude attempt to apply sedatives subcutaneously in the treatment of neuralgia by means of acupuncture-needles; but, according to Bartholow, it was not till 1839 that the method was regularly employed in the treatment of disease, when Taylor and Washington in America made use of it, the idea being suggested to their minds by Lafargue's method of inoculation. Sieveking claims that Kurzak of Vienna was really the discoverer of hypodermic medication; but, be this as it may, it was undoubtedly the late Dr. Alexander Wood of Edinburgh who brought this method of treatment into general use.

**METHOD OF APPLICATION.**—This is a matter of great importance. With due precaution, the pain in most instances is insignificant. In the case of some drugs, such as ergotone, more or less pain is inevitable, but by careful manipulation the unpleasant effects may be reduced to a minimum. The needle, which must be sharp and smooth at its point, is attached to the nozzle of the syringe, and the latter is charged with the drug to be employed. If a tabloid is used, it is dissolved in a syringeful, or less, of warmish water; if, on the other hand, a solution of the drug is used, the syringe should be completely filled, and the quantity to be injected accurately gauged by moving the button down to the required mark on the piston. By either plan the possibility of overdosage is guarded against. A fold of skin is pinched up between the left thumb and index finger, and the needle is pushed firmly and sharply into the cellular tissue. The barrel of the syringe is then steadied between the right index and middle fingers, and the fluid slowly and steadily injected by pressure with the right thumb on the end of the piston. As the needle is withdrawn, the left index finger should be applied to the point of puncture for a few seconds. In the case of ergotone and a few other irritating drugs it is well to modify this mode of procedure by making the injection directly into the muscular tissue: in uterine hæmorrhage, into the gluteal muscles.

To prevent any chance of sepsis, some recommend that the needle before being used should be dipped into carbolised oil. *Local ill effects* consist chiefly in pain, phlegmonous swellings and consequent abscess, slight bleeding, or ecchymosis; but with proper precautions most of these accidents may be avoided, and in nearly all cases the pain is both trifling and evanescent. Certain parts of the body should always, if possible, be

avoided in making hypodermic injections—*e.g.* regions where the subcutaneous cellular tissue is scanty, such as the skin over the tibiæ, parts such as the cheeks, forehead, ears, &c. It is well also to avoid the neighbourhood of large vessels. *General ill effects* may easily be produced from injections, especially of narcotics, in diabetics, alcoholics, and patients suffering from renal disease.

*Solutions* should be made with vehicles which fulfil the following conditions: (1) They should have as high a solvent power as possible. (Some substances, such as calomel, can only be injected in a state of suspension.) (2) They should, as far as possible, be permanent, not deteriorating by keeping. (3) They should be unirritating. Cherry-laurel water, carbolic acid, boric acid, camphor, and other substances, may in some cases be used to prevent the development of organisms. Paroleine (one of the petroleum series) is the best vehicle for the injection of antiseptics, such as carbolic acid, eucalyptol, iodoform, turpentine, &c. Tabloids and gelatine discs are convenient forms for use in preparing solutions.

**INSTRUMENTS.**—For ordinary hypodermic use a simply constructed and accurately graduated instrument is requisite. The capacity of the barrel should be twenty minims. The syringe may conveniently be had in a small case, with two needles, and a supply of silver wires to keep their canals clear. The *needles* are best made of golden platinum, which is both tough and non-corrosible. It is of great consequence that the points should be sharp and with smooth edges. The *nozzle* of the syringe, on which the needle is fitted when required for use by a simple sliding motion (not by a screw), should always be provided with a metal cap. This prevents drying and shrinking of the piston packing. The *packing* should consist of leather or wash-leather: and if by any chance it does not fit tightly, the top of the syringe should be unscrewed and removed, with the piston, and the packing is then to be well soaked in paroleine. The metal parts must not be attached to the barrel by cement, as this material is destroyed by ether and other fluids, and the syringe is consequently rendered utterly useless. The minims are best marked on the *piston*, which should have a screw-thread on which a button runs: by this means the dose is gauged accurately before the injection is made, and there is, therefore, no possibility of injecting more than was intended. The piston should have a simple sliding, and not a screw, action. It is terminated at the top by a flat knob, which is pressed with the thumb when the injection is made. For the injection of ether, alcohol, antiseptics, nutrients, saline solutions, &c., much larger syringes may be used to avoid multiple injections; such syringes are usually of a capacity of eighty to one hundred minims.

It is almost superfluous to add that hypodermic syringes must be kept scrupulously clean. After use, tepid water, to which an antiseptic may with advantage be added, should be drawn into the syringe and ejected through the needle. The latter should then be blown through, to expel any remaining fluid, and a silver wire is then passed through and left in its canal to keep it clear. The barrel of the syringe should consist of glass. Celluloid has been employed for this purpose, but it is soluble in ether and alcohol, and should therefore be avoided. The cap, nozzle, and piston should consist of nickelled metal or silver.

**ADVANTAGES.**—The advantages of the hypodermic method are numerous, and difficult to over-estimate; but it must be remembered that it has its limitations. There is, for instance, no useful end gained by the hypodermic injection of such drugs as Epsom salts, or of such powerfully irritating and depressing principles as colchicine and cantharidine. It is further important to point out that the hypodermic administration of such drugs especially as morphine and cocaine should be under the direct control of the medical man, and should in no case be entrusted to the patient. The advantages of the hypodermic, as contrasted with other methods of administering drugs, may be briefly summed up as follows: (1) There is greater rapidity of absorption, and consequently more speedy effect. (2) A rapid local as well as general effect can be more easily obtained. (3) Drugs can be administered in this way when other methods are impossible—*e.g.* in cases of vomiting, nausea, and other conditions originating in an irritable or diseased stomach; where there is mechanical difficulty in deglutition; and in certain cases of delirium, mania, or coma. (4) According to Eulenburg, drugs are less liable to produce a cumulative effect when injected subcutaneously than when administered by the mouth. (5) In cases of narcotic or other forms of poisoning, emetics hypodermically injected often produce a rapid and certain effect, when administration by the mouth is inadmissible.

### Remedies used Hypodermically, and Diseases for which they are Employed.

*Acidum Arseniosum*—*Arsenious Acid*. See Arsenium.

*Acidum Benzoicum*—*Benzoic Acid*. Benzoic acid 10 grains; alcohol up to 112 minims. *Dose*, from 8 to 16 minims. *Used* in chest-diseases to stimulate the respiratory centre, and in the collapse of acute fevers and uræmia. The solution should be slightly warmed before injection.

*Acidum Carbolicum*—*Carbolic Acid*. Pure carbolic acid 1 or 2 grains; paroline, or water, up to 112 minims. *Dose*, 8, 16, or 24

minims. Has been used in erysipelas, malignant pustule, and other infective diseases.

*Acidum Scleroticum*—*Sclerotic Acid* (obtained from ergot). The solution easily decomposes, and should be made as required. Twenty minims of a 4 per cent. solution, twice or thrice in the twenty-four hours, recommended by Ziemssen in hæmoptysis. Tabloids of  $\frac{1}{2}$  to 1 grain are convenient for administration. The indications for the use of sclerotic acid are the same as those for ergotinin.

*Aconitina*—*Aconitine*. There are various aconitines, and these are of various strengths. Morson's is of uniform strength, and should be prescribed. The dose ranges from  $\frac{1}{260}$  to  $\frac{1}{60}$  of a grain. For a first dose the quantity should never exceed  $\frac{1}{130}$  of a grain: aconitine (English) 1 grain; diluted sulphuric acid q.s.; distilled water to  $\frac{1}{2}$  ounce. *Dose*, 1 to 4 minims (Martindale and Westcott). Tabloids ( $\frac{1}{260}$  and  $\frac{1}{130}$  grain) may also be used. *Uses*: In various forms of neuralgia, especially of the trigeminal. It has also been employed in cardiac hypertrophy, angina pectoris, intermittent and puerperal fevers; but in such cases it is preferable, as a general rule, to administer the tincture by the mouth.

*Æther*—*Ether*. Sulphuric ether is generally used for hypodermic purposes, but acetic ether may also be so used. Ether may also be injected in combination with alcohol; or camphor, 1 to 2 parts dissolved in sulphuric ether 10 parts, may be administered. *Dose* of sulphuric ether 20 to 40 minims, repeated as required (*see* remarks on Syringes). *Uses*: In collapse of any kind, in the exhaustion of typhus and other fevers, pneumonia, &c.

*Alcohol* in the form of brandy is very useful hypodermically in cases of collapse. *Doses* of half a fluid drachm to a drachm may thus be given at a time and repeated as required. It causes little pain.

*Ammonia Liquor*—*Solution of Ammonia*. *Dose*, 2 to 5 minims hypodermically or intravenously. The latter method is of service in syncope from any cause, and in threatened death from embolism.

*Amyl Nitris*—*Nitrite of Amyl*. Nitrite of amyl 1 part; alcohol 9 parts. *Dose*, 8 to 12 minims. *Used* in threatened death during chloroform anæsthesia, in puerperal convulsions, and in irregular (hour-glass) uterine contractions. Also in strychnine poisoning; and, especially, in angina pectoris.

*Apocodeine Hydrochlorate*. Recommended by Dujardin-Beaumetz as an emetic. *Dose*,  $\frac{1}{8}$  to  $\frac{1}{2}$  of a grain in 16 minims of distilled water.

*Apomorphinæ Hydrochloras*—*Apomorphine Hydrochlorate*. *Dose*, tabloids  $\frac{1}{15}$  to  $\frac{1}{10}$  grain; or 2 to 8 minims of the pharmacopœial 2 per cent. solution in camphor water may be given. An over-dose may kill by

causing profuse bronchial secretion (Brunton). *Used* in various forms of poisoning as an emetic.

*Aqua—Water.* Large hypodermic injections of water, or of water with chloride and bicarbonate of sodium in solution, have been used in cholera, but intraperitoneal injections have been recommended in preference. Iced water has been used hypodermically to cure sciatica, and also in pyrexia. Hot water is sometimes useful in this way as an analgesic.

*Arsenium—Arsenic,* and its preparations. Arsenic causes slight local irritation. It may be used in the form of Fowler's solution (1 part to 2 parts of distilled water). It is of service in various nervous diseases—paralysis agitans, chorea, neuralgia, epilepsy, &c.; in skin diseases—lichen ruber, psoriasis, chronic eczema; in septic diseases—puerperal and typhoid fevers; in lymphoma, in various psychoses, and other diseases.

*Atropina—Atropine.* The neutral sulphate is the best salt. The solution used should be recent. Neutral sulphate of atropine 1 grain; distilled water up to 2 ounces, 160 minims (sixteen minims =  $\frac{1}{70}$  grain). It is well to begin with  $\frac{1}{150}$  grain. The prepared tablets  $\frac{1}{150}$ ,  $\frac{1}{100}$ , and  $\frac{1}{50}$  grain are useful. *Used* in neuralgia, asthma (when the mucous membrane is not dry), enuresis, spermatorrhœa, intestinal obstruction, epilepsy, tetanus, for the relief of pain generally, as an antispasmodic (especially in phthical sweats), and as an antisialagogue. Is of great service in combination with morphine. *See* Morphine, p. 912.

*Auri Chloridum—Chloride of Gold.* *Dose,*  $\frac{1}{35}$  grain in 20 minims of distilled water. *Used* in hysterical anæsthesia, and in syphilitic affections.

*Caffeina—Caffeine.* Caffeine 20 grains; salicylate of sodium 17½ grains; distilled water to 1 drachm (3 minims = 1 grain). *Dose,* 1 to 6 minims (Martindale and Westcott). Tablets of caffeine sodio-salicylate =  $\frac{1}{2}$  grain, may also be used. *Used* in malarial neuralgia and rheumatism, megrim, insomnia, chronic alcoholism, hysteria, cardiac dropsy (on account of its diuretic action), asthma, and in poisoning by opium and its alkaloids and compounds, alcohol, and cocaine.

*Calabarina, Physostigmatis Extractum—Calabarine,* and *Extract of Calabar Bean.* Calabarine is not used hypodermically, but eserine (physostigmine) and the extract have been so employed. The best preparation is that of Martindale and Westcott. Extract of Calabar bean 10 grains; rectified spirit 10 minims; rub together till smooth, and add gum acacia 10 grains; mix, and add gradually distilled water to ½ ounce. *Dose,* 3 to 12 minims. *Used* in tetanus, trismus neonatorum, strychnine poisoning, enuresis, &c.

*Calomel.—See* Hydrargyrum.

*Camphor.—See* Æther.

*Chloroformum—Chloroform.* Has been injected pure, in doses of 10 to 20 minims and upwards, as a substitute for morphine in neuralgia, lumbago, colic, cancer, &c.

*Cocaina—Cocaine.* One of the best, if not the best, of local anæsthetics. Injected hypodermically renders operations for hæmorrhoids, small tumours, &c., painless. To obviate toxic effects the re-crystallised salt should alone be used. The solution decomposes rapidly, and it should therefore be freshly prepared when required. Tablets of  $\frac{1}{2}$ ,  $\frac{1}{4}$ , and  $\frac{1}{2}$  grain are convenient. In addition to its use as a local sedative, cocaine has been employed in hemicrania and melancholia, to overcome the craving for morphine and alcohol, and to relieve various forms of neuralgia.

*Codeina—Codeine.* The hydrochlorate is the best salt for hypodermic use. *Dose,*  $\frac{1}{2}$  grain to 1½ grain. Codeine hydrochlorate 1 grain; distilled water up to 128 minims (16 minims =  $\frac{1}{3}$  grain). *Used* in diabetes mellitus; also in gastrodynia, insomnia, cancer, and asthma.

*Conina—Conine,* and its salts. Hydrobromate of conine 1 grain; distilled water 20 minims. *Dose,* 1 to 3 minims. *Used* in asthma, angina pectoris, pertussis, laryngismus stridulus, tic-douloureux, chorea, mania (Crichton Browne). Useless in tetanus and strychnine poisoning.

*Curara—Curare.* Is best given hypodermically. *Dose,*  $\frac{1}{10}$  grain in 1 per cent. filtered aqueous solution, or a tablet ( $\frac{1}{12}$  grain). *Used* to control the spasms of tetanus and hydrophobia.

*Daturina, Extractum Stramonii—Daturine* and *Extract of Stramonium.* *Dose,* of daturine,  $\frac{1}{100}$  grain; of stramonium extract,  $\frac{1}{16}$  to  $\frac{1}{2}$  grain. *Used* in emphysema, neuralgia, tetanus, and tremor (paralysis agitans).

*Digitalinum—Digitalin.* Must only be used in crystalline form; all other forms are impure and unreliable. *Dose,*  $\frac{1}{100}$  grain tablet. *Used* only exceptionally subcutaneously, in aconite poisoning, heart-diseases, spermatorrhœa, and exophthalmic goitre (Bartholow).

*Duboisina—Duboisine.* *Dose,*  $\frac{1}{350}$  grain. *Used* in exophthalmic goitre (Dujardin-Beaumez), phthical sweats (Gubler and others), and asthma.

*Ergotina—Ergotinine.* Ergotin is the watery extract of ergot. Ergotinine is the crystalline alkaloid. *Dose,* of Bonjean's ergotin 2 to 4 grains in glycerine and distilled water up to 20 minims; of ergotinine in tablet  $\frac{1}{300}$  or  $\frac{1}{150}$  grain. *Used* because of its action on involuntary muscular fibre—(1) to contract the uterus; (2) to constrict blood-vessels in hæmoptysis and other forms of bleeding. Should always be deeply injected.

*Ferrum—Iron.* The pyrophosphate of iron with citrate of sodium (2 grains in 20 minims), and ammonio-citrate of iron in

2-grain doses and upwards, have both been strongly advocated for hypodermic use in anæmia. Very rarely resorted to.

*Homatropina*—*Homatropine* hypodermically is less efficacious than atropine. *Dose*, in tabloid,  $\frac{1}{250}$  grain. *Used* to relieve cough, expectoration, and insomnia; also as an antidote to pilocarpine.

*Hydrargyrum*—*Mercury*, and its salts. The injection of the salts of mercury should only very exceptionally be resorted to, as the process is always painful and, without extreme care, is apt to result in inflammation and abscess. Where administration by mouth is, for any reason, undesirable, the endermic method by inunction with the oleates, or the oleates in combination with lanolin, is preferable to subcutaneous injection. The salts available for hypodermic use are the perchloride, the biniodide, the peptonate of mercury and ammonium, and calomel. If the perchloride is used, discs ( $=\frac{1}{18}$  grain of sublimate) or tabloids ( $=\frac{1}{60}$  and  $\frac{1}{30}$  grain of sublimate) are most convenient. If a solution is desired, the following formula may be used: Perchloride of mercury 3 grains; chloride of sodium and hydrochlorate of cocaine, of each 1 grain; distilled water up to 336 minims (16 minims  $=\frac{1}{2}$  grain of mercuric chloride).

The best solution of the biniodide is that of Martindale and Westcott: *Injectio hydrargyri iodidi rubri*, 1 in 64. *Dose*, 2 to 6 minims.

Calomel may be injected in doses of 1 or  $1\frac{1}{2}$  grain suspended in pure liquid vaseline or paroline, or Neisser's formula (modified) may be used: Calomel and sodium chloride, of each  $\zeta$ iv.; mucilage of tragacanth  $\zeta$ ij.; distilled water  $\zeta$ j ss.

*Hyoscina*—*Hyoscine*, and its salts. The effects of hyoscine are best produced by hypodermic use. The *dose* ranges from  $\frac{1}{200}$  to  $\frac{1}{25}$  grain. Tabloids contain  $\frac{1}{200}$  and  $\frac{1}{75}$  grain. The best solution is: Hyoscine 1 grain; distilled water 200 minims. *Used* in the treatment of delirium, excitement, and insomnia due to insanity and other diseases. Has been of service in the treatment of epilepsy and chorea. Of no use as an analgesic.

*Injectiones Hypodermicæ Nutrientes*—*Nutrient injections* are of very limited applicability. Almond, olive, and cod-liver oil may be thus employed. Sugar solution, egg albumen (alone or mixed with the yolk), milk, and defibrinated blood have all been injected subcutaneously, with more or less success. Blood-serum and salines have also been used. Milk has been injected intravenously, and sodium chloride intraperitoneally.

*Iodoform* may be given in solution in oil or ether, in doses of 1 grain and upwards. Iodoform 3 grains; sulphuric ether 15 minims; castor oil up to 32 minims. *Used* in scrofula, goitre, rickets, phthisis, &c.

*Morphina*—*Morphine*, and its salts. The

following salts are all more or less adapted for hypodermic use—acetate, tartrate, sulphate, hydrochlorate, and bimeconate. The pharmacopœial preparation contains the acetate. Morphine may be combined with cocaine or atropine: Cocaine hydrochlorate 3 grains; morphine hydrochlorate 1 grain; distilled water up to 224 minims—sixteen minims  $=\frac{1}{4}$  grain of morphine and  $\frac{1}{5}$  grain of cocaine. Atropine sulphate 2 grains; morphine acetate 40 grains; acetic acid 4 minims; water up to 1 ounce—twelve minims = 1 grain of morphine and  $\frac{1}{20}$  grain of atropine—Talfourd Jones. Morphine is used to relieve pain (as of neuralgia, gallstones, renal calculus, &c.), to relieve insomnia, to arrest nervous vomiting, in the treatment of diabetes, &c. It must be used with the utmost caution, and its administration should on no account be trusted to the patient. Small doses (of not more than  $\frac{1}{6}$  or  $\frac{1}{4}$  of a grain) should, as a general rule, be commenced with. Great care must be taken to avoid inducing the morphine habit (morphinomama).

*Muscarina*—*Muscarine* has been used by Ringer in doses of  $\frac{1}{5}$  to  $\frac{3}{4}$  of a grain. It may be employed in the night-sweats of phthisis, as an antagonist to atropine, and to arrest the secretion of milk. Its action is similar to that of pilocarpine, over which salt it possesses no advantages.

*Nitroglycerinum*—*Nitroglycerine*, or *Trinitrin*. Alcoholic solution of nitroglycerine (1 per cent.) 25 minims; cherry-laurel water up to 160 minims. *Dose*, 2 minims, repeated as required. Injections should be made deeply into the thighs or gluteal regions. *Used* in angina pectoris, asthma, megrim, Menière's disease, sea-sickness, puerperal convulsions, &c.

*Phenazone*. Doses of 2 grains and upwards dissolved in distilled water may be injected in pyrexia. The drug has also been used hypodermically in various neuralgiæ, lumbago, neuritis, megrim, and sea-sickness. It should be deeply injected.

*Picrotozinum*—*Picrotozine*. *Dose*,  $\frac{1}{60}$  to  $\frac{1}{20}$  grain. *Used* with great benefit in the night-sweats of phthisis. Administered at intervals of two to ten days. According to Gubler, useful in labio-glosso-laryngeal paralysis.

*Pilocarpina*—*Pilocarpine*, and its salts. Nitrate of pilocarpine 1 grain; distilled water 20 minims. *Dose*, 2 to 6 minims (Martindale and Westcott). *Antidote*, atropine. *Used* as a powerful diaphoretic in uræmia, asthma, puerperal convulsions, tabes spinalis, polyuria, &c.

*Quinina*—*Quinine*. The hydrobromate is the most useful salt for hypodermic purposes. Quinine hydrobromate 15 grains; distilled water 80 minims. Sixteen minims (= 3 grains) thrice daily. Tabloids containing  $\frac{1}{2}$  grain may also be used. *Used* as an

antipyretic in phthisis, fever, and as an anti-periodic in ague.

*Solanine, Hydrochlorate of.* This alkaloid belongs to the same class of poisons as eserine, pilocarpine, and atropine. The hydrochlorate is a gelatinous body readily soluble in water. The dose is 1 to 3 grains per diem. It has been used with marked benefit by Geneuil in sciatica, rheumatic neuralgia, prurigo, cystitis, and other ailments in which nervous symptoms preponderate. In children it is said to be a safe and useful substitute for morphine.

*Sparteine, Sulphate of* (an alkaloid obtained from the *Cytisus scoparius*). Dose,  $\frac{1}{15}$  to  $\frac{1}{2}$  grain. Used at first as a diuretic. Has since been strongly advocated by Dr. Oscar Jennings to overcome the morphine craving, alcoholism, &c.

*Strophanthin* (the active principle of *Strophanthus hispidus*). Tabloids ( $=\frac{1}{5000}$  grain) are the most convenient form for hypodermic use. Used in all forms of weak heart, in granular kidney, and other forms of Bright's disease, and in asthma due to cardiac and renal troubles.

*Strychnine*, and its salts. For hypodermic purposes the sulphate, nitrate, or hydrochlorate may be used. Discs ( $=\frac{1}{10}$  grain) or tabloids ( $=\frac{1}{100}$  and  $\frac{1}{1000}$  grain) are convenient. Strychnine hypodermically has also been used with great advantage in various forms of paralysis—e.g. of the vocal cords, in some forms of hemiplegia and paraplegia, also in urinary incontinence, tabetic pains, diphtheritic paralysis, &c. Drs. Lauder Brunton and Cash have shown that strychnine has a powerful action on the cardiac ganglia, and on the vaso-motor and respiratory centres. Hypodermic injections of the drug are of great value in feeble cardiac action, especially when due to acute pulmonary disease, in renal coma, and in threatened cardiac failure from shock or exhaustion. They are also of undoubted service in failing respiratory action due to opium poisoning. The best antidote to strychnine poisoning is chloral hydrate by mouth, or amyl nitrite hypodermically.

*Urethan* is useful in cases of simple insomnia and in that due to mental disease. Hypodermic injections cause trifling pain, and doses of from 2 to 5 grains may be given by this method.

*Vaseline* and other petroleum products, such as paroleine, are specially useful as vehicles for the hypodermic injection of antiseptics. Eucalyptol, turpentine, carbolic acid, iodoform, iodine, and camphor may be injected in this form without giving rise to local irritation.

ANDREW S. CURRIE.

**HYPOGASTRIC REGION** ( $\delta\pi\acute{o}$ , under; and  $\gamma\alpha\sigma\tau\acute{\eta}\rho$ , the belly).—The hypogastric region is conventionally described as lying between the right and left inguinal, below the umbilical, and above the pubic regions.

**ANATOMICAL RELATIONS.**—The surface of the hypogastric region in ordinary persons is flat, showing the muscular reliefs; it is rounded in children; and in some individuals, much emaciated from disease, it becomes concave.

The median furrow disappears below the umbilicus, owing to the approximation of the recti muscles. In women the furrow is rarely pronounced—a point of importance in ovariectomy.

The integument is very elastic and movable, especially at the sides.

The superficial fascia consists of two laminæ, between which lie the subcutaneous vessels; but in the middle line these laminæ are blended. It is strengthened at its lower part by the triangular fascia.

The aponeuroses of the external and internal oblique muscles are united in the linea alba, and form a portion of the sheath of the rectus.

The recti muscles themselves have their inferior attachments in this region, along the line extending between the spine and the symphysis of the pubes; their outer edges curve outwards, and become straight as they enter the sheaths.

The pyramidales, two small triangular muscles, arise from the pubes, lie in the same sheaths as the recti, and assist in closing in the abdominal parietes anteriorly and below.

Immediately beneath the recti is the fascia transversalis, with a little loose areolar tissue and fat, the fascia being tolerably adherent along the central line. Beneath the fascia transversalis there is a considerable amount of loose areolar tissue, between it and the parietal peritoneum, which in this region is very loosely attached, and reflected off the bladder on to the fascia transversalis. Enclosed in folds of the peritoneum lie, on either side, passing upwards to the umbilicus, the remains of the hypogastric arteries; and from the apex of the bladder in the middle line, passing to the umbilicus, is the obliterated urachus, which acts as the superior ligament of the bladder.

The viscera corresponding to the hypogastric region are—the bladder when full; and the small intestine, covered by the great omentum. When the bladder is full, the intestines are pushed aside, and the former then lies against the pubes and recti. In children, the bladder, being an abdominal rather than a pelvic viscus, always lies in this region. During pregnancy the uterus also corresponds with the hypogastric region.

The vessels are the deep epigastric, with the veins which pass obliquely inwards from the internal iliac. They lie between the peritoneum and the transversalis fascia.

The nerves are derived from the lower intercostals and lumbar. The lymphatics pass into the inguinal, superficial pubic, and lumbar glands.

**CLINICAL RELATIONS.**—The hypogastric region is of clinical importance chiefly from an operative point of view. It is in the median furrow (usually a brown mark, which sufficiently indicates to the operator his whereabouts) of this region that the operation of tapping in ascites is usually performed; and that the principal incision is made in ovariectomy, laparotomy for intestinal obstruction, and the Cæsarian section, supra-pubic lithotomy, and the many recent abdominal explorations and operations. The bladder is occasionally tapped above the pubes. The presence of the distended bladder or of the pregnant uterus, forming a tumour in the hypogastrium, has been already referred to. Enlargements here from these causes have to be diagnosed from pelvic tumours of various kinds, which, in growing upwards into the abdomen, occasionally occupy the middle region instead of either groin. Amongst the morbid conditions of the abdominal parietes in the hypogastric region that require special mention, are abscesses connected with disease of the lumbar spine, which occasionally point above the pubes, on either side of the middle line. The pus in such a case is normally situated between the peritoneum and the fascia transversalis. The anatomical conditions bearing on the operations for internal herniæ, &c., must be most carefully considered; as a rule, the mesial line is or should be selected for abdominal exploration.

EDWARD BELLAMY.

**HYPOGLOSSAL NERVE, Disorders of.**—The hypoglossal nerve is the motor nerve for the tongue, and for most of the other muscles which are attached to the hyoid bone, the exceptions being the stylohyoid, the mylohyoid, and the middle constrictor of the pharynx. It also supplies the sterno-thyroid muscle.

1. **Paralysis.**—Paralysis of this nerve is shown chiefly by the resulting interference with the movement of the tongue—'glossoplegia.'

**ÆTIOLGY.**—The hypoglossal nerve may be damaged in any part of its course by the growth of tumours, even outside the skull; but is most commonly affected at its origin from the medulla, by pressure, meningitis, syphilitic processes, or by caries of the upper cervical vertebræ. The tongue is also paralysed by disease of the nucleus of origin of the hypoglossal fibres, but its paralysis is then associated with that of the lips, and commonly also of the palate, pharynx, and glottis (*see LABIO-GLOSSO-LARYNGEAL PARALYSIS*). Such paralysis may be sudden, from local softening, or gradual, from nuclear degeneration. The latter form is often part of progressive muscular atrophy. Disease of the motor tract above the nucleus also causes paralysis of the tongue, together with the

face, arm, and leg of the same side. The loss of voluntary power of protrusion is much greater when the paralysis is on the right side, than when on the left, but probably only when the disease is in the cortex, and aphasia is associated with the hemiplegia. Bilateral glossoplegia commonly results from disease of the nucleus or its neighbourhood, rarely from symmetrical disease of the cortex in 'pseudo-bulbar paralysis.' Unilateral paralysis, when isolated, is generally due to disease of the fibres of the nerve within or outside the medulla.

**SYMPTOMS.**—In unilateral paralysis, the tongue at rest is in its normal position in the mouth, but its root is higher up on the paralysed than on the normal side, in consequence of the loss of the tonic, or voluntary, contraction of the posterior fibres of the hyoglossus. Within the mouth, the tongue is moved freely to the healthy side, but is not moved to the paralysed side. When protruded, it deviates towards the paralysed side, because the protrusion is the result of the action of the fibres of the genio-glossus, and the tongue is pushed over towards the weaker side. In bilateral paralysis the tongue lies in the mouth behind the teeth, and cannot be protruded. If the loss of power is complete, the tongue cannot be projected over the lower teeth. It is broad and flabby, if there is no atrophy, and sometimes when atrophy is associated with fatty overgrowth. When there is wasting, as is generally the case when the nerve or nucleus is diseased, the tongue is shrunken and its surface is wrinkled, on one side or both. The faradic irritability of the muscular fibres is then lost. In unilateral paralysis, articulation and deglutition are little impaired. The pronunciation of labials and the production of falsetto notes may, however, be difficult. In bilateral paralysis, articulation is impossible. Phonation is not impaired, unless the larynx is also paralysed. Mastication is impeded, because the food cannot be moved about in the mouth. Deglutition is also interfered with, because the food cannot be rolled into the fauces; and soft foods, when they reach the pharynx, may be driven again into the mouth, in consequence of the absence of the natural supporting movement of the tongue. Taste is not primarily affected, but may be somewhat dulled, because the patient is unable to move sapid substances over the surface of the tongue.

**DIAGNOSIS.**—The position of the lesion is indicated by the associations of the paralysis. If the disease is in the motor tract above the nucleus (pons, crus, or hemisphere), there is hemiplegic weakness on the side of the paralysis of the tongue. In disease of the nucleus the paralysis is commonly bilateral, is associated with paralysis of the lips and throat, and there is usually wasting. Disease of the fibres of origin within the medulla may

be associated with paralysis of the opposite limbs, so that the tongue deviates from the paralysed side. When the disease is at the surface of the medulla, the paralysis is commonly associated with that of the corresponding half of the palate and vocal cord, from disease of the adjacent spinal accessory. In all these cases there is commonly wasting. The diagnosis of the pathological cause of the paralysis rests on the course of the affection, and on the presence of any causal and associated condition. The chief sources of error in diagnosis arise from the fact that in hysteria the tongue is often voluntarily but persistently protruded towards one side; the cause is recognised by the freedom of movement within the mouth. In facial paralysis, with lateral deviation of the orifice of the mouth, the tongue may be protruded, consensually, in the middle of this, and therefore to one side of the middle line of the face. A knowledge of the fact will prevent error. See FACIAL PARALYSIS.

**PROGNOSIS.**—This is usually unfavourable, on account of the gravity of the disease which damages the nerve or centre. Even in syphilitic cases, recovery is often incomplete unless early and energetic treatment can be adopted.

**TREATMENT.**—The treatment of paralysis of the hypoglossal nerve is that of the causal disease. Tonics, counter-irritation, iodide of potassium and mercury, with the application of electricity to the tongue if there is reason to anticipate recovery of some nerve-fibres, are the most important remedies to be employed, according to the ætiological indication. The most convenient method of applying electricity is by means of a tongue depressor in a wooden handle, the blade being insulated by a coating of sealing-wax where it comes in contact with the lips.

**2. Spasm.**—Spasm in the parts supplied by the hypoglossal nerve is rare. The tongue participates in the convulsive movements in epilepsy, is jerked between the champing jaws, and thus becomes bitten. It is often the seat of tonic persistent spasm in hysteria. Cases have been met with in which the tongue is affected with a 'functional spasm' in speaking, analogous to 'writer's cramp,' but these are so rare as scarcely to need detailed description. W. R. GOWERS.

**HYPOSPADIAS** (*ὑπό*, under; and *σπᾶν*, I draw, or tear).—A malformation of the penis in which the orifice of the urethra is underneath or behind the glans. See PENIS, Diseases of.

**HYPOSTASIS** (*ὑπό*, under; and *στάσις*, standing, settlement).—**DEFINITION.**—This term is applied to that condition of the vessels of a part which consists in an overfulness, with a diminution in the rate of flow of the contained fluid, caused rather by a failure in the propelling forces of the venous circulation

than by an actual increase in the resistance ahead, as occurs in mechanical congestion. Among such causes, gravity, as exerted in the dependent position, is probably the most potent, especially when associated with lowered arterial tension.

**PATHOLOGY.**—The entire conditions of the venous circulation are such as to readily favour a stasis or stagnation of the blood-flow; and a trifling cause, such as would in no way affect the arterial flow, may easily impede the venous current. The veins also are, as a rule, less firmly supported by the surrounding tissues than the arteries; and this, with their thinner coats, slighter elasticity and resistance, render them easily liable to distension by the blood in congestion. If a dependent position be added to these conditions, thereby offering a resistance to the return flow of the blood, whilst it favours the circulation in the arteries, a combination of circumstances exists to which the term *hypostatic congestion* is applied. The liability of the veins of the leg to become congested, leading to a varicose condition and its results, is an example of this state. If, in addition to all these factors, the heart be enfeebled and the arterial tone be diminished, together with an impaired *vis à fronte* from weak inspiration, obviously another cause for stagnation is introduced, which of necessity will manifest itself most in those situations predisposed to stasis. Such a state is seen in the hypostatic congestion of the lungs which usually attends in a greater or less degree all pyrexial conditions, especially the typhoid state. The recumbent attitude, the enfeebled heart, and the lax vessels eminently favour an overfulness of the veins. It is obvious that, although the excess of blood primarily occurs on the venous side of the capillary system, very soon the arterial area will share in the engorgement, and the whole vascular system of the part become overfilled.

**RESULTS.**—The pathological results of such a state are very much the same as those following any congestion. The distended vessels, with the increased blood-pressure that co-exists, readily permit of a transudation of the fluid part of the blood, hence cedema; if the conditions be extreme, capillary rupture may take place, and hæmorrhagic effusions result. Any continuance of this state will lead to malnutrition of the tissues affected; the proper supply of arterial blood is interfered with; and the part is loaded with an effete venous blood, and is infiltrated with serum. Hence the structural repair of the tissues is improperly performed, and there is a tendency to the development of an imperfect form of connective tissue; or, on the other hand, the destructive rather than the productive aspect may predominate, and ulceration follow. When the hypostasis is associated with an acute general state, as of the lungs in any specific febrile disease, it is

very apt to pass on into a form of inflammation characterised by a want of acuteness. There are the same inflammatory products, the same changes in the vessels and tissues of the lungs, and very much the same symptoms as occur in the course of an ordinary pneumonia, but they are less severe in character, and on the whole do not tend so readily to a favourable resolution.

**TREATMENT.**—Recognising the cause, alteration of position is obviously the rational treatment of hypostatic congestion; additional support by bandaging is necessary. In acute febrile diseases cardiac stimulants are of much service in the prevention or relief of this condition in connexion with the lungs, should the heart's action be enfeebled.

W. H. ALLCHIN.

**HYSTERALGIA** (*ὀστέρα*, the womb; and *ἄλγος*, pain).—Pain in the womb, frequently supposed to be of a neuralgic nature. See **WOMB**, Diseases of.

**HYSTERIA** (*ὀστέρα*, the womb).—**SYNON.**: Fr. *Hystérie*; Ger. *Hysterie*.

**INTRODUCTION.**—'Hysteria' is a term the etymology of which is misleading, and had best, therefore be disregarded. It is often improperly applied to cases of simple malin-gering, and others which do not admit of ready explanation. Its use is best restricted to a condition of the nervous system fairly defined, but the intimate pathology of which is not known, characterised by the occurrence of convulsive seizures and by departures from normal function of various organs, leading to very numerous and often perplexing symptoms. These are apt to simulate those commonly arising from definite alterations of structure, but differ from the latter in the fact that they may often, even when at their worst, be removed instantaneously, usually under the influence of strong emotion. It would seem that there is a disturbed or congenitally defective condition of the cerebral substance, involving in all cases the highest nervous centres, and in various examples extending more or less also to some of those which preside over automatic phenomena. Partial or complete suspension of inhibitory influence would appear to be the most patent result of the condition, whatever it be; and this is recognised as well in regard to the mental as to the more evidently physical processes belonging to cerebral function. A laugh which cannot be checked, but continues until tears flow or the limbs become convulsed, is a typical example of such a suspension of control, and, if studied, throws light upon the nature of a considerable portion of the phenomena of hysteria. The jerking expirations of laughter arise from excitation of the respiratory centre; and when this excitation, uncontrolled by higher centres, acquires an abnormal strength, it extends to other parts of the medulla oblongata and spinal cord,

and produces general convulsions. It overflows, as it were, into other nervous centres, which in health would receive none of the exciting impulse. Between the lowest (automatic) functions of the cerebro-spinal nervous system and the highest (psychical) there is an ever-increasingly complex system of excito-motor processes, which may be in part or wholly under the pathological influence, whatever it be. Hence the *bizarre* character of the hysterical phenomena, and the circumstance that the symptoms always include modifications of those processes which underlie the mental faculties. The suspension of the power of control possessed by the higher centres explains the irregular movements, spasms, and convulsions. In hysteria, hyper-æsthesia and pain are probably dependent upon such a molecular change being initiated in the sensory ganglionic centres as is ordinarily propagated from the periphery. Hysterical paralysis, on the other hand, signifies that the power of the higher centres in liberating movements is in abeyance. In hysterical anaesthesia it is probably feeling or sensory perception, and not the function of the more immediate sensory apparatus, which is in abeyance, whilst the reflex actions which result from excitation of sensory nerves are performed in an orderly manner. A patient may work a needle with fingers which can be touched or pricked without the act being felt. Tactile impressions are conveyed to the ganglionic centres by the afferent nerves, and excite the action of efferent nerves so that the muscles are contracted. What is wanting is the participation of those higher centres in which consciousness runs parallel to this physiological action.

**ÆTIOLOGY.**—*Predisposing causes.*—It is probable that a state of more or less imperfect development of the higher nervous centres, of congenital origin, very frequently underlies the various circumstances which apparently conduce to the hysterical conditions.

The female sex is much more prone than the male to the affection, which usually occurs between the ages of fifteen and thirty, and most frequently of all between fifteen and twenty. Luxury; ill-directed education, and unhappy surroundings; celibacy where not of choice, but enforced by circumstances; unfortunate marriages; alcoholism; premature cessation of ovulation; and long-continued trouble—all predispose to hysteria. A somewhat frequent antecedent is a long and wearisome nursing of a sick relation, with much broken rest. The disorder is only exceptionally found in women suffering from diseases of the genital organs, and its relation to uterine and ovarian disturbance is probably neither more nor less than that which obtains in other neuroses. Exception must be made in the case of prostitutes affected with venereal disorders, who are

very prone to hysteria. In this class, however, the condition is complicated by the physical and moral influences to which their life subjects them, and amongst these alcohol frequently occupies a very important place. Like epilepsy, migraine, and some forms of insanity, hysteria is prone to be intensified at the catamenial period.

The occurrence of hysteria (although comparatively rare) in males is sufficient of itself to disprove the uterine theory of causation.

*Determining causes.*—These include painful impressions; long fasting; strong emotions; imitation; and shock to the nervous system, physical or moral.

*SYMPTOMS AND DIAGNOSIS.*—In the limited space in which it is necessary that the subject of hysteria should be treated, it will be best to describe together some of the most frequent forms which the neurosis takes, and the principles upon which a diagnosis can be made. Hysteria produces symptoms which may be referred to every function of the body. For consideration they may be roughly classed in the following groups, it being understood that all may occur either coincidentally or in succession: (1) *Mental*; (2) *Sensory*; (3) *Motor*; (4) *Circulatory*; and (5) *Visceral*.

1. *Mental.*—The intelligence may be apparently of good quality, the patient evincing sometimes remarkable quickness of apprehension; but, carefully tested, it is found to be wanting in the essentials of the highest class of mental power. The memory may be good, but judgment is weak, and the ability to concentrate the attention for any length of time upon a subject is absent. So also regard for accuracy, and the energy necessary to ensure it in any work that is undertaken, are deficient. The emotions are excited with undue readiness, and when aroused are incapable of control. Tears are occasioned not only by pathetic ideas but by ridiculous subjects, and peals of laughter may incongruously greet some tragic announcement. Or the converse may take place: the ordinary signs of emotion may be absent, and replaced by an attack of coma, convulsion, pain, or paralysis. Perhaps more constant than any other phenomena in hysteria is a pronounced desire for the sympathy and interest of others. This is evidently only one of the most characteristic qualities of femininity, uncontrolled by the action of the higher nervous centres, which in a healthy state keep it in subjection. There is very frequently not only a deficient regard for truthfulness, but a proneness to active deception and dishonesty. So common is this, that the various phases of hysteria are often assumed to be simple examples of voluntary simulation, and the title of disease refused to the condition. But it seems more reasonable to refer the symptom to impairment of the highly complex nervous processes which form the physiological side of the moral faculties.

2. *Sensory.*—Pain, hyperæsthesia, and anæsthesia occur with perhaps equal frequency. The diagnosis of the hysterical origin of such alterations of sensibility is effected partly by excluding the presence of other causes, and partly by consideration of any accompanying or antecedent peculiarities of manner and conduct. Hysterical *pain*, where it is associated with some evident local change, is found to be greatly in excess of that which would ordinarily accompany the observed cause. Where pain or hyperæsthesia is complained of in situations and of a character which would commonly point to some existing inflammation, it is necessary, by examination of the pulse and temperature, to exclude such a condition. Hysterical pain is apt to cease suddenly when the attention is diverted, and to be increased by inquiry and sympathy. Some of the most common seats of pain and tenderness are the following:—

(a) The lower part of the side of the chest (usually the left) simulating intercostal neuralgia, but distinguished from it by the tenderness being widespread, superficial, and not confined to certain points. Pressure here will sometimes occasion disturbances of respiration and circulation.

(b) Some of the vertebral spines, usually in the cervical and upper dorsal region. From the error of mistaking this for commencing disease of the vertebræ numbers of young women have been confined to a couch for months or years, and their health permanently damaged. The points of diagnosis are the patient's antecedents: there is often a history of aphonia, or paralysis, or hysterical fits. Or it may happen that, long after the pain has been first complained of, the patient has been seen to take a prodigious amount of exercise *on some one occasion* without complaint. A very much slighter pressure, too, causes pain than is at all usual in vertebral caries. It has to be remembered, however, that a patient affected with vertebral disease may also be hysterical.

(c) Acute pain in a joint, occurring usually some little time after a slight injury and giving rise to suspicion of inflammation, but distinguished from this by the fact that after a few days of great pain the joint does not feel hot to the touch, and is not swollen, and that the thermometer shows no rise of temperature. The pain is more easily excited, too, by touching the skin than by pressing the articular surfaces against each other.

It is necessary to remember that in locomotor ataxy there may be exquisite pains (of a shooting character) having their seat in a joint or its neighbourhood, and accompanied by some localised hyperæsthesia of the skin. The disease rarely affects young females, but it may do so, and the condition is then extremely liable to be mistaken for hysteria. The chief points of diagnosis are, that in

locomotor ataxy there will usually be—(1) absence of patellar tendon reflex; (2) a peculiar character of the pains—lightning-like; (3) probably some analgesia of the extremities; and (4) an ataxic gait. See TABES DORSALIS.

(d) Tenderness of the mamma or darting pains through its substance, recalling those of scirrhus. The absence of any lump, and the effect of engaging attention, will serve to insure the distinction.

(e) Pain in the head of very severe character, 'like a nail being driven into the skull' (*clavus*). This is probably neuralgic, and is by no means confined to the hysterical. There is also a more diffused pain, described as of great violence and exceedingly obstinate. This pain is sometimes suggestive of cerebral tumour, from which, however, it may often be distinguished by the fact that the ophthalmoscope shows no optic neuritis, and that there is no vomiting. But great caution is necessary in coming to a conclusion that severe and long-continued pain in the head is hysterical. And here it may be well to say that in an accurate knowledge of the characteristics of the disease supposed to be simulated lies the only safety as regards the diagnosis of hysteria. Nor must it be forgotten that persons with serious organic disease are frequently affected also with hysterical symptoms.

(f) Epigastric tenderness. Careful pressure will often show that the tenderness is at the origin of the recti abdominis muscles, and not in the stomach. But there is sometimes pain in the stomach itself, and this may be associated with disgust for food or depraved appetite.

(g) Tenderness in one or other iliac region, deep pressure upon which will sometimes evolve hysterical symptoms, and also in some cases of hysterical convulsions will check the paroxysm.

*Anæsthesia* may involve (though rarely) the whole body. It is more commonly confined to one half, and this the left, and is then frequently associated with tenderness in the iliac region of the same side; or it may be limited to a small patch. The sense of touch often remains whilst painful impressions and those of temperature cannot be perceived. The anæsthesia may be confined to the surface, or involve as well the deeper structures, into which pins may be stuck without evoking signs of pain. The left conjunctiva is often the seat of anæsthesia, so that it may be touched or even rubbed without any reflex movements of the eyelids being excited. So also the pharynx may be tickled without exciting the ordinary spasmodic contraction, and the epiglottis touched by the finger without inconvenience. A very frequent symptom is loss of reflex contraction when the sole of the foot is tickled with a feather. This is sometimes complete; in other cases prolonged

titillation will evoke a plantar reflex of greater or less extent. Such affections of cutaneous sensibility or of reflex action may have to be looked for, as they are often unsuspected by the patient herself. As regards diagnosis, the existence of peripheral nerve-lesions may be excluded by the absence of trophic disturbance. The condition is not likely to be confounded with hemiplegia, unless perhaps when it has immediately followed a convulsive attack, and is accompanied by apparent loss of power in the limbs. Examination of the patient and her history will suffice for the diagnosis.

The other special senses also may be disordered in hysteria. There may be intolerance of light, subjective sense of taste or smell, roaring noises in the ears; or, conversely, loss of sight (either in half of both eyes, or in one eye), loss of smell, or taste, or hearing. Or there may be feelings as of a limb or other part being enormously enlarged, of the body being confined in a stiff case, of the feet being drawn up by strings under them, of 'pins and needles' around the waist, or of numbness and coldness in one half of the body (almost always the left). It may be said generally of the disorders of sensation that they are capricious in their appearance, coming and going as they would not did they depend upon organic disease (this recurrence is especially significant); that they are very apt to ensue upon some moral shock or convulsive seizure; and that careful examination will prove them to be unaccompanied by such other symptoms as would be likely to be present did they depend upon the organic alteration which they simulate.

3. *Motor*.—The principal motor symptoms in hysteria are local spasm, more or less general convulsion, and paralysis. A common symptom of hysteria is the *globus hystericus*. A lump appears to the patient to arise from the epigastrium like an egg, and, travelling upwards to the throat, causes a sensation of choking, and is often accompanied by an outburst of tears.

Spasm affecting some out of the various muscles concerned in the respiratory acts gives rise to a great variety of symptoms highly characteristic of hysteria. There may be cough of a peculiarly sharp, ringing character, constant except during sleep, unaccompanied by expectoration, strongly influenced by moral causes. A little observation will show that the cough does not occur when the patient is quite alone and apparently no one within earshot; but, on the other hand, is greatly intensified by inquiry and solicitude. It ceases during sleep. Sometimes, instead of cough, a loud expiratory sound is produced, of most discordant character, resembling, perhaps, a railway whistle, the quacking of a duck, or the barking of a dog; and this may take

place irregularly, or may be marked by a curiously distinct periodicity. Or there may be rapid, deep whooping inspirations, with signs of suffocation. Occasionally with the hysterical cough there is a hypersecretion of mucus; and if, as often happens, there is also disturbance of digestive functions and consequent tendency to emaciation, and at the same time such constriction of the air-passages as gives rise to sibilant râles, a *primâ facie* resemblance to phthisis is presented, which can only be distinguished by prolonged observation, aided by the stethoscope and thermometer. Laughing and crying are very frequent forms of expiratory spasm. Yawning, hiccough, and sneezing are also met with. *Clonic spasm* of muscles, especially of those moving the head and shoulders, or back, is not uncommon. Or one of the muscles of the thigh may be so affected, and the apparent pulsation caused by the rhythmical contractions give rise to a suspicion of aneurysm.

*Tonic spasm* of one or more muscles of a limb is still more frequent. It is often very obstinate, and after enduring for months or years may suddenly resolve without any permanent alteration being left behind.

Contracture of a limb thus produced may continue during sleep, and even resist the influence of chloroform inhalation, unless this is pushed to its full extent. Should one of the abdominal muscles be thus affected, an abdominal tumour is produced, which may be mistaken for some growth in the cavity; and if the pulsations of the aorta should be communicated to it, a strong *primâ facie* resemblance to abdominal aneurysm is caused. The best mode of diagnosis is by faradisation, which, if persevered in for several minutes, will exhaust the muscular contractility and cure the ailment if it be of this kind. If it is not a 'phantom tumour' of this description, but a genuine growth, the muscle will be contracted by the current, and it may then be possible to feel the tumour as something evidently distinct from the muscle.

*Convulsive seizures* are of common occurrence, and are usually preceded by a sense of suffocation, difficulty of swallowing, pain in the belly or stomach, headache, vertigo, or some indescribable sensation in one of the extremities. There is often a cry as of one being choked, unlike the peculiar wailing shriek which ushers in the epileptic seizure. Usually there is not the extreme suddenness of attack which characterises epilepsy, but the patient may be manifestly struggling against the seizure for a small but appreciable interval. When at last she falls, she does not usually do so with violence enough to receive severe injury, and positions of danger are generally avoided. The epileptic often falls in the fire, the hysterical patient never. The spasms of muscles which suc-

ceed are often tetanic in character, and sometimes wear an aspect of design—the patient grips articles with her hands or teeth. The face may be more or less red. In epilepsy it is usually first pale and then livid.

There is often more or less complete opisthotonos, which is usually absent in epilepsy. It has been doubted whether consciousness is ever completely lost in an hysterical fit; but though for the most part perhaps it is retained during the attack, there are certainly cases in which it is to a great extent in abeyance. It is characteristic of hysteria that, however rapid and violent the contortions, the patient usually avoids inflicting any serious injury upon herself. During the attack gesticulations and language are apt to be used which may be reproachful, or marked by an amatory character as regards some bystander, such as is calculated to cause him considerable embarrassment. There may be a single convulsive seizure, terminated by a fit of weeping and the passage of a large quantity of almost colourless urine of low specific gravity. Or there may be a succession of attacks extending sometimes over several hours. The tongue is not bitten. As a rule, the hysterical patient rapidly returns to her ordinary condition after the outburst is completed, and fails to show the heaviness and tendency to sleep which is characteristic of epilepsy. The variety seen in the character of hysterical convulsions suggests that the pathological influence involves the nervous centres, sometimes more and sometimes less extensively; giving rise to various kinds and degrees of muscular movements, from those of a highly co-ordinated or quasi-voluntary kind, down to those of a simply tetanic form. These latter would appear to indicate either that control of the reflex function of the spinal cord is temporarily suspended, or that the cerebellar influence, as Dr. Hughlings Jackson thinks, is being allowed to have full play, owing to some peculiar condition of the cerebrum interfering for a time with its normal power of antagonism. But it must be borne in mind that the hysterical patient may, like others, become epileptic, and that there is nothing on the other hand to prevent the chronic epileptic from betraying occasionally symptoms of hysteria. Such mixed cases are often difficult of diagnosis, and it is usually only a prolonged observation which succeeds in distinguishing the nature of the condition. Movements which somewhat resemble those of chorea are occasionally met with, but their character and the surrounding circumstances usually make it easy to distinguish them.

*Paralysis* may affect any of the limbs in hysteria, but paraplegia is the more usual form. Hemiplegia is comparatively rare. The muscles retain their nutrition. There

is often at first a slight loss of irritability to induced currents, but after a very few applications this becomes normal. At first, too, considerable electro-cutaneous and electro-muscular insensibility may be present. If the form of paralysis be hemiplegic, the mouth is not affected; if paraplegic, the sphincters are not paralysed, and although incontinence of urine sometimes occurs, there is no cystitis, and the urine does not become ammoniacal. There is never any bed sore. If the arm be the limb affected, and the examiner, after flexing it slightly, leaves go, it will sometimes remain in the flexed position, which it would not do in hemiplegia. It is noteworthy that, in half the cases of hysterical paralysis, there is no history of antecedent convulsions. Cutaneous anæsthesia of the extremities will give rise sometimes to a pseudo-paralysis, and muscular anæsthesia may cause symptoms of ataxy. These may be distinguished from the result of organic change by careful examination. Hysterical speechlessness may be distinguished from aphasia by the patient being able to write down with great facility the wishes she is unable to express in speech; and from localised paralysis of the tongue, by her being perfectly able to protrude the organ and to swallow.

4. *Circulatory.*—There may be syncope which will simulate dying. After an indescribable sensation at the heart—a fullness or stifling feeling—the pulse becomes almost imperceptible, the patient is speechless, and, for periods varying in length, is apparently in a most precarious condition, recovery taking place after prolonged sighing. Or there may be tumultuous action of the heart. The abdominal aorta (and sometimes also other arteries) is occasionally the seat of powerful pulsations, which are visible in their effects upon the abdominal wall and strongly suggest the existence of aneurysm. The capillary circulation may be deranged in the two directions of hyperæmia and ischæmia. In the former there is a patch of redness of the skin, accompanied by a feeling of burning and tenderness; in the latter, which is especially seen in conjunction with analgesia, the skin is pale and no bleeding follows the pricks of a pin. In a recorded case, pressure upon a tender spinous process checked the radial pulse for a time.

5. *Visceral.*—Vomiting is sometimes a very obstinate symptom, all food taken being speedily ejected, the condition lasting a surprisingly long time, often for many months, sometimes for years, usually without so much prostration as might be expected, but nevertheless with great loss of weight. Or there may be such an active aversion from food as renders it very difficult to support nutrition; or a depraved appetite may cause substances to be swallowed which have no nutritious property. In the belly there is frequently a

hyper-secretion of gas with spasm of the bowels, causing borborygmi and noisy eructations. Intestinal gas may be imprisoned between two points of spasmodic contraction of the intestine, giving origin to a tumour capable of being moved about in the abdominal cavity, and of sudden resolution. These 'balloons,' as they have been called, are probably sometimes mistaken for tumours of the spleen, kidney, or other organ. It is not uncommon to have retention of urine, the bladder becoming greatly distended, but contracting at once and expelling its contents if the patient be placed in a hip-bath, and a bucket of cold water thrown over the pelvis. In other cases there is an unduly frequent desire to empty the bladder, and there may be some incontinence. The secretion of urine may be suppressed almost entirely (but this is very rare), the little urine that is passed containing an unusual proportion of urea, which is also found in the vomiting accompanying this condition.

There is sometimes very obstinate constipation, extending over weeks or even months, and giving rise to enormous impaction of fæces—occasionally also diarrhoea.

Cases occur in which the symptoms of cerebro-spinal sclerosis of the disseminated form, after persisting perhaps for many months in a young woman, rapidly or even suddenly disappear. This circumstance is often supposed to show that the case was one of hysteria. It is probable, however, that the view still generally held that the shifting of loss of power from one limb to another is really characteristic of hysteria is quite an error. The hysterical woman who has lost all power in her legs will, it is true, very often later on (whilst still paraplegic) lose the power of one arm (usually the left), but in the writer's experience she is not prone to lose the power in a limb, then recover it, and then lose it in another. The idea of this shifting of powerlessness being strongly suggestive of hysteria has probably arisen from the mistakes in diagnosing as hysteria cases of disseminated sclerosis, which must have been continually occurring before the latter disease had been differentiated. So in regard to vision. The hysterical patient will become quite blind of one eye, whilst the patient affected with disseminated sclerosis will only describe more or less obscurity of vision. The writer cannot call to mind, since he has been better acquainted with disseminated sclerosis, any case of simple hysteria in which first one eye lost some amount of vision for a time, and recovered, and afterwards the other eye behaved in a similar fashion. This is not at all uncommon in the course of disseminated sclerosis. The marked and almost constant occurrence of hysterical symptoms in the earlier stages of disseminated sclerosis often tends to obscure the diagnosis, and a very guarded prognosis becomes necessary.

It is common to class such cases with those of hysterical paralysis; but it is probable that they are examples of disseminated sclerosis recovering only for a time, as is characteristic of the disease. There is good reason to think that cases of disseminated cerebro-spinal sclerosis are not at all infrequently supposed to be simple examples of hysteria. The disease is characterised in its early stage (the only period when a mistake is possible) by some general weakness of limbs, accompanied by slight tremors on voluntary movement only, and an utterance which is slow and drawing, with occasional slurring of words. Careful examination, especially noting the circumstance that, for example, the arm only shakes when the patient is directed to take hold of an object, is usually sufficient to distinguish the disease from hysteria, but there is sometimes considerable difficulty, and great caution should be observed in avoiding hasty conclusions.

**SEQUELE.**—Hysterical symptoms sometimes pass into those of mania, melancholia, and occasionally also of dementia.

**PROGNOSIS.**—This is favourable as regards life, death from hysteria being very rare. Recovery for a time is common enough, but too often there is a return of the disease, the symptoms being usually of a different kind. Some patients will run through almost every conceivable phase of the disorder in turn. As a rule there is a tendency to cessation of the disease after the climacteric period. It occasionally happens, however, that the disease is continued into an advanced period of life.

**TREATMENT.**—If Medicine were in a position to regulate the mode of life, food, education, and especially the selections for propagation of the species, it is probable that in succeeding generations hysteria would become more and more rare in the race. In many cases it can do but little for the individual. Intercurrent maladies must of course receive the treatment proper to them. Where anæmia is present, much good may often be done by iron. States of malnutrition tend to precipitate and intensify hysterical symptoms; and to remedy these is often to do much for the concomitant nervous disorder.

But probably the greatest amount of benefit which can be brought to bear upon the hysterical patient is through her surroundings. A girl who has not spoken above a whisper for months whilst at home, will often recover her natural tone of voice in a week if placed under the judicious discipline of strangers. This is a well-known circumstance, and the fact has tended very much to the belief that hysteria is simply vicious simulation. Such an inference is unjust.

That an altered relation of the ganglionic nerve-cells to the blood-supply forms at least a part of the pathology of hysteria appears probable from the effects of fasting in provoking hysterical outbursts, and the influence of

food and stimulants in postponing them. Ammonia inhaled by the nostrils is a well-known and valuable agent for the purpose. Alcohol should be avoided altogether, as there is great danger of excess.

It is through the sensory nerves that the most rapid influence is brought to bear upon the hysterical condition. Thus cutaneous anaesthesia and hyperæsthesia may often be rapidly cured by the application of strong induced currents to the affected portion of skin; aphonia by acting in a similar way upon the skin covering the larynx. Paralysis of the limbs is in many cases quickly cured by the same means.

Hysterical convulsions may almost always be cut short by douching the patient very freely indeed with cold water. This should be poured from a height upon the face. For a few seconds there is no perceptible effect, then the breathing becomes gasping, and the patient seeks by moving away to avoid any further application. It often happens that the remembrance of this treatment serves to prevent a repetition of convulsions, but it would be wrong to conclude from this that the proceedings of the patient had been voluntary. The effect of the cold douche is to create, through the medium of the cutaneous nerves, a sudden change in the character of the blood-circulation, which may well influence the state of the ganglionic nerve-centres. The supposition seems fair that to remember the shock is to have a weak excitement of the nerve-centres which were strongly excited by the application.

Bromide of potassium, which is of such value in the *grand mal* of epilepsy, has no influence in preventing hysterical convulsions. In a doubtful case the exhibition of this drug is therefore useful for purposes of diagnosis. Valerian (the powder or tincture) has an unquestionable effect in the convulsive and spasmodic symptoms of hysteria, little or none probably upon the paralytic phases. Asafoetida by enema is useful in tympanites and colic of hysterical origin. Small doses of strychnine and opium are useful in relieving some of the distressing feelings complained of by hysterical patients.

Dr. Weir Mitchell, of Philadelphia, has introduced a mode of systematic treatment of hysteria and nerve prostration, which has lent important aid to our means of dealing with cases of this kind. Its introduction into this country we owe to Dr. Playfair. The treatment essentially consists in the complete isolation of the patient, who lies in bed and is fed and attended by an intelligent nurse. Under the influence of massage of the muscles of the trunk and extremities, conducted twice a day for an hour or more, the patient becomes able to take large quantities of milk (from 60 to 100 oz. in twenty-four hours), besides three full meals of highly nutritious food. By these means the nutrition

of the body undergoes a remarkable improvement, one or two stone of weight being often added; the muscles become firm, the skin soft and elastic; the complexion assumes the hue of health; the patient regains natural sleep if this has been lost; whilst various morbid symptoms—pains, local tenderness, contracture, loss of power in the limbs—disappear. It is especially in cases where signs of emaciation have been present that this mode of treatment achieves extraordinary success.

It is often a question whether the hysterical should marry. Where the disorder is slight and the general health is good, marriage may be advised, supposing that the prospects of a happy union are favourable. But in very severe cases, and especially when there is also a strong neurotic history in the family, it should be discountenanced. Nothing but harm can be expected from the strain of domestic cares upon a congenitally defective nervous system.

T. BUZZARD.

**HYSTERICAL INSANITY.**—Almost every variety of insanity may present in certain patients features which are commonly known and termed 'hysterical.' Melancholic individuals will be afflicted with hysterical paraplegia or other paralyses. Some will become cataleptic or apparently unconscious. Others will display all the phenomena of hystero-epilepsy. Not infrequently do we see a violent outburst of acute mania culminating and subsiding in a brief period of time, resembling in this an ordinary attack of 'hysterics.' It may be doubtful, however, whether hysterical insanity should be looked upon as a special variety of the malady. It seems more correct to look upon it as insanity occurring in hysterical patients, and characterised by the phenomena peculiar to them. We may expect sudden changes of symptoms, sudden improvements, and sudden relapses.

**PROGNOSIS.**—The prognosis is unfavourable, as this form of insanity is found in patients of an unstable nervous organisation, prone to frequent derangement. Even if recovery takes place, attacks are not unlikely to occur subsequently.

**TREATMENT.**—Such persons above all others require moral treatment. Medical treatment should be directed towards the improvement of the general health rather than the removal of special symptoms.

G. F. BLANDFORD.

**HYSTERITIS** (*ὕστέρα*, the womb).—Inflammation of the womb. See WOMB, Diseases of.

**HYSTERO-EPILEPSY.** — **SYNON.**: Fr. *Hystéro-Epilepsie*; *Hystérie Épileptiforme*; Ger. *Hysteroepilepsic*.

**DEFINITION.**—A term applied to a form of hysteria of unusual gravity, the convulsions

in their violence recalling those of epilepsy, and characterised by the occurrence of remarkable forms of anæsthesia, paralysis, and contracture of muscles.

**ÆTIOLOGY.**—The condition is one which must be classed with hysteria, and not with epilepsy, and the circumstances which tend to the production of the former disease are here equally potent. See HYSTERIA.

**SYMPTOMS AND DIAGNOSIS.**—It is to the French school, and especially to Professor Charcot of Paris, that we owe the most important descriptions of this disease, which would appear to be more common on the Continent than in England. The symptoms may be divided into:—(1) *motor*; and (2) *sensory*.

1. *Motor*.—Convulsive seizures occur, preceded by an hysterical aura (abdominal or epigastric), which usually gives the patient timely warning enough to enable her to place herself in a position of safety. Then there is a shriek, the face is pallid, and she falls (perhaps whilst endeavouring to quit the room), the features become distorted, and the limbs pass into a state of tonic rigidity. There is foaming at the mouth, sometimes the tongue is bitten, and there may be some clonic convulsions with lividity of features. Relaxation of the muscles and a more or less comatose condition succeed, to be followed shortly, however, by contortions and gesticulations of a violent character, coarsely suggestive of the influence of various passions—wrath, fear, disgust, lust. Or there may be meaningless writhings, presenting a hideous aspect. To this phase sometimes succeeds hallucination of vision, or of hearing. Rats and serpents and other objects of horror are seen. The attack ends with sobs or hysterical laughter. There may remain a temporary inability to empty the bladder or to swallow food.

Convulsive seizures of this co-ordinated or purposive character, although much more common in the female than the male sex, are by no means confined to the former. The condition is not infrequently observed in boys; less often in men. In the latter there is usually the history of some great moral shock antecedent to the first outbreak.

In patients liable to attacks such as have been described, it is not uncommon to find a *contracture* of one or more limbs. This may assume the *hemiplegic* or *paraplegic* form; and it may be of a passing character lasting a few days only, or enduring for many years. The attitude may be either that of rigid flexion or extension; and it is found to remain during sleep, only relaxing under the profound effect of chloroform narcosis. The limb so affected does not suffer in its nutrition, and the reaction of the muscles to electrical currents remains normal.

The contractures, as well as the sensory disturbances described below, may be said to be practically confined to patients of the female sex. It is very rare indeed to find them in males.

2. *Sensory*.—It has been noted that in female patients thus affected (as indeed is common in hysteria generally) there is apt to be a pain in one or other iliac region, most often the left, which is sometimes constant, and in other cases is only discovered by pressure. The seat of this pain is thought to be the ovary, and it is found that whilst a moderate pressure in this region may determine the production of the aura and sometimes of a hysterical attack, energetic compression at the spot will very often cut short the convulsive seizure.

*Anæsthesia* and *analgesia* are apt to be found sometimes in both sides, but much more frequently in one half of the patient's body, parted off from the other by the median line, and thus involving apparently half the head, face, and trunk, as well as the upper and lower extremities, though it may be in different degrees of intensity. It not seldom happens that the patient is herself unaware of the existence of this insensibility until examination has disclosed it. The loss of sensibility sometimes affects also the special senses; and smell, taste, hearing, sight, and the perception of colour, may each or all be lost on one side.

Accompanying the analgesia it is often seen that the pin-prick employed to test the condition fails to draw blood on the affected side, whilst readily doing so in the opposite limb.

Where there is loss of power of the limbs with contracture and anæsthesia following a convulsive seizure, it is not difficult for the condition to be ascribed to an attack of hemiplegia, resulting from organic disease, and there is sometimes a doubt on this matter which is not easily resolved. The points of most value in making a differential diagnosis are the following:—(a) The absence from the first of any deviation of the tongue or facial paralysis. After a time, no doubt, in some cases of hemiplegia of organic origin these symptoms become scarcely visible, but it may be said that practically at the onset they are always present to a greater or less extent. (b) The extent and completeness of the analgesia, especially the mode in which it affects the trunk, which ordinarily escapes in hemiplegia. Such complete anæsthesia as occurs in these cases is rarely observed in hemiplegia of cerebral origin. In spinal hemiplegia, again, it would occupy, as Brown-Séquard has pointed out, the side of the body *opposite* to that affected with motor paralysis. Besides these there is rarely much difficulty in finding in the symptoms, surroundings, or history of the patient, circumstances which, combined with those

described, throw a strong light on the nature of the condition. The hysterical patient may present, for example, retention of urine, ovarian tenderness, and tympanites; and in her history there may be an account of aphonia, convulsive seizures occurring under emotion, hysterical cough, or some other feature which tends to stamp the case as one belonging to this great neurosis.

Occasionally these contractures remain permanent. Much more frequently they relax, after a longer or shorter period, and the relaxation almost always takes place suddenly, usually under the influence of some moral shock.

It will sometimes happen that paresis and contracture of a limb will occur without any previous history of hysterical symptoms, and the possibility of this must always be borne in mind. Where there is during many hours a long-continued succession of fits with brief intervals of immunity, considerable doubt may arise as to the nature of the attacks, because a very similar, numerous, and rapid recurrence of fits sometimes takes place in true epilepsy. We are indebted to Charcot for the observation that whilst in the case of the epileptic seizures of this kind the temperature is observed to rise greatly (attaining, for instance, a height of 105° F.), no such great increase is noted when the fits are of hysterical origin—a slight elevation only occurring.

In cases of hystero-epilepsy it has been noted that there is often colour-blindness, affecting the eye on the same side as the hemianæsthesia—the order of disappearance, in most cases, being the following: violet, green, red, orange, yellow, blue. Violet is the colour most easily lost, red and blue, according to Charcot, being those which persist most, except in those cases where achromatopsia is absolute—that is, where the patient looking at a painting sees nothing but black and white.

Remarkable results have been known to follow the application of metallic plates and the approach of a powerful magnet to the anæsthetic side of the body. It is found that, if a small plate of some metal be applied for a few minutes to the skin, a return of sensibility occurs, touches and pricks previously unperceived are felt, and colour-vision is restored in the affected eye. The particular metal which will effect this change has to be sought by experiment: in one person gold, in another silver, in others again iron, tin, or copper, alone producing the effect. So again it is stated that the approach (without actual contact) of a powerful horseshoe magnet will produce a similar effect to the contact of a metal. In either case, *pari passu* with the return of sensibility on the affected side, it is noted that the other half of the body acquires the anæsthetic state. There is a transference

apparently of the phenomena. The approach of a magnet, it is also found, will cause a contracture to relax, the relaxation lasting for many hours. But in this case, again, the corresponding limb of the previously normal side is found to present evidence of paresis. In two instances Rosenthal and the writer have found defective electrical excitability, as tested by sudden interruption of a strong voltaic current, in the cerebral hemisphere opposite to the side which is marked by anæsthetic symptoms. In the writer's own case the experiment was followed by considerable improvement in the cutaneous sensibility.

**TREATMENT.**—Cases of hystero-epilepsy are not amenable to any treatment by drugs. Powerful moral impressions, especially energy and commanding influence in the medical attendant, are more potent than anything else in bringing about recovery, but it is not always that these can be brought

to bear. It is desirable, therefore, that in suitable cases the influence of the measures just described should be tested. In cases of convulsion strong pressure should be made upon that ovarian region in which tenderness is discovered. In examples of contracture and hemianæsthesia an application of metallic plates of various kinds may well be made to the skin of the affected side.

Very useful effects are often produced by the application of strong faradic currents, which may require to be persisted in ere any influence is produced. Blisters also, repeated every three or four days, are occasionally very useful in causing the relaxation of a contracted limb and the return of sensibility to an anæsthetic skin.

Removal of the patient from the family circle and the society of frightened or sympathising friends, is in most cases a *sine qua non* of successful treatment.

T. BUZZARD.

## I

**ICE, Therapeutics of.**—See **COLD**, Therapeutics of.

**ICHORRHÆMIA** (ἰχθῶρ, puriform matter; and αἷμα, the blood).—A morbid condition of the blood, caused by the absorption of septic materials. See **PYÆMIA**; and **SEPTICÆMIA**.

**ICHTHYOSIS** (ἰχθῦα, a fish-scale). **SYNON.**: Fish-skin Disease; Fr. *Ichthyose*; Ger. *Fischschuppenausschlag*.

**DEFINITION.**—A disease which has obtained its name from the division of the cuticle into polygonal plates somewhat like the scales of the fish, although no overlapping exists. The skin is dry, rigid, rough, and greyish-green, often of the hue of the upper surface of the turbot; and the cuticle exfoliates, in some places as dust, in others as thin shining laminae like mica or bran.

**ÆTIOLOGY.**—Ichthyosis may be regarded as a defect of development of the skin, usually, however, appearing some months after birth. In rare instances it has arisen in adults. Males are affected twenty times as often as females. Heredity sometimes obtains. The degree of its manifestation may depend on various circumstances, especially those relating to scanty food and want of cleanliness. It may be regarded as endemic in the Molucca Islands.

**ANATOMICAL CHARACTERS.**—The cuticle is abnormally copious; the fibrous tissue of the derma is condensed; the papillæ are enlarged and lengthened, and are sometimes

apt to bleed on slight injury; the areolar layer is lax and fatless, and the whole integument wants succulence and elasticity. Further, the cuticle is hard and brittle, the inorganic matter being increased fourfold, and it cracks along the lines of motion or wrinkles of the skin; the fragments being powdery upon the neck, front of the trunk, and flexures of the joints, angular and prominent on their extensor aspects, and smooth and polyhedral on the rest of the limbs. The follicles are filled with dry epithelium and sebum, which in other places may concrete and add to the thickness of the crust. The skin as a whole forms coarse wrinkles, and from the laxness of the subcutaneous areolar tissue it moves freely over the fascia. The oily, and sometimes the aqueous, secretions are scanty, an unpleasant odour is often exhaled, and the transparency and lustre of the healthy skin are wanting. The health is usually good, but cardiac hypertrophy has been noted. The cold of winter is felt severely, and death often results from pulmonary complaints.

**MODIFICATIONS.**—Ichthyosis varies according to site. On the limbs it is most symmetrical, and the scales are largest; on the hands and feet the cuticle is horny, and there are deep wrinkles; and on the face the detached edges of the plates cause great roughness, and the complexion is altered to a brick-red. Varieties are also produced by the amount of sebum, which may form either thick scales or projecting spines. Such

## ICHTHYOSIS

modified forms have suggested several synonyms. Thus, when dryness of the skin is conspicuous it has been termed *xeroderma*. When the network of lines bounding the scales is regular and widely stretched, from the tightness of the whole integument, the name 'harlequin skin' has been applied, such instances being usually congenital and the birth premature. When the smooth surface has a mother-of-pearl polish it has been called *ichthyosis nacrea*. When the concreted epidermic and sebaceous substances resemble the scales of reptiles, the term *ichthyosis serpentina* is applied, although the monitor is the reptile whose skin it is most like; and, finally, the variety in which long spines appear is designated *ichthyosis hystrix*, the 'porcupine disease.' Malformations of the eyelids, ears, and fingers have been frequently found in ichthyotic subjects. The disease has been known to disappear after eruptive fevers.

**TREATMENT.**—The principles of treatment are as follows: First, we must promote an improved nutrition of the body by the use of a generous diet, cod-liver oil, arsenic, iron, or other tonics. Secondly, it is necessary to remove the excess of epidermic matter and sordes, which is best effected, when the disease is limited, by resorcin or solutions of salicylic acid; when it is extensive, by ablutions with soft water and soft soap, and especially by the Turkish bath and shampooing. Thirdly, we have to stimulate the circulation and innervation of the skin by friction and inunction with such substances as lanolin, cod-liver oil, or cacao-butter mixed with glycerine. The salve mulls and super-fatted soaps, as used by Unna, give great relief in localised cases. Injection of pilocarpine has been used to increase sweating and thus soften the cuticle.

ERASMUS WILSON. E. D. MAPOTHER.

**ICTERUS** (*ικτερος*, a weasel; with yellow eyes).—A synonym for jaundice. See JAUNDICE.

**ICTUS SOLIS** (*ictus*, a stroke; *sol*, the sun).—A synonym for sunstroke. See SUN-STROKE.

**IDAHO HOT SPRINGS**, in Clear Creek County, Colorado, U.S.A.—Thermal waters. See MINERAL WATERS.

**IDIOCY** (*ιδιωτης*, a person private or apart).—SYNON.: Feeble-mindedness; Fr. *Démence Innée*; *Idiotisme*; Ger. *Die Sprechreihenheit*; *Blödsinn*.

**DEFINITION.**—Mental deficiency occurring during infancy or the early periods of life.

The term *idiocy* is not a scientific one, but it is convenient to employ it here to include a class of maladies which differ essentially from insanity, both as to their nature and treatment. The strict meaning of the word idiot is, 'a solitary one,' but it has become so much used as a term of opprobrium that it

were well if the phrase, 'feeble-minded,' could take its place.

**DESCRIPTION.**—The term 'idiocy' covers such a large area, and includes such a great variety of cases, that there is endless gradation in its manifestations, from slight departure from a normal condition, to that state of profound idiocy in which the unfortunate subject thereof sees nothing, feels nothing, does nothing, and knows nothing. The typical condition, however, is best illustrated by reference to an average state. For the most part, the lesion is not only a psychical one, but profoundly affects the physical, and frequently the moral life. The stature is less than normal, and there is a tendency to assume a stooping posture. The skin is often coarse, deficient in elasticity, and lax, with increased development of areolar tissue. The muscles are weak and flabby, and respond feebly and irregularly to the action of the will. The bones are often yielding and deformed. The circulatory system is usually weak, rendering the patient liable to destructive chilblains and frostbite, inducing perilous effects from exposure to a low temperature, and rendering slow any reparative process. The lungs are extremely liable to inflammatory attacks, both in their bronchial tubes and parenchyma, and prone to tubercular disease if the subject be resident on a clay soil. The digestive system is liable to be deranged by defective mastication of food, and alternately subject on the one hand to constipation from defective innervation, and on the other hand to diarrhoea from catarrh, resulting from alternations of temperature. The sexual functions are often abnormal; there is a tendency to masturbation very early in life, while puberty itself is generally delayed, and often sterility exists. There is not infrequently phimosis and undescended testis in the male, and non-development of the ovary in the female. The motor functions are also abnormal; there is usually defective co-ordination, resulting in a deficiency in purposive acts, while there is a tendency to the production of purely rhythmical and automatic movements. There is diminished sensibility, so that what is painful to others is borne with complacency. Speech is defective, partly from want of co-ordination of the muscles of the tongue, partly owing to malformations of the mouth and palate, and partly to inability to convert ideas into words. The sight is often impaired, due to hypermetropia, to imperfect retinal sensibility, to congenital cataract, or to diminished accommodation. These conditions are frequently associated with strabismus or nystagmus. The sense of smell is lessened, and the discrimination of odours almost *nil*. The sense of taste is also defective, leading to the eating of things of an unpalatable and even repulsive nature. The faculty of hearing is not much interfered with,

except in cases where there has been destructive disease of the ear. The faculties of observation and attention are limited. There is generally great fondness for music, and simple airs are often readily learned. The memory is generally good, and there are often instances of remarkable power in this respect. There is very little imagination or ability for abstract thought, while judgment and reasoning power are almost entirely absent.

**CLASSIFICATION.**—The best classification of idiocy, the one which most assists in the prognosis and treatment, is that which is based on its ætiology. The whole of the cases may be divided into three important groups, which groups afterwards admit of subdivision. The primary groups are:—(1) *congenital*; (2) *developmental*; and (3) *accidental*.

1. *Congenital idiocy.*—This includes all those cases which at the period of birth manifested signs of defective mental power, associated usually with conditions of the head, skin, and other organs, which are indicative of a congenital origin. They are cases which have never possessed ordinary mental power. The congenital group contains the following subdivisions:—(a) strumous; (b) microcephalic; (c) macrocephalic; (d) hydrocephalic; (e) eclamptic; (f) epileptic; (g) paralytic; and (h) choreic.

2. *Developmental idiocy.*—The developmental group includes a smaller number of cases, where the child manifests an average intelligence through infancy, or even up to the commencement of puberty, but, from causes which have influenced the nutrition of the embryo during its intra-uterine life, is born with a proclivity to mental break-down during one of the developmental crises; the crises being the periods of the first dentition, of the second dentition, and of puberty. The group includes those cases in which speech and mental faculties are lost in children in whom previously the intelligence was good—cases where the brain and nervous power were sufficient for their early years, but are insufficient to carry them through evolutionary stages. They usually present outward signs, in their prow-shaped cranium or other structural deviations, that the tendency to catastrophe was born with them. The developmental group embraces the following subdivisions:—(a) eclamptic; (b) epileptic; and (c) choreic.

3. *Accidental idiocy.*—The accidental group includes all those cases of idiocy where the child has been born with a normal nervous system, free from any present or potential defect, when unfortunately a fall, a fright, epilepsy, the result of some peripheral irritation, disease of the bones of the ear sequential to measles or scarlet fever, meningitis, or other cause, may lead before puberty to mental break-down—a break-down not of a genetic, but of a purely accidental origin.

The accidental group includes:—(a) traumatic; (b) inflammatory; and (c) epileptic idiocy. For the group of idiots produced by endemic influence, see **CRETINISM**.

**ÆTIOLOGY.**—The production of idiocy is multifiform in its causation: often more than one factor has been at work. The *congenital* kinds are produced by neuroses, struma, tuberculosis, alcoholism, over-intellectual work, over-sexual indulgence, and constitutional debility of the progenitors. Syphilis holds but a very unimportant place. Inter-marriage of relations, where there is a *constitutional taint*, in consequence of its insuring the existence of two potent factors; fright and emotional disturbance, or anxiety of any kind on the part of the mother during her pregnancy; and prolonged parturition and suspended animation at birth, are also to be reckoned as causes of congenital idiocy. The *developmental* kinds have their proclivity given to them by causes affecting the nutritive life *in utero*; notably emotional disturbances, and sickness produced by the pregnancy or by a prolonged sea-voyage. The exciting cause is a developmental crisis, such as occurs at the periods of dentition, and the evolution of puberty. Masturbation is a most important factor in determining this kind of idiocy. The *accidental* kinds are produced by injuries of any kind to the cranium, sunstroke, exanthematous disease, tubercular or other forms of meningitis, inanition, and epilepsy referable to worms, masturbation, or other sources of peripheral irritation.

**PROGNOSIS.**—In idiocy the future of the patient will be forecast by reference to the nature of the case. Other things being equal, patients paralysed or epileptic are less amenable to treatment than others, but the most unsatisfactory results are obtained among cases of accidental origin. It is important to recognise the fact that congenital cases, with marked traces of their infirmity in their faces and bodies, are, for the most part, more susceptible of improvement than the developmental, and these again than the accidental, who may have no appearance of idiocy in their faces or bodies; that, in fact, a favourable prognosis is often inversely as the patient is winsome, fair to look upon, and comely.

**TREATMENT.**—The treatment of idiocy consists of a judicious combination of medical, physical, moral, and intellectual agencies. The patient should be rescued from his *solitary* life, and have the companionship of his peers. He should be surrounded by influences, both of art and nature, calculated to make his life joyous, to arouse his observation, and to quicken his power of thought. The basis of all treatment should be *medical* in an enlarged sense. Success can only be obtained by keeping the patient in the highest possible health. The dietary should be liberal, containing a fair proportion of nitrogenous ele-

ments, while rich also in phosphatic and oleaginous constituents. The food should be presented, too, in a form suited to the masticatory power of the patient. It is of importance that the rooms he inhabits should be well-ventilated, whilst kept warm; and daily baths with shampooing should be employed. Of first importance is the soil: a clay soil is fatal to all proper progress, inducing tuberculosis, and lowering the vital power. *Physical* training forms an important part of treatment. The attenuated muscles have to be nourished by calling into exercise their functions, and the automatic and rhythmic movements have to be replaced by others which are the product of the will. The simplest movements should be first taught, then the more complex, thus causing to grow up together the mandate and the result. From purposeless acts the idiot thus builds up a series of co-ordinated and voluntary movements which are applicable to the wants of daily life. The training has to be carried out in minute detail, so that every voluntary muscle and every congeries of muscles may be called into action, and trained to fulfil with rapidity the end for which they are designed. The *moral* education is of paramount importance. The pupil has to be taught to subordinate his will to that of another. He has to learn obedience, that right doing is productive of pleasure, and that wrong doing is followed by deprivation thereof. Corporal punishment should be forbidden; the affective faculties of the patient should be so cultivated that the withdrawal of the love of his teacher should be felt as the greatest punishment, and the manifestation of it his highest reward. In no case should the punishment interfere with hygienic treatment. The *intellectual* training must be based on a cultivation of the senses. The patient should be taught the qualities, form, and relation of objects by their sense of touch; to apprehend colour, size, number, shape, and relation by sight; to understand the varieties of sound when addressed to the ear; the qualities of objects by the taste and smell. These lessons should be of the simplest at first, and gradually cumulative. Nothing should be left to the imagination. The idiot must be taught the concrete, not the abstract. It is in this way we should give him the basis upon which the reasoning and reflective faculties can be built up. Synchronously with this, use should be made of the physical powers which have been cultivated. He should be taught to dress and undress himself, to acquire habits of order and neatness, to use the spoon or knife and fork, to walk with precision, to handle with tact. The defective speech is best overcome by a well-arranged plan of tongue-gymnastics, followed by a cultivation of the purely imitative powers.

J. LANGDON DOWN.

**IDIOPATHIC** (*ἰδιος*, peculiar; and *πάθος*, a disease).—A term applied to a morbid condition when it arises primarily, and not in consequence of some other disease or injury. It is used in contradistinction to *symptomatic* and *traumatic*.

**IDIOSYNCRASY** (*ἰδιος*, peculiar; *σύν*, with; and *κράσις*, constitution or temperament).

**DEFINITION.**—This term, like many others used in science, has a more restricted application than its etymology would indicate. From meaning the personal constitution of an individual, it has come to mean any peculiar and not obviously correlated reactions against external influences exhibited by any individual. It is not to be confounded with 'constitution,' which is the foundation of the individual, his powers, capacities, and organisation; nor with 'temperament,' which denotes the correlation of powers and tendencies with the physical conformation of the individual, which has therefore a generic or race application, and which is defined by some writers as the 'general form' of the man. Commonly, any single peculiarity of a person is spoken of as 'an idiosyncrasy,' so that one individual may, in this sense of the word, manifest several idiosyncrasies or personal attributes.

**DESCRIPTION.**—Idiosyncrasies, so defined, may be mental or physical; may be innate or acquired; may be permanent or temporary.

The existence of idiosyncrasies being declared chiefly through the agency of nerves, and their operations being mostly capable of being brought under laws of innervation, Prochaska, Claude Bernard, and other authorities have regarded them as 'a peculiar affection of the nervous system.' But a review of the phenomena admitted to belong to the class will be found to compel us to recognise in many cases a more comprehensive relation, involving the whole organism, or parts of it other than the nervous system.

In the enumeration of the principal kinds of idiosyncrasies, to purely mental manifestations of likes and dislikes toward persons, things, and pursuits—affections covered by such terms as 'sympathy,' 'antipathy,' 'predilection,' &c.—must be added reactions in which the mind, the emotions, and the organic nervous system are affected simultaneously, but in varying proportions, by impressions received through organs of sense.

For example, vision may be the channel of affection. Syncope is produced in some persons by the sight of blood; or, as is related by Prochaska, swooning may invariably occur on the sight of beetroot. As regards olfaction, some people are distressfully affected, in both bodily and mental ways, by the exhalations from certain animals, the cat in particular; in others, horror and fainting are induced by the odour of roses or of

apples. And so on through the rest of the senses.

In another group of idiosyncrasies the higher nervous centres play no part, the phenomena being of reflex production through the spinal centres, or being due to direct poisoning of the system or of organs. Among foods or drugs swallowed, among gases or dusts inhaled, among substances brought into contact with the skin, many, harmless to the majority of men, are for this or that individual irritants or poisons. For instance, eggs, honey, sugar, or fish may produce gastric pain, nausea, or vomiting; strawberries are to a few persons a most virulent poison, producing symptoms of intense nervous shock; convulsive spasms may be excited by the smell of musk or civet; asthma by the inhalation of the powder of ipecacuanha; urticaria by the eating of shell-fish, or even by the application of the yolk of egg to the skin.

Some men there are love not a gaping pig;  
Some that are mad if they behold a cat;  
And others, when the bag-pipe sings i' th' nose,  
Cannot contain their urine.

*Merchant of Venice.*

Idiosyncrasies confronting the use of drugs have a special interest for the medical man. They may be of a qualitative nature, as in the production of unusual symptoms with dangerous or fatal results by anæsthetics, or in iodism; or of a quantitative nature, as in the case of opium and belladonna, minute doses of which will poison some persons, while doses of them, large enough to destroy a dozen average individuals, may be taken by one here and there with impunity.

The consideration of this part of our subject introduces the question of the variations of idiosyncrasies. The transient oddities of susceptibility arising in pregnancy, hysteria, and madness, are excluded by some authors from the category; but as they only differ in their transitory character from other idiosyncrasies, we shall here associate them with those modifications of reactive sensibility to which the term is commonly extended. Seeing that the causes of innate idiosyncrasies are for the most part unexplained, we may search out in varying or acquired idiosyncrasies varying correlations which may help us to the ultimate better understanding of the former group. Thus we know that intolerance of opium may arise in some morbid states the nature of which is fairly known; that tolerance of, or comparative indifference to, the same drug may be attained by its constant use. As in pregnancy new idiosyncrasies appear, so age, habits, and state of body may each and all modify the reaction of any individual towards his surroundings; may change his behaviour under the influence of drugs; may at one time charm him against morbid poisons, at another time leave him their

easy victim; may make him inflame sometimes in a suppurative, sometimes in a plastic way. From this point of view, we may, with Claude Bernard, summarise idiosyncrasies as being 'mere manifestations of the ordinary laws of physiology.'

*Imaginary idiosyncrasies.*—Persons are not infrequently met with who, held by prejudice, or misled by fancies or unsound judgments, declare that particular foods and medicines disagree with them. It may often be found, on investigation, that the assertion is incorrect. The obstacles offered to effective treatment by such fancies are sometimes of grave importance, tasking severely the sagacity of the medical man in the way of analysis, and his skill in the way of counteraction. But when the idea of their existence shall have been proved in any case to be unfounded, it is generally possible to evade such obstacles by tact, or to undermine them by argument, and, most of all, to dissipate them by firmness.

WILLIAM M. ORD.

**IDROSIS** (*ιδρώς*, sweat).—A synonym for hyperidrosis. *See* SUDORIPAROUS GLANDS, Diseases of.

**ILEO-TYPHUS.**—A synonym for typhoid fever. *See* TYPHOID FEVER.

**ILEUM**, Diseases of.—*See* INTESTINES, Diseases of.

**ILEUS** (*εἰλέω*, I twist).—A synonym for an intestinal obstruction. *See* INTESTINAL OBSTRUCTION.

**ILIAC REGION.**—The iliac region, or region of the iliac fossæ, is limited laterally and superiorly by the crest of the ilium, anteriorly by Poupart's ligament, and internally and below by the brim of the true pelvis or inner edge of the psoas magnus muscle.

**ANATOMICAL RELATIONS.**—That portion of the abdominal cavity which corresponds to these boundaries, contains the following viscera on the right side:—the cæcum, the vermiform appendix, some coils of the small intestines, and the ureter; and on the left side:—the sigmoid flexure of the colon and small intestines, and the ureter.

In front of the cavity is the ilio-inguinal region, which forms its anterior parietes, and from which surface all examinations of the region are instituted. Exploration is difficult in obese persons, and to facilitate it we must relax the abdominal parietes by flexing the thigh, and by pressing the fingers immediately above the crural arch.

The *peritoneum* is but very slightly united to the subjacent tissues, and is easily separated from them. It completely covers in the left iliac fossa; but on the right side, owing to the presence of the cæcum, this investment is incomplete. *See* LUMBAR REGION.

The *sub-peritoneal cellular tissue* may be regarded as being composed of two distinct layers. The first, immediately beneath the peritoneum, is a continuation of the lax cellulo-fatty envelope of the kidney and cæcum, passing with the femoral vessels into the crural canal, and with the spermatic cord into the scrotum. The deeper or sub-aponeurotic layer lies beneath the sheath of the iliaco-psoas muscle, being continuous above with the sub-pleural cellular tissue, and accompanying this muscle downwards as far as its insertion into the lesser trochanter.

The *arteries* of this region are the common and external iliaes, and their branches.

The *nerves* of the iliac fossa are the lumbar plexus (the trunks of which lie in the iliaco-psoas muscle) and its branches; and the solar, renal, hypogastric, and lumbocortic branches of the sympathetic.

The *fascia iliaca* is attached above to the entire inner lip of the iliac crest. Internally it is blended with the sheath of the psoas at the level of the promontory of the sacrum; and below this point it becomes fixed to the brim of the true pelvis, passing behind the vessels and giving off a thin cellular lamella in front of them.

The *sheath of the psoas muscle* is attached above to the ligamentum arcuatum internum; it encloses the psoas anteriorly (the posterior portion of its envelope being formed by the lumbar vertebræ); it is blended externally with the sheath of the quadratus lumborum; whilst internally it is attached to the anterior common ligament. Inferiorly it is continuous with the fascia iliaca.

The *osseous layer* corresponds with the iliac bones.

**PATHOLOGICAL AND CLINICAL RELATIONS.**—The viscera which have been enumerated above, as being contained in the iliac region of the abdomen, present various diseased conditions, which cannot be satisfactorily diagnosed without a practical knowledge of its anatomical relations, and especially of the fasciæ, sub-aponeurotic, and aponeurotic structures of the iliac region. Thus in the right iliac region the physician meets with tumours and other diseases of the cæcum, vermiform appendix, and lower part of the ileum, including the local lesions of typhoid fever (see CÆCUM, Diseases of; and compare HYPOGASTRIC REGION). In the left iliac region, the diseases of the sigmoid flexure possess equally important relations. The tumours, extravasations, and abscesses, which may commence in the pelvis, frequently make their way into either iliac region; and this is also the seat of morbid conditions connected with the ureter in the middle portion of its course, as well as partly of the pain in renal calculus. In the sub-peritoneal tissue we meet with bloody or urinary infiltrations, fæcal abscesses, and perityphlitic or idiopathic

abscesses independent of any intestinal lesion. Collections of pus beneath the fascia are often dependent on caries of the vertebræ, and may either be confined to the iliac fossa, or lie within the sheath of the psoas muscle (psaos abscess). Again, lumbar abscesses may point anteriorly, and be diagnosed by palpation of the abdominal walls.

This region is the seat of ligature of the common, external, or internal iliac arteries. The pulsation of the two former can generally be felt; and it must be borne in mind that the abdominal aorta pulsates in the left iliac region. The bowel (large intestine) may have to be opened here, in cases where lumbar colotomy is contra-indicated, and in the writer's opinion where it always should be done.

EDWARD BELLAMY.

**ILLUSION.**—A false or mistaken perception of one of the senses, as when a person sees or hears something, and takes it to be something else. The term has been used as synonymous with delusion and hallucination. Illusions may occur in the sane as well as in the insane. See HALLUCINATION.

**IMBECILITY.**—See DEMENTIA; and IDIOCY.

**IMMUNITY** (*immunis*, exempt).—**DEFINITION.**—Immunity to disease may be defined as a condition, natural or acquired, of the animal body, which renders it resistant to the invasion of one or more infective disorders.

**GENERAL CONSIDERATIONS.**—An animal (or man) in such a condition is said to be *protected* against the particular disease from which it is immune. All the disorders in which the question of immunity occurs are *infective*, that is, capable of being transmitted from one animal to another by means of a contagium vivum, virus, or *primary infective agent*. The question of immunity is closely bound up with the study of this primary infective agent, with its morphology, its physiological life-processes, and the modes by which it produces its pathological effects. The notice here taken of these points must of necessity be brief; but they will be found fully described in the article MICRO-ORGANISMS.

**Morphology of the primary infective agent.**—In only a few of the infective disorders in man is the primary infective agent known (see MICRO-ORGANISMS). In these (*e.g.* tubercle, anthrax, diphtheria, glanders, tetanus, relapsing fever, erysipelas, pyæmia, actinomycosis) specific micro-organisms have been found and recognised to be the cause of the disease. In some other diseases—typhoid fever, cholera, and gonorrhœa—the proof that the micro-organisms found are the cause of the diseases is not yet complete; while in one disease (malaria) there is some evidence to show that the primary infective

agent is a low form of animal life—a plasmidium or amœba-like body. Until the identification of the infective agent in these and other diseases (such as scarlet fever, measles, small-pox, &c.) is made, the nature of immunity to them, which is of such great importance to man, must remain unsolved. Of greater importance in immunity is the mode of propagation or division of the pathogenous micro-organisms. Some of these, such as micrococci, the vibrio cholerae asiaticæ, the bacillus diphtheriæ, do not form spores, but multiply by fission; while others form numerous spores, which are very resistant to the external conditions of moisture, light, and heat. On the other hand, these conditions affect the non-spore-bearing micro-organisms, and so diminish their virulence. The physiological life-processes of the pathogenous micro-organism are important to the question of immunity under two headings. For their development and growth these infective agents require a suitable medium, which in any individual disease is formed for them or by them from the fluids of the body. In the interstitial fluids of the tissues, and in the fibrin and proteids exuded from the blood in pathological effusions, these infective agents find suitable media for rapid development and growth, and from such media they effect their second important physiological rôle, viz. of producing poisonous chemical substances, which, circulating in the body, become the means by which the primary infective agent produces the symptoms, both general and specific, of infective disorders. Unless the infective agent find a suitable medium on which to grow, it cannot form its poisonous chemical products, and cannot reproduce the disease of which it is the cause. This fact is of great importance in considering immunity; and why no suitable medium is present in an immune animal will be discussed later as far as in our present knowledge it is possible.

Besides the fact just mentioned of the physiological life-processes of the infective agents, there are others of equal importance connected with the *pathological effects* of these agents. We have to take into account in considering immunity the following pathological problems: (1) *the degree of virulence or attenuation of the virus*; (2) *the localities in which it grows in the body* in natural disease; with which is associated its mode of distribution and its fate in an animal after experimental inoculation into the circulation, under the skin, or into one of the cavities of the body.

**1. Degree of Virulence or Attenuation of the Virus or Infective Agent.** One of the facts about the virus of infective diseases which was surmised by clinical experience and has been accentuated by the scientific work of recent years, is that the virus exists in two forms, one virulent and

the other attenuated. Thus some cases of typhoid fever undoubtedly die from the intensity of the poison, and may exhibit anatomically no greater changes than exist in a milder but still fatal case. Experiments, however, with pathogenous micro-organisms show this fact in a more exact manner. Thus, Pasteur was able to 'attenuate' the bacillus anthracis, so that when injected into a sheep it did not kill the animal, but produced a mild febrile illness. The bacillus of fowl-cholera was attenuated in like manner. It is well known also to bacteriologists that if a virulent culture of bacillus anthracis be kept for some time, it is apt to become weakened in virulence; and one can approximately express the virulence of such preparations as a 'two-days' or 'four-days' anthrax, this being the time in which the preparation would kill a given animal (guinea-pig or rabbit) when subcutaneously inoculated.

Again, if a virulent culture of the bacillus diphtheriæ on agar be kept for some months, even in this state it will be found to become less virulent, and will finally die. Other examples might be given of similar results with other pathogenous micro-organisms. A distinction has been drawn between an attenuated virus and a virus which is simply weakened by age or partial drying, &c. It is said that the attenuated virus always breeds true in artificial cultivation or in the animal body, whereas the slightly toxic virus may by manipulation be made virulent again. It is, however, at present impossible to generalise on this point. The methods of attenuation which are in use are: (1) the passage of a continuous current of air through the liquid in which the bacterium is growing; (2) the successive inoculation of the virus through a series of different animals or of a series of refractory animals; (3) the application of heat and of antiseptics to the virus. Thus many pathogenous bacteria are attenuated by keeping them at a temperature of 45°-50° C. for several hours. Essential oils (such as that of mustard) will also weaken and, if in large quantity, kill pathogenous bacteria. The difference between the attenuated and virulent micro-organisms is not that they produce different chemical products, but that the attenuated have a shorter existence; that, even when they have outside the body the nutrient medium the most favourable to their growth, they stop growing and degenerate. This has been found by some observers also to occur in the body itself—in the case, e.g., of the bacillus anthracis when the attenuated organism has been injected into a sheep. This tendency of the attenuated organism (the vaccine) to die under favourable conditions is an important point in connexion with immunity. The natural history of epidemics has also shown that the virus may be a weak or a virulent one; it is possible that in a mild epidemic we have to do

with an attenuated virus, in a virulent epidemic with an intensified one. It is, however, not a complete explanation of the facts, as we shall see.

A weak virus can in some instances be intensified or made virulent again. This has been done by repeated cultivations of the micro-organisms on suitable nutrient media, or by passing the virus through a series of animals—*e.g.* inoculating the first animal with the micro-organism, inoculating the second animal with the blood or tissues of the first, and so on, for five or ten times. Cultivations of the micro-organism from the last animal are found to be very virulent. The vibrio cholerae asiaticæ may be intensified by this method, using guinea-pigs as the animals. Ordinary cultivations of the vibrio are not very toxic, but the intensified virus causes rapid death. A similar micro-organism (vibrio Metschnikovi) has been intensified in like manner.

The fact of the existence of a virus in two states—an attenuated and an intensified—has a bearing on many experimental observations on preventive inoculation, which have led to hasty conclusions, owing to the fact that the virus used to test vaccination was an attenuated and not an intensified one. It is evident that a procedure which would protect against the former might be ineffectual against the latter, and indeed so it has been found.

**2. Seat of Growth of Pathogenous Micro-organisms in the Living Body.** According to the locality of growth of the virus, infective disorders may be divided into three chief classes:—

(*α*) In one, the infective agent distributes itself throughout the tissues of the body. Anthrax is a type of this class. Wherever inoculated, under the skin, into the peritoneal cavity, into the circulation, or into the anterior chamber of the eye, the bacillus anthracis passes to all the fluids and tissues of the body. Glanders is another example: from the usual seat of inoculation, the nasal mucous membrane, the bacillus enters the circulation and the lymph-stream.

(*β*) In a second class, the infective agent becomes limited to the seat of inoculation, producing its effects by the diffusion into the circulation of its poisonous chemical products. In diphtheria, *e.g.*, the bacillus is limited to the membrane, and is never found in the organs of the body; in tetanus, also, the bacillus is limited to the seat of inoculation. In typhoid fever and in cholera it is also extremely probable that the virus is limited to the intestinal walls, never entering the circulation.

(*γ*) There is a third doubtful class, in which the infective agent, although entering the circulation, is limited to the formed elements of the tissues. Thus, in leprosy the bacillus is confined almost entirely to the interior of

the cells of the leprosy nodule; in malaria, the plasmodium is in the red blood-corpuscle.

It is evident that the question of immunity must be different in these three classes of infective disorders, and that it is fallacious to generalise as to the mode of production of immunity from experiments performed on any individual infective disorder. It is noteworthy, for example, that some of the infective agents, which are localised in their growth (class 2), are incapable of living in the tissues of the body. The bacillus diphtheriæ, if injected into the circulation of the rabbit, rapidly dies, although it will kill the animal.

Having shortly discussed the nature and effects of the agents which cause infective disorders, it is necessary to see more particularly how the results affect the question of immunity.

Immunity may be (1) *natural*; or (2) *acquired*.

**1. Natural immunity.**—Natural immunity includes those cases in which man or any animal does not contract in the ordinary course of events a particular infective disorder, as well as those cases (some of which are now well known) in which it is found difficult, and, in fact, impossible, to infect an animal with a particular disease. Such cases as these are the best examples of natural immunity. The injection, for example, of a virulent infective agent into one animal will not kill it, while a similar or even smaller dose into an animal of another species will with certainty be fatal. Warm-blooded animals, therefore, including man, may, from this point of view, be divided into two classes—those which are *susceptible* to a particular infective disorder, and those which are *refractory*.

The subject will be discussed chiefly from the animal point of view, as this may help to explain the natural immunity to some disorders which is enjoyed by man. The discussion of natural immunity in man includes many other factors besides the condition of health of the tissues of the body. The questions of exposure to infection, of susceptibility to disease according to age, of the influence of climate and temperature, would come more properly under the heading of *Ætiology of Disease*. There remain, however, these facts: Man is subject to certain infective diseases (*e.g.* typhoid fever, cholera, scarlet fever, measles, diphtheria, &c.), which are not prevalent in animals. Animals (domestic and farm) are subject to certain diseases (*e.g.* swine-plague, contagious pleuro-pneumonia, distemper, black leg, &c.), which do not affect mankind, and, lastly, there are certain infective diseases from which both mankind and animals suffer, viz. tuberculosis, anthrax, hydrophobia, glanders and farcy, pyæmia and pus infection, tetanus. In many of these diseases the primary infective agent is unknown; the

question of immunity in them must therefore remain practically open. It is impossible, for example, to discuss the question, in the present state of knowledge, as to why animals do not get cholera or typhoid fever. In others, however, the virus is known, and in some well studied. Special interest attaches to the question of immunity in those diseases which are natural diseases in both man and animals, or which are natural diseases in man and which are infective to animals by means of inoculation of the virus, although these may not suffer from them in the natural course of events. We have in such diseases opportunities for experimentally testing the conditions producing immunity. *Tuberculosis*, of which the primary infective agent is the bacillus tuberculosis, is a natural disease in man, cattle, and pigs. Goats, sheep, horses, and dogs are practically immune to it. It is difficult, although not impossible, to produce tuberculosis in dogs by the injection of the tubercular virus. Positive results are obtained with very large doses injected into the peritoneal cavity. *Anthrax*, a natural disease in oxen, sheep, and horses; but experiment has shown that adult white rats, Algerian sheep, dogs, and pigeons are difficult to infect, and are in fact refractory to the disease. To *diphtheria*, a natural disease in man, rabbits, guinea-pigs, dogs, cats, monkeys, and cows are susceptible; while rats and mice are refractory, being naturally immune to the bacillus diphtherie. To *cholera* no animal can be said to be susceptible, although the intensified vibrio cholerae asiaticæ (Koch) is fatal to dogs and guinea-pigs, but is not so active in rabbits. In many instances this natural immunity, as tested by inoculation, is not an absolute quantity: it is, in fact, a quality possessed in varying degree by the several animals called refractory to a disease. Thus dogs may be regarded as practically immune to the bacillus tuberculosis, even large doses inoculated subcutaneously producing no effect. To the bacillus tetani they are immune to a less degree: a large dose subcutaneously will be fatal, while a small dose will produce a mild illness ending in complete recovery. Adult white rats are refractory to the bacillus anthracis only in a minor degree. If a large number be inoculated with a virulent virus, less than half will die of the disease, the remainder showing only a passing illness or no illness at all. Pigeons, too, can be killed with the bacillus, although many survive and show a lesion at the site of inoculation. It is asked, What is the reason of this immunity—what physiological difference is there between the cow and the dog, that the former is very susceptible to tuberculosis, the latter eminently refractory? or between the French, Russian, or English sheep and the Algerian, or between the young rat and the adult white

rat, that the former acquire anthrax and the latter not? or that a well-bred pig is susceptible to swine-erysipelas and a mongrel refractory? The investigation of these facts is the point of the question under discussion, and has led to the discovery of important facts bearing on immunity to disease.

2. *Acquired immunity*.—In man there is perhaps some evidence to show that one attack of an infective disorder protects the individual against subsequent attacks; but the truth in the matter is difficult to ascertain owing to the absence of correct records. There is no doubt that an attack of small-pox is protective, as the results of the now obsolete inoculation showed. It is, however, an assumption that one attack in every case of an individual disease confers immunity. There is, indeed, some evidence to show that an attack of erysipelas or of tuberculosis not only does not confer immunity, but actually predisposes to subsequent attacks. But this point of view of the subject is so hedged by difficulties that little good would come from a discussion of it. It will be best, therefore, to pass on to the question of immunity acquired experimentally. The basis of most of the experiments performed is that an attack of a disease protects against a subsequent attack, and that, although the first attack must be mild, it must not be too mild, or no protection is afforded. It will not be too much to say that all recent experiments in immunity have been based on the idea of the protection afforded by vaccination against small-pox. See SMALL-POX; and VACCINATION.

**Preventive Inoculation.**—*Preventive inoculation* has been claimed to have been performed by three classes of products—(1) by the *attenuated virus* of the disease; (2) by the *chemical products* of the virus or primary infective agent; (3) by *chemical substances which are not the products of bacterial growth*.

(1) *Immunity conferred by the attenuated virus*.—There are now several examples of this known to experimental science. The best is that of anthrax (Pasteur). The bacillus anthracis when attenuated may be obtained as in two kinds of vaccine, called *premier* and *deuxième vaccin*. The *premier vaccin* is injected subcutaneously in sheep, and is followed after an interval by the injection of the more powerful *deuxième*. The animal is after a time, but not immediately, immune against anthrax, and the immunity lasts probably nine or ten months. The changes which the inoculation produces will be discussed later. Pasteur similarly attenuated the bacillus of fowl-cholera, and thus manufactured a vaccine against the disease. It has not been found possible to attenuate the virus of all diseases so that a vaccine can be prepared. The attenuated cultures of the bacillus tetani do not confer immunity against subsequent inoculations of virulent cultures.

of the bacillus; rabbits, however, have been made immune by injecting the bacillus and treating the animals subsequently with trichloride of iodine. This substance prevents the fatal result, but appears to allow the bacillus to grow sufficiently to produce immunity. Tizzoni and Catani, utilising the fact that dogs and pigeons are refractory to tetanus, obtained a vaccine from the blood of these animals *after* they had been inoculated with the bacillus tetani, which produced an illness but not death. This vaccine will be the subject of further consideration. The bacillus diphtheriæ which has been attenuated simply by the *age* of the culture does not act as a vaccine. Brieger and Fränkel have, however, found that a three-weeks-old broth-culture of the bacillus, when warmed to 60°–70° C., protects guinea-pigs against a subsequent inoculation of the virulent bacillus, provided that the second inoculation does not take place until fourteen days after the first. Behring also obtained a diphtheria vaccine by growing the bacillus with trichloride of iodine. By different methods, therefore, an attenuated virus can be obtained which will act as a vaccine. The method employed by Pasteur for obtaining an attenuated virus of hydrophobia is described elsewhere. See HYDROPHOBIA.

(2) *Immunity conferred by the chemical products of the virus or primary infective agent.*—There are a few examples which show that the chemical products of the virus may afford a certain amount of protection against a subsequent inoculation of the virulent virus. Thus, with anthrax, the bacillus was grown in solution and killed by heat; the solution remaining acted as a vaccine against anthrax (Toussaint, Chauveau). Similarly with the bacillus pyocyaneus (the bacterium of blue pus) and with the vibrio cholerae asiaticæ. The products of the bacillus tetani can serve as a vaccine if treated by heat. Cultures of the bacillus are filtered to remove the micro-organism and subsequently heated to between 50° and 60° C.; when the liquid is repeatedly injected into rabbits the animals are rendered immune to the disease (Vaillard). Instead of heat, a solution of iodine may be used to weaken the poison. To hog-cholera, the infective agent of which is the *cocco-bacillus suum*, immunity may be produced in rabbits by heating to between 54° and 58° C. the blood of animals killed by the micro-organism. It is probable in this case that the vaccine is really the chemical products of the *cocco-bacillus*. In many instances of chemical vaccination the vaccine is inadequate, and the injection of vaccinating fluid must be regularly performed before protection against the disease is obtained. It has been stated that one class of bacterial products, viz. albumoses, is very efficient as a vaccine, but this has not been confirmed.

(3) *Immunity conferred by chemical substances of a non-bacterial origin.*—Most of the facts ascertained under this heading are still *sub judice*, but they promise a fruitful field for investigation. Wooldridge showed that some protection in rabbits was afforded against anthrax by the intravenous injection of a solution of tissue-fibrinogen, a peculiar form of globulin obtained from certain tissues; and that this effect of protection was intensified if the bacillus anthracis was previously grown a short time in the solution of fibrinogen, and removed before the solution was used as a vaccine. These experiments were the starting-point of many others which tended to show that there existed a class of bodies provisionally called 'defensive proteids,' which were vaccines for individual infective disorders. Thus experiments have been performed to show that the blood and spleen of animals naturally refractory to a disease contain chemical bodies ('ferments' or 'proteids') capable of vaccinating against the disease. The blood of the dog, an animal refractory to anthrax, *e.g.*, has been said to contain a 'ferment' which is a vaccine against the bacillus anthracis; and the spleen of the rat, which is also refractory to the same disease, is said to contain a vaccinating globulin. These experiments have not been confirmed, and the vaccinating influence of these bodies is very slight, if it exists at all.

**Changes occurring in the Animal Body, the Result of Inoculation.**—The injection of the attenuated virus or of the bacterial chemical products produces a definite illness which is transient; it consists in a rise of body-temperature, lasting a variable time, and a few general symptoms, described as malaise and lassitude, &c. The intimate chemical changes are but little known. Some of the experiments which have been performed are, however, not without interest. Immunity to the bacillus tetani has been conferred on rabbits, as previously stated, by treating the animals with trichloride of iodine; the animals were subsequently inoculated with the bacillus. The blood drawn after some days from the carotid artery was found not only to confer immunity against the bacillus tetani in mice, which are very susceptible to the disease, but to destroy the tetanus chemical poison (Behring and Kitasato). Pigeons and dogs are refractory to tetanus; inoculations with the bacillus made the animals ill, but did not cause death, the animals being after treatment immune against the injection of very virulent cultures of the bacillus. The blood-serum of these protected dogs killed the tetanus poison (outside the body) and conferred perfect immunity on another dog and on mice, but did not protect rabbits and guinea-pigs against inoculations with the bacillus (Tizzoni and Catani). In the case of rabbits protected

against hog-cholera, the serum of the blood acts as a vaccine against the *cocco-bacillus* suum, but it possesses no bactericidal or anti-toxin property, and does not attenuate the bacillus (Metschnikoff). This change in the blood shown in these experiments with tetanus or hog-cholera is a remarkable one, and cannot at present be expressed in any chemical terms. The experiments require confirmation. The fate of the attenuated virus in the body has been studied with somewhat fruitful results. In anthrax it may be limited to the site of inoculation, or it may to some extent distribute itself over the body, especially in the spleen. In either case it dies, becoming degenerated. A similar effect has been observed when the virulent virus has been inoculated into a protected or a refractory animal. It is noticeable that in many cases protective inoculation is effective for only a very short period, reckoned by days, while in other cases there is no protection until *after a certain period* reckoned by days, and that 'vaccinated' animals inoculated with the virulent virus before this period has elapsed actually show a greater susceptibility to infection than the non-vaccinated.

**Nature of Immunity.**—The intimate changes, both chemical and anatomical, occurring in the bodies of immune animals are imperfectly known. It is not, therefore, surprising that an explanation of the real nature of immunity is not at present forthcoming. Both in natural and acquired immunity there is an antagonism between the body and the invasion of the virus or primary infective agent—an antagonism which results in the victory of the tissues. In susceptible animals there may be some antagonism, but the virus is triumphant. The question is, What is there in the body of an immune animal which antagonises the growth of the virus, preventing it producing its chemical products, which are the cause of death? Does the antagonistic agent reside in the fluids of the body or in the cellular elements? Is immunity a question of chemical antagonism or one of vital antagonism—the combat of cells against the virus (*phagocytosis*)? Much harm has undoubtedly been done by generalising from isolated experiments. The virus acts in a particular manner in individual infective disorders: in anthrax it is diffused through the body, in diphtheria it is localised to the throat, and in tetanus to the seat of inoculation, as already stated. It is, therefore, improbable that the explanation of conferred immunity is the same in all disorders; and, as we have seen, no single method of conferring immunity is common to all infective diseases. With vaccines which consist either of the attenuated virus, or of the chemical products of the virus, it is important to bear in mind the following considerations. The attenuated

virus injected into an animal produces an illness by means of the chemical products it forms, which are carried through the blood and lymph-stream over the body. These products are formed slowly, and are eliminated slowly, so that their effect on the tissues is a prolonged one, lasting probably after all evident symptoms have subsided. These products, at any rate in the case of the bacillus anthracis, are of the same nature as those produced by the virulent bacillus; they may, therefore, by their specific action prepare the tissues for receiving a larger dose of the poison—they may accustom the animal to the bacterial poison. This idea is rather confirmed by the fact that in some cases (*e.g.* diphtheria) the animal is not immune until some time after vaccination; if inoculation with the virulent virus is made before the period has elapsed, the animal dies *more* quickly than usual. In the case of some chemical poisons this action is well known, as in the establishment of the 'morphine-habit,' and it has been demonstrated with snake-poison (Sewall) and the poison of *Abrus precatorius* (jequirity). Both these poisons bear a close relation to some bacterial poisonous products; and in both, repeated small doses protect an animal against a single large and fatal dose. With a living virus the case is, however, different: the effect of a chemical poison lasts but a short time; that of a living virus is constantly increasing until the susceptible animal dies. We are still, therefore, confronted with the question why the virulent virus does not produce its fatal results in a protected or refractory animal. No bacterium will flourish unless it has a suitable medium; and pathogenic micro-organisms are peculiarly sensitive to their surroundings. The vaccine may change the cultivating medium usually existing in the susceptible animal, so that the virulent virus will not develop; and this is in accord with the fact that experimental vaccination confers, as a rule, a very temporary protection. The isolated experiments which have been adduced to prove that the cultivating media (*i.e.* the fluids of the body) are so altered in both natural and acquired immunity that they are inimical to the primary infective agent, are not very satisfactory. The following examples may be quoted as showing that no explanation of the problem is at present complete from a study of the experiments already performed. The blood-serum of the rabbit, an animal susceptible to anthrax, kills the bacillus anthracis; while the blood-serum of the dog, an animal refractory to anthrax, is not harmful to the bacillus. Similarly, the serum of a vaccinated sheep is not more harmful to the bacillus anthracis than that of an unvaccinated. The experiments with the serum of rabbits protected against hog-cholera have already been quoted. Such experiments are, however, not conclu-

sive, as serum does not exist as such in the living body; and immunity may still be found to be in part effected by altering the cultivating medium in the living body. In one case of vaccination (that for tetanus) there is a profound change in the blood; the experiments have already been quoted. In them it was shown that after a dog had been inoculated with the tetanus bacillus its blood-serum had the power not only of conferring immunity on another dog and on mice, but of killing the tetanus chemical poison. The supposed substance has been called tetanus 'anti-toxin.'

**Phagocytosis.**—The chemical examination of immune animals is of great importance; but the facts now known are so inconclusive that it is impossible to state their exact bearing on the question of immunity. An anatomical study of the tissues of immune animals has led to the discovery of interesting facts, dealing chiefly with the fate of the virus when it is injected into protected animals or those naturally immune. The theory of phagocytosis is held by Metschnikoff to explain the phenomena of natural and acquired immunity. Phagocytes are cells which have the power of taking into their interior solid particles, dead or living; and are of two kinds. One form, called *microphages*, are the leucocytes or migratory cells of the body, with a lobed or multiple nucleus; the other kind, *macro-phages*, are such as 'the fixed cells of connective tissue, the lining cells of the pulmonary alveolus, and all other cells capable of taking in solid particles, and containing a single large nucleus less easily stained than that of the microphages.' According to this theory, natural immunity is due to the fact that the phagocytes take into their substance the virulent virus when it gains entrance into the body and destroy it. In animals susceptible to any particular disease, on the other hand, the phagocytes, although they may take in the virus, are unable to destroy it; it gains the upper hand and produces the disease. In acquired immunity the phagocytes are so affected that they are enabled to successfully combat the micro-organism and destroy it, losing this acquired property when the animal becomes again susceptible, as it usually does. The fact that leucocytes ingest particles has long been known; they are active agents in the normal absorption of fats from the intestine, and in normal conditions they have been found to contain some of the numerous bacteria of the intestinal contents. Metschnikoff has made an extensive investigation into two cases of natural immunity, viz. that of the pigeon and of the adult white rat against anthrax. Although the immunity is not perfect, many animals recover from the inoculation of virulent bacilli anthracis; and in such cases he has stated that there is an abundant phagocytosis, the microphages and macrophages containing the majority of

the injected bacilli. When abundant phagocytosis does not occur, the animal becomes affected with anthrax and eventually dies. The bacilli injected are not solely the dead ones, but are in many instances at least living.

When protected animals, moreover, are inoculated with a virulent virus, Metschnikoff states that an abundant phagocytosis occurs; for example, in animals vaccinated for anthrax by the attenuated virus. Also in rabbits vaccinated against hog-cholera in the manner already described, when they are inoculated with the virulent cocco-bacillus suum, these bacilli are taken up by the microphages and macrophages and destroyed by them. In this case, although the blood-serum is a vaccine, it cannot kill or attenuate the bacillus; and Metschnikoff concludes that it probably stimulates the phagocytes. In other cases it is supposed that the chemical products of the virus (such products being the vaccine) attract the leucocytes to the spot, and stimulate them to attack the bacterium. Some chemical substances are known to attract leucocytes, others to repel them; the former are described as exerting a *positive chemotaxis*, the latter a *negative*. Chemotaxis is an observed phenomenon; but as a theory in connexion with phagocytic immunity it fails to explain what gives the power to the leucocyte and other cells of the immune animal to destroy the virulent virus. The exact rôle which phagocytosis plays in immunity, and that which the chemical constituents of the body play, must at present remain unsettled.

**IMMUNITY AND CURE OF DISEASE.**—The question of immunity is closely connected with that of the cure of disease. In the present state of knowledge, but little, except of a theoretical kind, can be said on this point. Immunity is the prevention of the growth of a virus after it has been introduced; cure is the prevention of its further growth after its development has commenced and the symptoms of disease have already appeared.

**Antagonism of infective agents.**—Only brief notice can be taken of this point. The growth of two infective agents may be concurrent in the same animal body. They may or may not neutralise each other. The concurrent or after development of pus micro-organisms in connexion with tubercle, diphtheria, and typhoid fever is known—here there is no antagonism. There are, however, several experimental examples of antagonism of micro-organisms. Thus the bacillus pyocyanus or its products will counteract the bacillus anthracis; and it has been stated that the bacillus pneumoniæ (Friedlander) and the micrococcus erysipielitis will each counteract the bacillus anthracis. Such isolated facts as these are not without importance, as they indicate one method by which it may in the future be found possible to treat an infective disorder.

SIDNEY MARTIN.

**IMPETIGINODES.** — Impetiginous; that is, having the character of impetigo; hence, *eczema impetiginodes*. See **IMPETIGO**.

**IMPETIGO** (Lat. A scabby eruption).—**SYNON.**: *Impetigo Contagiosa*; *Porrigo Contagiosa*; *Pustular Dermatitidis*.

**DEFINITION.**—An inflammation of the skin attended with a vesico-pustular eruption, which is easily reproduced by inoculation.

**ÆTIOLGY.**—One of the most interesting facts with regard to pus formation in the skin is the ease with which it can be inoculated, producing an eruption which under favourable conditions develops into impetigo. The important point is, that the source from which the pus is derived does not much affect the character of the eruption; it may be obtained from another pustular eruption, a whitlow, or even a boil, or some other source; and, provided the inoculation is made under favourable conditions for its development, a vesico-pustular eruption on the skin is the result. It is true that some forms of pus are more active or virulent than others; but the general fact holds good, that all pustular eruptions are inoculable. From this we should expect that impetigo would be a local affection, and more or less of traumatic origin; and this is generally the case. The development of pustules may occur in the course of many diseases of the skin, such as eczema, scabies and morbus pedicularis, but in all these cases the formation of pus is not a necessary part, but rather an accident of the inflammatory process. It will be seen from the above that impetigo is not a well-defined disease like psoriasis or zona, but simply a local pustular eruption. As all forms of impetigo are inoculable, it is not necessary to draw any distinction between impetigo and 'impetigo contagiosa.' It would be impossible to describe every circumstance under which impetigo may occur. In its best-known and most common form it is met with on the scalp and face of the poor and dirty children of our large towns, and is often associated with pediculi capitis. In these cases the pustules are so quickly broken by the scratching of the child that they are seldom seen in a perfect state. On examining the head we find scabs, which mat the hair together and cover a superficial excoriation. The pus is often transferred from the scalp, by scratching, to the nose and lips, in which region the real character of the eruption can be more easily examined.

**DIAGNOSIS.**—The following points will especially distinguish impetigo from ordinary eczema: (1) Each spot appears as a single vesicle or small group of vesicles, without the surrounding skin being red and inflamed, very unlike the first appearance of a patch of eczema; (2) it is easily inoculated; (3) it is usually unsymmetrical, and more distinctly a local affection than eczema; (4) it is un-

attended with itching, unless complicated with pediculi or eczema; (5) it is almost entirely a disease of the poor and dirty classes, and chiefly confined to children; (6) the children who suffer long from it are always pale and badly nourished; (7) pediculi capitis being the most common cause, it follows that the occiput is the most common primary seat of the disease; it is thence transferred to the nose and corners of the mouth, or from person to person; (8) when the occiput is affected, the glands in the neck are quickly enlarged.

**TREATMENT.**—Impetigo of the scalp is easily cured by white precipitate ointment, which heals the sores and at the same time removes the cause. On the body the best local application is a thick lotion made of calamine, zinc oxide, and lime-water; this should be painted on with a brush and allowed to dry on, so that a crust is formed. If pus collects under this crust, it should be removed, and the excoriated surface painted again with the lotion. When the crusts remain firmly attached, the cure is soon effected. Tonics, especially iron and wine, are useful; and attention must be paid to diet.

ROBERT LIVEING.

**IMPETIGO CONTAGIOSA.** — See **IMPETIGO**.

**IMPOTENCY** (*in*, not; and *potens*, capable).—**SYNON.**: Fr. *Impuissance*; Ger. *Impotenz*.

**DEFINITION.**—Impotency may be defined as incapacity in the male for copulation, and is to be distinguished from sterility, that is incapacity for procreation. Cases of impregnation without penetration have been recorded, but, as a rule, impotency involves sterility. A man may be sterile without being impotent.

Impotency may be complete or partial, permanent or temporary.

**CAUSES.**—Deformity, congenital or acquired, of the external genitals may produce impotency. Extreme degrees of epispadias or hypospadias, or a permanent rudimentary condition of the penis, may cause it. Elephantiasis of the penis or scrotum, a large hydrocele of the tunica vaginalis, a large irreducible scrotal hernia, or tumours, benign or malignant, of the penis may mechanically hinder penetration. Cicatrices, resulting from wounds or disease of the penis, a rigid contracted frenum præputii, or even a permanently adherent prepuce may cause such distortion of the organ during erection as to render copulation impossible.

Usually, however, impotency is of nervous origin. Erection of the penis is caused by an increased inflow of blood combined with diminished outflow. The latter is effected by compression of the dorsal vein at the root of the penis by fibres of the accelerator urinæ

muscle. The increased inflow is due to relaxation of the muscular fibres in the supplying arteries and in the cavernous tissue of the penis. This relaxation is produced by the *nervi erigentes*, which transmit impulses originating in the ganglion cells of the erection centre in the lower part of the spinal cord. This centre is itself excited either reflexly by stimulation of peripheral nerve-endings, or perhaps sometimes automatically by the quantity or quality of the blood supplied to it. Partial or complete loss of the glans penis may thus, by destruction of the nerve-endings, impair or prevent the reflex excitation of this centre. But it is also connected by fibres in the spinal cord with higher centres in the cerebrum, which can inhibit or excite its action. Impotency may therefore result from disturbance of the due relation between the several parts of this complex nervous apparatus, or from injury, disease, or degeneration of it.

Impotency of nervous origin may be classified as *psychical*, *irritable*, and *paralytic*.

*Psychical* impotency results from the undue predominance of the cerebral inhibiting centres. In newly married men, who have previously led chaste lives, it may be induced by want of self-confidence—excessive apprehension of inability to perform well the duty of the sex, a feeling which is often greatly aggravated by the perusal of the productions of quacks and other impostors. In such cases a tonic may be prescribed, and the patient be directed to abstain from all attempts at intercourse whilst under treatment; and we may rest satisfied that not many days will pass over before nature asserts her empire. Encouraging assurances will do more in effecting a cure than stimulating medicines, or any sort of medical treatment. A single success banishes at once all fears, and gives security for the future. But in cases where excessive venery or masturbation has been practised, there may be diminished excitability of the lumbar centre. The condition is usually of very brief duration. More rarely impotency may be experienced in attempted intercourse with one person and not with others. Possibly the inhibition in such cases is reflex, and excited by some peculiarity in the female.

*Irritable* impotency is the result of undue excitability of the nervous centres. It occurs after habitual masturbation or excessive venery. The semen is ejaculated before penetration has been effected, and the erection speedily subsides.

*Paralytic* impotency may be due to injury, disease, or degeneration of the nervous centres. The numerous recorded cases where impotency, permanent or temporary, has followed injuries to the head or back, can only be explained by the assumption of some affection of the nervous system. Diseases

and injuries of the spinal cord, producing paraplegia, necessarily prevent active copulation; but if the spinal lesion be above the erection centre, erection and connexion might be possible, the man being a passive agent. Cases are recorded where even impregnation has occurred in such conditions.

Some drugs are credited with producing impotence. Arsenic, according to Charcot, when taken for a long time, has this effect, but virility is regained when the drug is no longer taken. Opium-eating and excessive indulgence in tobacco have been similarly accused. Diuretics, as the nitrate of potassium and bicarbonate of sodium, are well known to act as anaphrodisiacs.

Virility is more or less affected by constitutional diseases. Few complaints have greater influence in impairing the generative functions than those of the kidney. In irritative dyspepsia, with deposits in the urine of earthy phosphates or oxalate of lime, there is generally some inability. Impotency in these cases is only one of the manifestations of defective assimilation and depressed vital force. In diabetes and albuminuria the reproductive organs are weak and often quite inactive, but may regain tone as the organs are restored to a healthy state. Gout and hepatic disorder also may temporarily produce a similar condition.

Impotency sometimes occurs in middle life without any obvious cause. In such persons the writer has noticed a constitutional change similar to that which occurs in eunuchs, but less marked. They have been observed to grow sleek and corpulent, to have a scanty beard, and to be indisposed to active muscular exertion. In general they evince no unhappiness at their altered condition. In atonic impotency, the external organs afford indications of the want of power. Not only are the testicles soft and flaccid, from the absence of blood in the vessels and sperm in the tubes, but the penis is small and shrivelled, and the glans relaxed. The scrotum is also loose. These parts are pale, feel cold, and their sensibility to contact is diminished.

**TREATMENT.**—Certain medicines, reputed to possess the property of stimulating and invigorating the sexual organs, have been classed as *aphrodisiacs*, and some of them are said to be used, especially in the East, by the sensualist, to excite the organs when exhausted by satiety and excess. Several act on and stimulate the urinary organs, and thereby give temporary power to the function of erection; but they produce little or no effect on the special sexual organs. They determine blood to the penis, and cause morbid erections, without any voluptuous sensations and desires. Such appears to be the character of the influence produced by cantharides, the

most common of this class of medicines, and the chief ingredient of quack medicines for impotency. There are, however, certain cases in which cantharides is useful. In an atonic state of the organs, in which the erections are feeble, unstable, and insufficient, ten to fifteen minims of the tincture may be given every three or four hours for a short time before the occasion arises for the exercise of the sexual functions. Diluted phosphoric acid, phosphate of iron, strychnine, and ergot of rye are remedies which may be given in impotency. The conditions to which these aphrodisiac remedies are chiefly applicable is when the intromittent organ is but feebly excited, and does not maintain the physical state necessary for penetration, during the period of congress. Such torpidity may exist in persons in whom desires are at times strongly felt, and the functions of the testicles properly performed. In these cases, also in timid persons, and in others whose organs are inexcitable from long disuse, stimulating treatment may conduce to success, and ensure confidence for the future. But these remedies exert no influence in a constitutional apathy of the sexual functions. They have rarely, also, more than a temporary effect; and in persons advanced in life, when the parts, having fulfilled their office, are experiencing their natural decline, they operate injuriously, and tend to produce congestion of the prostate and local disease. In those cases in which the sexual organs are weakened or prematurely exhausted by excess, they are likewise hurtful, as well as fruitless. After such abuses a period of repose is required; and by the avoidance of all sources of excitement, and by diet and remedies adapted to invigorate the body, such as the preparations of iron, a gradual restoration of the procreative functions may be hoped for.

Electricity is a remedy of some efficacy in certain forms of impotency. Interrupted currents (faradic) may be passed in two directions, from the perineum to the glans penis in cases of defective erectile power, and from the groin along the spermatic cord to the testicles in cases where these organs are soft and flaccid, and where secretion is languid. The results are often disappointing. Still, a few applications of the electric current, by rendering the glans more sensitive, may cause a more persistent distension of the organ under the natural excitement. Electromagnetism sometimes succeeds in impotency of this character in rousing a dormant power, causing secretion to be resumed and erections to return. The special treatment required in cases due to injury of the central nervous system, after recovery from head-symptoms, is the use of electricity, applied from the occiput along the spine. See STERILITY IN THE MALE.

T. B. CURLING. JEREMIAH MCCARTHY.

**IMPULSE** (*impello*, I thrust forwards).—A sensation of a stroke communicated to the hand, for example, by the action of the heart or by the pulsation of an aneurysm; or by the sudden movement of a fluid when agitated in any way (see PHYSICAL EXAMINATION). The term is also employed in connexion with a mental condition in insanity. See INSANITY, Varieties of.

**IMPULSIVE INSANITY**.—See INSANITY, Varieties of.

**INCARCERATION** (*in, in*; and *carcer*, a prison).—That condition of a hernia in which it cannot be reduced, on account of obstruction at the neck of the sac or from some other cause. See HERNIA.

**INCOHERENCE** (*in*, not; *con*, together; and *hæreo*, I stick).—Inconsecutive or 'wandering' thought, as expressed in speech. See CONSCIOUSNESS, Disorders of.

**INCOMPETENCE** (*in*, not; and *competo*, I meet accurately).—In its general sense this term signifies inability of a part to perform its functions. It is mainly applied to imperfection in the closing apparatus of an orifice, such as the valves of the heart (insufficiency) or the pylorus. See HEART, Valves and Orifices of, Diseases of.

**INCOMPRESSIBLE**.—Incapable of perceptibly yielding to pressure. Usually applied to the pulse. See PULSE.

**INCONTINENCE** (*in*, not; and *contineo*, I hold).—In medical language incontinence signifies inability to retain the urine or fæces, so that they are discharged involuntarily. See DEFÆCATION, Disorders of; and MICTURITION, Disorders of.

**INCUBATION** (*incubo*, I hatch).—**DEFINITION**.—The development of disease from infecting particles, and the time occupied in the process.

**PERIOD OF INCUBATION**.—The period of incubation is the interval between exposure to infection and the appearance of the resulting disease. It is divided into two stages—those of *latency* and of *invasion*. The one has reference to supposed changes in the infecting particles, the other to noticeable changes in the health of the person infected. The first stage is of variable duration and without definite symptoms; and in the second stage the disease is progressing and the patient is said to be 'sickening for it,' its duration being nearly constant for each special disease. The division is not well-marked. There are changes in the so-called latent period which do not always escape detection, while those of the invasion have often to be considered as part of the disease. Where the latent stage is much prolonged a *dormant* period is inferred, for it is possible that infection may remain in the body for a time dormant. This term is only applicable to

certain conditions under which infection is transmitted; and instead of speaking of the long intervals of inactivity at which epidemics recur as periods of latency or of incubation, it is to these periods of quiescence that the term 'dormant' should be restricted.

The results of septic infection are manifested in the body without any true or definite interval of incubation.

Incubation properly refers only to the latent periods of infection in the acute specific diseases; still it is sometimes convenient to reckon the period of incubation as extending to the full development of the more characteristic signs of each of these diseases.

The duration of this period differs for different classes of infectious diseases, and in a less degree for each disease. It varies within certain limits for the same disease, but is sufficiently constant to afford some distinctive characters useful in diagnosis, and a knowledge of which is essential to preventive medicine. The germs of all the infective diseases are reproduced in the bodies of the sick; to stop infection the susceptible who have been exposed to it should not mix with others till the incubation-period has passed with no signs of illness. Hence it is important to ascertain the laws regulating the incubation of each specific infection, and to define the limits of variation. The invasion-stage of all these diseases is already infectious.

1. *Variola* and *Vaccinia*.—In small-pox, after infection, the eruption occurs in fourteen days, and marked illness begins two days sooner. This is so well established as to afford a guide to the source of infection, by which it may frequently be discovered. Dr. Gregory's experience only furnishes one case in which the interval was prolonged to fifteen days. As short an interval as twelve days may be met with, though rarely, in small-pox modified by vaccination or by a previous attack. Small-pox by inoculation is developed in nine days; a local vesicle appears on the fourth day, there is glandular sympathy on the sixth, and febrile disturbance on the seventh and eighth days. Exactly this course is observed in vaccinia resulting from vaccination.

2. *Varicella*.—Chicken-pox has an incubation-period varying from ten or twelve to fifteen or even nineteen days. The eruption begins with the first symptoms of illness.

3. *Morbilli*.—Measles has twelve to fourteen days of incubation, reckoning to the full rash, or eight to ten days from infection to the sickening. The latent period may be only four or six days, that of invasion may extend to six instead of four days. The general experience of schools and hospitals gives ten days from the eruption in the first cases to the appearance of the rash in the next series of attacks. Measles results from inoculation in seven or eight days; the shortest instance from infection, one of fifty cases traced by

the writer, was eight days; the two longest, sixteen and eighteen days; but it may possibly extend to twenty-one days.

4. *Rubella*.—Rubella has an incubation-period of not less than ten to fourteen days, frequently extending to seventeen or twenty-one days. A short illness, not exceeding one day, precedes the rash.

5. *Mumps*.—Mumps usually takes from fourteen to twenty-one days for incubation; the shortest period is ten days; the longest that has come under the writer's observation was twenty-two days. Symptoms referable to the invasion-stage may be noticeable for a week before the swelling of the parotids; the latent stage may continue from eight days to twenty.

6. *Pertussis*.—This generally has one week, or even two, of incubation before the first febrile and catarrhal symptoms appear; there is often a latent period of only four or five days, as some cough may precede the fever. The distinctive cough is seldom heard till after the second week; the shortest period in which it has been known to occur is eight days.

7. *Influenza*.—Influenza has a short incubation, from a few hours to two or three days.

8. *Scarlet Fever*.—Scarlet fever has a short incubation-period. From three to five or six days is the time in which the disease is usually declared; it may appear in less than two days; the longest interval is seven or eight days. There are instances where exposure to this infection has produced no effect until after some accident or surgical operation, the rash then appearing in from three to five days. Supposing a previous infection, twelve days would not be too much to allow for such possible extension of the incubation-period. After removal from a source of scarlet fever, those who have no symptoms of the illness within one week will generally escape.

9. *Diphtheria*.—Diphtheria may be developed in three or four days; an interval of from six to eight days is not infrequent. Sometimes two or three days' fever precedes the first local signs; or these may appear at the very commencement, and the incubation be reduced to a single day. On separating the healthy from the sick, more than a week—possibly a fortnight—should elapse before immunity can be predicated.

10. *Typhoid Fever*.—In enteric fever, the occurrence of the first symptoms, in a large number of cases traced by Sir George Buchanan, was eleven days after the operation of the cause, many other cases occurring two or three days later. Dr. Hunter, of Linlithgow, gives one case, with a single definite exposure, where the prodromata occurred in the early part of the third week, with a rigor at the end of it, twenty-one days elapsing before the fever was marked. Certain interruptions to the febrile process may still

further prolong the interval. An incubation-period of only five days has been noted, once from contaminated milk, and once from infection. There are instances of an eight days' incubation when some of the poison had been inhaled.

11. *Typhus*.—Typhus has, in the great proportion of cases, twelve days of incubation. The late Dr. Murchison gives three cases that have exceeded this period by two or three days, and one of twenty-one days; ten of his cases fell short of it by two, six, or eight days; one of these did not exceed two days; and in two of them the latent period must have been a few hours only. The distinctive rash appears on the fourth or fifth day of illness.

12. *Relapsing Fever*.—This fever has five days of incubation; this exactly agrees with the life-history of the *spirillum* observed in the blood during the fever. The incubation-period may, however be prolonged to seven, nine, or twelve days, or shortened to two days. Many cases have followed almost immediately on exposure to a concentrated infection.

13. *Plague*.—Plague is communicated in from two to five days. The fever may commence on the first day, the glandular swellings on the third, or sometimes glandular tenderness begins with the fever. A period of four days suffices to set up the constitutional symptoms when the plague is conveyed by inoculation.

14. *Yellow Fever*.—Yellow fever has a short incubation of from two to six days, rarely exceeding eight days. Dr. Cargill, of Jamaica, gives a case fatal the day after exposure to infection.

15. *Dengue*.—Dengue is quickly developed—usually in three days. The febrile ingress is sudden, and precedes the rash only by a few hours.

16. *Cholera*.—Incubation in cholera lasts from a few hours to four days. The reports of numerous commissions give from one to five days, within five days, before the third, and not later than the fourth day. Individual cases often fall on the second or third day; the premonitory diarrhœa should be considered as part of the disease.

17. *Malaria*.—The infection of true malaria is not given off by the bodies of the sick, but is limited to certain localities. This kind of miasm, to which the term 'infection' is sometimes improperly restricted, has a marked period of incubation, which is often a lengthened one. The non-contagious malarious fevers of the West Coast of Africa show an incubation-period of ten to twelve days. In July 1810, our troops in Sicily fell ill with remittent fever thirteen or fourteen days after exposure to malaria. Men continued to fall ill for twelve days after their removal from the infected site. When Irish harvestmen came to our fen districts, they either took

ague at the end of their month's work, or not till two or three weeks after their return. Ague may appear months after residence in a marshy district.

18. *Syphilis*.—The induration of a syphilitic sore appears after ten days; six weeks later the rash or sore-throat. The localised changes of the initiatory period may occupy from three days to three weeks or even longer; in the shortest instances with immediate adenopathy the eruption follows in six weeks. John Hunter gives the interval from the local to general infection as two months; this may extend to eighty days. Other constitutional symptoms occur at much longer intervals. The contagion of secondary syphilis requires five weeks or longer to become manifest.

19. *Rabies*.—Hydrophobia presents us with the longest, and also with the most variable period of incubation; this, however, has a limit of great practical value. The shortest interval exceeds a week, so that ill effects felt in a less time than this, or following rapidly after the bite, are readily distinguished from rabies. The usual period of incubation is six weeks. Three weeks is an exceptionally short period, three months not exceptionally long. Dr. Mead gives cases occurring after eleven and fifteen months. A latency of thirteen years has been recorded. After the second month has elapsed, the fear of any ulterior consequences becomes less and less. The incubation of the dumb form of rabies inoculated by M. Pasteur's method in the rabbit is reduced to seven days. In the dog the usual interval is from three to six or eight weeks; it has occurred after three months, and in one instance recorded by Youatt after seven months. The shortest interval given by Regnault, from the bite to signs of rabies in the dog, is ten days; of his sixty-eight cases ten were from a fortnight to three weeks, and fifty-seven at longer intervals up to three months.

The incubation-periods of these widely differing diseases, while retaining distinctive differences, merge into each other. The longest intervals observed for scarlet fever correspond with the shortest observed in measles; yet many diseases are separated widely by their length of incubation, as small-pox from cholera, yellow fever and plague from typhus, and still more widely from the recurrent paludal fevers.

WILLIAM SQUIRE.

**INDIAN RINGWORM.**—See EPIPHYTIC SKIN-DISEASES.

**INDICATION** (*indico*, I point out).—That which suggests or clearly demonstrates the course to be pursued and the remedies to be adopted by the practitioner, either for the prevention or in the actual treatment of disease.

**INDIGESTION.**—Difficulty in digestion. See DIGESTION, Disorders of.

**INDURATED CHANCRE.**—A synonym for the initial manifestation of syphilis. See SYPHILIS.

**INDURATION** (*induro*, I harden).—A term applied to the process or condition of hardening of the tissues from any cause.

**INFANTILE CONVULSIONS.**—See CONVULSIONS; and INFANTS, Diseases of.

**INFANTILE PARALYSIS.**—See PARALYSIS, INFANTILE.

**INFANTILE REMITTENT FEVER.**—By some authorities a disease thus named has been regarded as a special kind of fever; but the condition is probably either typhoid fever of a mild type, or febrile disturbance accompanying disorders of the alimentary canal.

**INFANTS, Diseases of.**—There are few disorders which can be said to be peculiar to infancy and childhood. The diseases to which children are liable are, as a rule, those which attack older persons, and present the same pathological characters. But disease as it occurs in children does yet require especial study, for the symptoms by which it is accompanied often differ widely from those with which ordinary hospital practice has rendered us familiar. Children are not merely adults in miniature. They have special peculiarities of constitution, which impress their own stamp upon all acute diseases, and often raise up a number of accessory phenomena which overshadow the main symptoms, and obscure a case which but for them would be simple and clear.

**GENERAL CHARACTERS.**—The most striking peculiarity of childhood is the marked excitability of the nervous system; for the promptness and intensity with which the whole system reacts against any source of irritation is a cause of continual embarrassment to the physician. A fragment of indigestible food, for example, may produce high fever, or alarming agitation, and even throw the child into convulsions; a slight irritation of the larynx may produce severe spasm, and simulate for the time all the symptoms of true diphtheritic croup. The beginning of acute disease is almost invariably accompanied by profound general disturbance; but disturbance as profound may be excited by the simplest functional disorder, so that the severity of the symptoms is no guide at all to the severity of the lesion with which we have to deal. In all cases, therefore, it is of importance, if possible, to pick out the local symptoms—those, namely, which point to mischief of any special organ—and separate them from others which are expressive merely of the

general distress. Such local symptoms are the cough, rapid breathing, and active nares which point to acute lung-disease; the squinting and immobility of pupils which are so characteristic of cerebral affections; and the peculiar jerking movement of the legs, which, combined with hardness of the abdominal muscles, betrays the existence of colicky pain. Such local symptoms are not, however, always to be discovered, and even if present may not furnish trustworthy indications; for so great is the sympathy in the young child of distant organs with one another—linked together as they are by the impressionable nervous system—that the organ from which the more definite symptoms appear to arise may not be the organ which is actually the seat of disease. The two organs which are most frequently found to present these deceptive manifestations are the stomach and the brain. The sympathy of the stomach with an irritable condition of other parts of the body continues more or less through life: the vomiting of pregnancy and of disordered uterine function in the female, and of cerebral and renal diseases in both sexes, being matter of common observation. In the child, however, this sympathy is carried to its highest point. Vomiting is a common symptom at the beginning of every acute disease, and in many children any casual disturbance is apt to be attended by it. The brain, again, exhibits a close sympathy with irritation of the more important organs. In some cases of pneumonia, notably those in which the inflammation is seated at the apex of the lung, headache, vertigo, delirium, and stupor may be so marked that the ordinary symptoms of the disease are completely obscured, and the case is mistaken for one of meningitis. Again, the violent nocturnal delirium so often excited by the irritation of worms in the alimentary canal, must be within the experience of all.

The nervous excitability of children, and its influence upon the system generally, is well illustrated by the high temperature noticed in many children on the first evening after admission into the wards of a hospital. The elevation varies in degree in different subjects; but if the patient be not a mere infant, it is usually over 100°, although the complaint be one not ordinarily attended by pyrexia.

Perhaps, however, the most familiar instance of the impressibility of the nervous system is seen in the case of convulsions. A 'fit' in the child has a very different meaning to a similar attack in the adult. In the latter it is usually evidence of a grave centric lesion, and its occurrence occasions the greatest anxiety. In the child, on the contrary, it is a common expression of the perturbation of the nervous system, set up in response to some excentric irritation, and

often, as in the case of the onset of acute disease, is analogous to the rigor which ushers in an acute attack in older persons. Sometimes, it is true, convulsions are produced in the child, as in the adult, by severe cerebral disease; but in such cases the fits are frequently repeated, and are succeeded by rigidity, paralysis, and other signs of centric irritation. As a rule, single fits, or convulsions occurring without other signs of nerve-lesion in a healthy child, are purely reflex, and have no gravity whatever.

The impressibility of the nervous system is increased by causes which produce a sudden depression of strength, such as a bad attack of diarrhœa, or loss of blood, and in one chronic disease—rickets—the nervous irritability is very great. The effect of chronic wasting upon the child is, however, usually to produce an opposite result; and under the long-continued influence of enfeebling disease the excitability of the nervous system becomes gradually less and less manifest, until it finally disappears almost entirely. It is of importance to the practitioner to bear this fact in mind, for in a child much reduced by chronic illness, the presence of an intercurrent acute complication—such as inflammation of the lung—may be indicated by very few symptoms, the system having become almost insensible to nervous impressions.

Another peculiarity which strikes the attention of anyone accustomed only to disease as it occurs in the adult, is the vast preponderance in infantile disorders of mere disturbance of function, and the disastrous consequences which may ensue from such derangements. Infants rapidly part with their heat, and are easily chilled. They are therefore excessively sensitive to changes of temperature. A catarrh is a common ailment in the young child, and is attended by various dangers according to the part of the mucous tract which is affected by it. Gastric catarrh with violent and repeated vomiting, and intestinal catarrh with uncontrollable diarrhœa, are answerable for a large proportion of the deaths amongst young children during the warmer months. Even in cases where the catarrh affecting the digestive organs is of a less acute and violent character, the issue is often very serious. The gradual failure in nutrition, which is the result of such an impediment to the digestion of food, is a common cause of wasting in young children; and unless measures be taken early to restore the proper working of the alimentary functions, the case may end fatally. In the autumn and winter the bronchial mucous membrane is more frequently attacked. In such cases, however apparently slight may be the catarrh, a weakly infant is always exposed to the danger of pulmonary collapse; and a rapid interference with the respiratory function,

such as takes place when collapse of some extent of lung is quickly brought about, is often a cause of sudden death.

It is in consequence of this frequency of functional derangements, and their dangerous character, that *post-mortem* examinations in infants are so often unsatisfactory in not finding any appearances explanatory of the cause of death.

CLINICAL EXAMINATION.—The clinical examination of young children requires tact and patience, but unless the child be very unruly it is not difficult. The patient cannot himself describe his symptoms, but all necessary information can be gained from the parents. Mothers are, as a rule, good observers, and allowing for their natural anxiety and a slight tendency to exaggeration, their statements can usually be relied upon. We can thus learn the previous state of the child, the exact date at which his symptoms began, and the order in which they appeared. Infants should be always stripped for examination, so that the whole body may be exposed to view. Before, however, ordering the removal of the clothes, we should be careful to satisfy ourselves upon certain points which can only be properly observed while the child is in repose. Thus, in order to count the pulse and respiration, perfect quiet is indispensable, for the least movement quickens the heart's action, and alters the rapidity of the breathing. At the same time the temperature can be taken by the thermometer in the rectum. The whole body should then be examined for spots or swellings; the condition of the skin can be noted—whether dry or moist; the fontanelle examined; and we can ascertain the state of the belly, with regard to hardness or softness of the abdominal walls, and the size of the liver and spleen. If the child cry at the time, we mark the character of the voice, for hoarseness is an early sign of congenital syphilis.

In the physical examination of the chest in a child, it is important to attend to the following points: To employ percussion of the two sides at the same period of the respiratory movement, that is, during expiration or during inspiration; to strike gently with *two* fingers, for by this means a larger volume of sound is brought out, and slight dulness is more easily detected; always to use a stethoscope instead of the unassisted ear, in order to limit the area listened to; and to manage so that the child's mouth be open during auscultation, so as to hinder the transmission of sounds from the throat. In an infant the back is best examined by placing the child on the nurse's left shoulder, with his left arm round her neck. If the chin be now depressed by the nurse's hand on the child's head, the muscles of both shoulders are relaxed. The front and sides of the chest can be examined as the infant

lies on his back. We must remember that the breath-sounds, especially that of inspiration, are of a more blowing quality in the child than they are in the adult; and that there is naturally less resonance at the right base, on account of the proportionately greater size of the liver.

At the end of the examination the mouth should be looked at for signs of aphthæ or thrush; and the condition of the gums, as to heat and swelling, should be ascertained. Lastly, the throat is to be inspected—depressing the tongue with the handle of a spoon. If there be disorder of the digestive apparatus, such as sickness, constipation, or diarrhœa, it must not be forgotten to examine always and carefully the discharges. The urine should not be overlooked.

**DIAGNOSIS.**—Diagnosis in the young child is sometimes very difficult, but it is often easy enough. Being aware of the nervous excitability in young subjects, we are prepared for evidences of general disturbance, and look for more special symptoms—such as will indicate local distress, and direct our attention to a particular organ. We are also guided by a history of the attack, as gathered from the mother, and can put our suspicions to the test by a careful exploration of the whole body. In the investigation our general knowledge of the course of disease will be of service. Thus, many disorders have known pathological consequences, and are apt to be followed by special sequelæ. Measles and whooping-cough leave behind them a tendency to catarrhal pneumonia, and a liability to tuberculosis. Scarlatina often leads to acute desquamative nephritis and dropsy. Other diseases, again, encourage particular susceptibilities—as rickets, which renders the body exceptionally sensitive to changes of temperature, and provokes catarrhal derangements. In doubtful cases we must not forget to take prevailing epidemics into account, as the beginning of zymotic disease is often excessively puzzling. In all cases, especially if the patient be an infant, it is important to inquire into the hygienic and dietetic arrangements to which the child is subjected. When we are still undecided, after having exhausted all means of investigation, we must be contented to wait for further indications, and no positive opinion should be hazarded while any doubt remains.

**TREATMENT.**—Children, as a rule, respond well to treatment. This may be explained partly by the large proportion of mere functional derangements in the illnesses to which they are subject, and partly by the state of constant change through which the body is passing; growth and development are active in organs, and the tendency is to repair. The term ‘treatment,’ however, includes far more than the mere giving of physic. A complete change in all the influences acting

upon the patient—a reconstruction of the dietary, and a reformation in the hygienic arrangements, especially with regard to air, light, and clothing—will often prove of immense service, and be of far more value than actual drug-giving in furthering the recovery of the child.

In the treatment of acute illness we must remember that young children cannot bear lowering measures; but we must not therefore rush to the opposite extreme, for unless suffering from temporary exhaustion, they are far from being benefited by profuse stimulation. In the beginning of acute inflammatory diseases stimulants are injurious. Even in chronic ailments, such as rickets, where a certain amount of alcohol is often of service, wine should be given with caution, and its effects upon the digestion carefully watched; it can only be given with advantage so long as it improves the appetite, and increases the digestive power.

With regard to medicines little need be said in this place. It may be remarked that, on account of the tendency to acid dyspepsia in all children, alkalis are of especial service; and that they should be always combined with an aromatic, on account of the value of the latter in stimulating the alimentary mucous membrane, and relieving the flatulence and other painful consequences of indigestion. It is important also to remember that children are wonderfully tolerant of certain drugs, while they bear others very badly. Belladonna may be given to infants and children in comparatively large doses. They are also more tolerant of arsenic than their elders. To the action of opium, however, they are excessively susceptible, and the drug should be given—to infants especially—with extreme caution.

EUSTACE SMITH.

**INFARCT** (*infarcio*, I cram in).—This term was formerly applied to any kind of infiltration of an organ; but its use is now almost confined to the expression *hæmorrhagic infarct*. A hæmorrhagic infarct is a firm, red, usually wedge-shaped patch, which is found in certain organs as the effect of arterial embolism, or, more immediately, of the congestion and extravasation of blood to which the embolism gives rise. See **EMBOLISM**.

**INFECTIO**  
**INFECTIOUS** } (*inficio*, I put in, dye,  
**INFECTIVE** }

stain).—There is much ambiguity and want of precision in the application of these terms, and it is only intended here to attempt to define them according to their several uses, the reader being referred for fuller illustration to other appropriate articles, and especially to that on **CONTAGION**. Usually, they are coupled with diseases which are known to be capable of transmission from one animal

to another of a different class, or from one individual to another of the same species. In general language such diseases are said to be *infectious*, and to be conveyed by *infection*. These words are, however, often employed in a more definite and limited sense, as signifying the transmission of affections of this kind without the necessity of any direct contact between the individuals, or of any obvious application of the morbid agent to the body, or its immediate introduction into the system, this agent being conveyed through the atmosphere, and taken in mainly during the act of respiration. This limited meaning is employed in contradistinction to *contagion* and *contagious*—which then imply direct contact, and to *inoculation*; in this sense some affections being regarded as infectious but not contagious or inoculable, and *vice versâ*. The word *infection* is sometimes used as synonymous with the *contagium* or agent by which a communicable disease is conveyed. An important application of the term *infective* relates to the effects resulting from certain morbid products, such as tubercle, pus, septic materials, &c., which, when introduced into the system by inoculation or in other ways, produce corresponding definite changes by an infective process, manifested by tuberculosis, pyæmia, or septicæmia. When certain morbid conditions have been established within the system, other parts, more or less distant from the primary seat of mischief, often become involved by infection within the body itself—*auto-infection*—in consequence of the infective elements being conveyed by the blood-vessels or lymphatics to these remote parts, and there undergoing further multiplication and growth. Indeed the whole system may thus become tainted, including the blood and other fluids. Illustrations of this signification of infection are afforded by cancer, syphilis, tuberculosis, and suppuration, the last-mentioned not only originating secondary collections of pus, but also setting up pyæmia. See CONTAGION.

FREDERICK T. ROBERTS.

**INFILTRATION** (*in*, into; and *filtrô*, I strain through felt).—This term was formerly applied to the effusion of a fluid into the interstices of a tissue, especially connective tissue. Now, however, its meaning has been extended to imply the diffusion of any solid or fluid morbid product in the midst of tissue-elements, such as is seen in calcareous, albuminoid, fatty, and tubercular infiltration. See DEGENERATION.

**INFLAMMATION** (*inflammo*, I set on fire).—SYNON.: Fr. *Inflammation*; Ger. *Entzündung*.

DEFINITION.—Very numerous definitions have been given of inflammation. The most generally received has been that attributed

to Celsus, which gives the four marks of inflammation as *rubor, tumor, calor, dolor*; but this appears to have been really due to Erasistratus, who, according to Galen, first gave precision to the conception of simple burning, as understood by the older Greek physicians, and applied the name *φλεγμονή* (previously synonymous with *φλόγωσις*) to a swelling, which had also the characters of heat, pain, throbbing, and resistance. The definition was thus based upon the notion of *swelling*, and would hardly have taken this particular form had it been derived from superficial inflammations. Although the four 'cardinal' signs may still be recognised in what we call inflammation, the definition is best derived from a cause known to be capable of producing it, and we say that *inflammation is a series of changes in a part produced by injury, which may be mechanical, chemical, or physical; provided the damage produced does not entirely destroy the vitality of the part*. When, as generally happens, the injury does destroy the vitality of certain parts, it is only those which just escape destruction which can be said to be inflamed.

In all animals with a complete blood-circulation, injury to any vascular part sets up disturbances of the nervous and vascular mechanism, resulting in hyperæmia, which in slight injuries may be the only visible effect. In severer injuries it always accompanies the other changes, and hence is an integral part of the process. (The changes in non-vascular parts will be afterwards spoken of; those produced by injury in the lower animals need not here be considered.) Bearing this in mind, a gradation may be traced in the effects of injury. A very slight injury to the skin, for instance, by friction, heat, or a chemical irritant, produces transitory hyperæmia; a more severe or more lasting injury sets up inflammation (blister or eczema); one still more intense, necrosis or sloughing. The same gradation may be seen in the after-effects of cold, which, according to its duration or intensity, may lead to mere heat and throbbing, or to inflammation (chilblains or mild frostbite), or to actual gangrene. Even in apparently spontaneous inflammations a cause of injury may always be traced, such, for instance, as a diffusible virus in rheumatic inflammations, or the presence of micro-organisms and their products in infective inflammations, or disturbances of circulation and innervation in the secondary inflammations of circulatory and nervous diseases, or a mere lowered state of nutrition, as in various chronic cachexias. But in most such cases there is a combination of injuries, the presence of some noxious substance coinciding with nervous, vascular, or nutritional disturbance. Hence an inflamed part is always a damaged part, and the state of inflammation may be broadly said to be a state of damage.

*Inflammation and Repair.*—Since animals are so constructed that injuries short of death always call forth some attempt at repair, we find that along with damage there is always some sign of restoration. The combination of these two factors constitutes inflammation, distinguishing it from necrosis on the one hand, and simple hyperplasia on the other. Accordingly a part of the inflammatory process may be regarded as conservative, tending to compensate the disturbance produced by injury. Hence inflammation often accompanies repair, but can hardly be considered as a necessary part of the latter process. Hunter maintained that inflammation was only a hindrance to healing of wounds, and the results of anti-septic surgery confirm his views. The truth, however, appears to be that in 'clean' or aseptic wounds, however extensive, there is little damage to the tissues. Damage is caused by the secondary injuries resulting from the entrance of bacteria or irritating substances, and when this occurs there is inflammation. In this case the delayed healing which follows cannot be attributed to the original injury, nor, strictly speaking, to inflammation, but to the secondary complications which produced the inflammation, and which this process actually tends to compensate and remove.

In order to exhibit the nature and functions of the several parts of the inflammatory process, we shall consider separately: (1) the vascular processes—hyperæmia and exudation; (2) the changes in the tissues.

**1. DIRECT OBSERVATION OF THE VASCULAR PROCESSES IN INFLAMMATION.**—When the mesentery or tongue of a frog is drawn out and placed under the microscope, the contact of the air soon determines inflammation; and, by means which need not here be described, both the vessels and the tissue-elements may be observed for hours together. The earliest change seen in the vessels of a part thus exposed is dilatation, first of the arteries, then of the veins, the capillaries being little affected. The dilatation of the vessels is accompanied at first by acceleration of the blood-stream, most noticeable in the arteries; but the acceleration does not last more than half an hour or an hour, and it then gives place to retardation, which continues as long as the inflammation lasts. The 'primary acceleration' is, however, sometimes absent or too short in its duration to be noticeable. So far, the process is doubtless the same as in active hyperæmia produced by local irritation. Whether the first dilatation is due to direct paralysis of the muscular walls of the arteries, or to a reflex action passing through the spinal cord, or to an inhibitory action passing through nerve-ganglia in the arterial walls, is uncertain. There need not, however, be any action connected with the spinal cord,

since dilatation may take place when the part is disconnected from the great nerve-centres.

From this point begin the phenomena peculiar to inflammation. The dilatation of arteries may go on increasing for ten or twelve hours, till they have double their original diameter, and pulsation becomes very prominent in them. The capillaries look as if gorged with corpuscles, forming a quasi-solid mass. The blood-current in all the vessels becomes slower and slower, till it is almost stagnant. This condition has been called *stasis*; but it is better to reserve this name for an absolute stagnation, which is no necessary part of inflammation, but is produced in artificial inflammations by local causes, chiefly, probably, by drying up of the tissues. In true inflammation the amount of blood passing through the part is always greatly increased, as may be seen by the fuller current in the veins leading from it. At the same time certain peculiarities are observed in the behaviour of the red corpuscles and leucocytes in the veins. In ordinary conditions of the circulation, the central part only of the vein (as of the artery) is occupied by the corpuscles, which move on mingled together. But during retardation the corpuscles, especially the leucocytes, spread over the marginal portion of the vein usually free from them, and the leucocytes begin to drag along the walls of the vein, as if adherent, till at length they form a layer lining the wall of the vessel, while the red corpuscles are carried on by the current. In the capillaries, this marginal layer is never perfectly established, though leucocytes may be seen momentarily adhering to, or moving slowly along the walls. In the arteries, no such process is observed, except (according to Cohnheim) it may be for a moment during the diastole of the pulse. When this *marginal position* of leucocytes is established, begins the process which, observed long ago by Waller, and less clearly though earlier by William Addison, was re-observed and brought into notice by Cohnheim. In the words of the last-named observer: 'On the outer contour of the wall of a vessel, usually a vein, in which the marginal layer of leucocytes is well developed, sometimes first in a capillary, is seen a small projection which enlarges in length and breadth, and becomes a roundish colourless lump. This again enlarges, puts out new pointed projections, and gradually withdraws itself from the wall of the vessel, till it is attached to it only by a long narrow stem. Finally this attachment also is broken, and we see a colourless contractile body with one long process and several shorter, with one or several nuclei—in fact, a *leucocyte*.' The same process is going on meanwhile at other points of the veins and capillaries, till at length, either quickly or slowly, the outer surface of all the visible veins becomes covered with several

rows of leucocytes, while their interior shows the same appearance as before. That the leucocytes seen outside were formerly inside the veins, having simply passed through the walls, admits of no reasonable doubt, though it is often difficult for the eye to seize the precise moment of passage. In the arteries nothing of the kind is seen. In the capillaries the emigration of leucocytes is very evident, and accompanied by the passage of red corpuscles also through the walls, which does not take place in the veins proper. The latter process was observed by Stricker before the revival of the observation of emigration of leucocytes.

Accompanying the extravasation of the blood-corpuscles there is always a copious exudation of fluid, which goes far beyond physiological limits. The fluid also differs from the ordinary physiological transudation and more resembles blood-plasma. Aided by this and by their own spontaneous movements, the leucocytes are carried far and wide into the tissue, till the whole field, that is, the whole mesentery, is so crowded with them that nothing else can be seen. The red corpuscles, on the other hand, remain more closely in the neighbourhood of the vessels. When the exudation, carrying with it the corpuscles, and containing, as it does, coagulable material, reaches the surface, it forms the layer or false membrane of inflammatory lymph, seen in inflammations of serous surfaces. The tissue, at the same time, becomes more or less infiltrated with lymphoid corpuscles, producing the condition known as 'small-celled infiltration.'

*Cause of retardation and cell-emigration.*  
The processes just described mark inflammation off sharply from simple hyperæmia. Their production was at one time ascribed to thickening of the blood from transudation of serum; to coagulation in the vessels; to the adhesiveness of leucocytes; and to a change in the tissues external to the vessels. But there is little doubt that the essential factor is a change in the constitution of the vascular wall, though it is possible that this may be connected with a change in the tissues outside, especially with a loss of elasticity, which may be traced in all injured tissues—as may be seen, for instance, in the production of a 'wheal' by a blow on the skin. The same change, while it retards the passage of blood, makes the vascular wall permeable to the corpuscles, and favours exudation. What the change is we cannot say, but it seems probable that it is the same as the walls of vessels undergo when inadequately nourished. Two classes of experiments throw light on this point.

It has been found by Ryneck that other fluids than blood, such as milk, pass with greater difficulty than usual through the vessels of an irritated part; and that stagnation can be produced in the vessels of a frog,

which is kept alive by the circulation of salt-solution instead of blood in its vessels. Cohnheim has also shown that a state of the vascular walls similar to that which may be presumed to exist in inflammation, may be produced by shutting off the blood from the vessels for a certain time. He put a ligature round the tongue of a frog, and observed the vessels after the ligature was cut. If the blood had been excluded for twelve to twenty-four hours only, the vessels on the return of the blood passed into a condition of simple hyperæmia; but if longer, stagnation, marginal position of leucocytes, and extravasation of corpuscles were observed, and these phenomena were more marked the longer the ligature had remained, provided it was not long enough entirely to destroy the vitality of the part. From these experiments we must conclude that the cause of the stagnation and its attendant phenomena is not in the blood or the cells, but in the walls of the vessels, and that the change in these is of the nature of degeneration.

2. CHANGES OF THE TISSUE-ELEMENTS.—  
*Tissue-changes in non-vascular parts.*—The changes of the tissue-elements are most simply seen in non-vascular parts. Alterations in cartilage-cells during ulceration were observed long ago by Goodsir and Redfern, but were not then thought to belong to inflammation, non-vascular parts being not thought liable to this process. Lately the cornea has been taken as the type of non-vascular tissues. If the cornea of a frog be irritated by touching a small spot with nitrate of silver, and cut out within from twelve to twenty-four hours afterwards, many of the fixed corpuscles are found to be already altered. Their processes are become shorter and thicker; their bodies of irregular shape; and they often show amœboid movements, and become converted into many-nucleated protoplasmic masses, resembling what are in some other parts called giant-cells. Finally, in their place may be seen groups of new small or lymphoid cells. But there are always some corneal corpuscles which remain quite unchanged. These changes cannot be traced farther, as the cornea soon becomes turbid, and the whole field of observation crowded with leucocytes.

It is still a matter of dispute whether these changes are truly reproductive or only degenerative; and whether any new cells are really formed from the fixed corpuscles of the cornea, or whether the origin of these leucocytes is the same as of those which are seen surrounding the veins and capillaries of the inflamed mesentery of the frog. Various arguments have been urged, which space forbids us to discuss. The ultimate conclusion appears to be that young cells, indistinguishable from leucocytes or pus-corpuscles, may be formed by a process of growth, cell-division, and germination, affecting the fixed

corneal corpuscles, but that the number of these is inconsiderable compared with those which, even in a non-vascular tissue like the cornea, are derived from the vessels.

The process of inflammation in other non-vascular parts, such as cartilage, has been studied in the same way as in the cornea, and the general result is the same.

*Tissue-changes in vascular parts.*—These, though apparently more ambiguous than in non-vascular parts, are yet sometimes susceptible of more minute study. In the omentum of mammalia, changes occur during inflammation in which unbiassed observation can see nothing else than cell-division, growth, and germination. There is indeed reason to believe that such appearances are found normally, as evidence merely of growth, but the inflammatory changes are distinguished from the normal by their greater frequency and luxuriance. In fibrous connective tissue similar processes have long been observed, and, since attention was drawn to them by Virchow, have been regarded till lately as showing, in the clearest manner, that the fixed cells of the tissue germinate and produce new elements. This ‘proliferation of connective tissue’ is an obvious fact, but its importance is chiefly, if not entirely, in relation to tissue-formation, and not in the production of lymphoid or pus-cells, often ascribed to it. Around inflamed parts the connective tissue is often found infiltrated with lymphoid cells, the origin of which was formerly set down to proliferation. But it seems more natural to regard this ‘small-celled infiltration,’ which is a very frequent accompaniment of inflammation, as resulting from migration of leucocytes from the veins and capillaries of the inflamed part. The possibility, however, of the production of some lymphoid cells by proliferation of the fixed elements cannot be actually denied.

There is also reason to believe that the leucocytes emigrated from the vessels may further divide and ‘proliferate.’ Stricker and Klein have both observed actual cell-division take place under the microscope; so that some of the new cells may be regarded as the descendants of emigrated corpuscles. We may then compare the small or lymphoid cells of inflamed parts to the population of a colony, where most are emigrants, or the descendants of emigrants, but some few trace their descent from the aboriginal inhabitants.

*Changes in the tissue-elements: retrograde and progressive.*—More important than the infiltration of lymphoid cells are the definite changes which the fixed elements of the tissues undergo. These always show the combination of retrograde or degenerative with progressive or germinative changes, characteristic of inflammation, sometimes the one, sometimes the other, predominating, according to the severity of the injury and the nature of the tissue.

In *connective tissue* the retrograde change consists in swelling and softening of the fibres, which may end in liquefaction, especially when there is suppuration. The progressive changes consist in proliferation and formation of new cells for the purpose of repair. Special histological methods generally permit us to distinguish these elements from migratory lymphoid corpuscles. Nuclear changes, karyokinesis and karyomitosis, may often be traced in inflamed parts by proper methods, and are the most certain evidence of cell-proliferation (*see CELL*). Tissue-repair begins with the formation of so-called epithelioid cells or fibroblasts (fibroplastic cells of the older microscopists), which sometimes enlarge into giant-cells. (The theory of Ziegler, that such cells may be formed out of leucocytes, is now given up by its author.) The nuclei of these cells become the nuclei of the new tissue-elements, the fibres of which are probably formed by splitting up of the protoplasm of the cells. Outgrowths from the surrounding blood-vessels run into the new tissue and vascularise it. The whole process is thus one of continuous growth, quite independent of the migratory lymphoid cells, and of the hyperæmia and exudation, except in so far that the latter help by affording a more abundant supply of nourishment. The relation of the lymphoid cells to repair is thus very obscure, and it is possible that they may be merely a hindrance; though the notion has been started that they serve as pabulum to the growing tissue-cells. But they perform one important function, that of removing injurious foreign particles from the seat of inflammation. Molecules of fat, pigment-granules, and other *débris* of tissue, may be clearly seen to be taken up by them; and there is reason to believe that they also carry away bacteria. Whether their action on these organisms is precisely that which Metschnikoff calls *phagocytosis* is a disputed question, the discussion of which hardly belongs here. *See IMMUNITY*; and *PHAGOCYTOSIS*.

*Muscular tissue* becomes granular and loses its striation under the influence of inflammation, and sometimes undergoes another peculiar form of degeneration. But at the same time the muscle-nuclei (unless the tissue be absolutely killed) show signs of growth and proliferation, by which in the end new fibres are produced and the tissue regenerated.

*Nerve-fibres* undergo rapid disintegration when inflamed; but the nuclei preserve their vitality, and are capable of producing new nerve-tubes, as is seen after section of nerves.

*Ganglionic nervous tissue* never shows any signs of regeneration after being inflamed, and is probably killed by comparatively slight and transient inflammations, though this tissue is very rarely thus affected.

*Epithelium* on mucous surfaces shows very characteristic changes. A large number

of elements die and are shed off, so that the desquamation of epithelium is far more rapid than under normal conditions. Many cells show excessive mucous transformation and—in chronic inflammations—much fatty degeneration. There is also a copious formation of new epithelial cells, produced, as in normal conditions, apparently from the basement membrane.

*Glandular epithelium*, when inflamed, shows the characteristic change called granular degeneration or cloudy swelling (*see* DEGENERATION), which has been also called parenchymatous inflammation, and is evidence of the simplest kind of injury to the cells. Severe injuries cause fatty degeneration and breaking down, as seen in various kinds of poisoning. There is probably always a regeneration of gland-cells, but this is often traced with great difficulty.

The above are the tissue-changes usually met with in ordinary inflammations. When the inflammation takes the form of *suppuration*, destruction of tissue is far more complete, and repair is prevented, at least temporarily.

We must now consider the products and results of inflammation, without further distinguishing between the share of the vessels and of the tissues.

**PRODUCTS AND RESULTS.**—All inflammatory products result either from exudation (with or without participation of the tissues) or from new-growth. The exudative products are serum, mucus, and fibrin, the latter of which, in combination with leucocytes, forms the so-called 'inflammatory lymph.' New-growth takes place from the vessels in the form of vascular connective tissue, of which a special form are granulations. With regard to exudations, no clear line can be drawn, at any stage of the process, between serous and fibrinous exudation or inflammatory lymph. But the production of pus seems to require some special irritant or morbid factor.

*Exudations.*—Serous and mucous exudations can only be regarded as products of inflammation when excessive. The fluids formed in inflammations of serous cavities differ from those produced in passive exudations (or dropsies), in containing more fibrin and more albumen. But inflammatory exudations vary in this respect, and are sometimes scarcely to be distinguished from simple serous effusions. The fluids poured out on serous surfaces in acute inflammation always coagulate, and even in chronic cases can often be shown to be capable of coagulation. On mucous surfaces, on the contrary, the exudation does not as a rule coagulate. That this is owing in some way to the action of the epithelium seems most probable, whether it is that filtration through epithelium alters the composition of the fluid, or whether the living epithelium prevents coagulation in the

same way as the endothelium of the vessels prevents coagulation of the circulating blood. When, however, the epithelium is removed, a fibrinous layer may be produced on the exposed sub-mucous surface; and the same result seems to follow the application of very powerful irritants, as in croupous inflammations. Mucous exudations contain mucin, as well as serum-albumen, in variable proportions.

Fibrinous exudations, in coagulating, entangle whatever leucocytes may be either extruded with the exudation or present in the tissues. The properties of the coagulated mass differ according to the proportion of corpuscles. These differences do not necessarily show any corresponding differences in the composition of the blood, but depend upon the facility with which the corpuscles leave the vessels, and probably on the state of nutrition of the latter. The product called *inflammatory lymph* consists of coagulated fibrin entangling leucocytes, the two constituents being in varying proportions. The fibrin does not differ from that of blood-clot; and may therefore be formed in the same way by a reaction between the exuded constituents of blood. But it is not a constant product of inflammation, and hence has been thought to owe its production to local causes—that is, to reaction between the tissues and the exudation. Thus fibrin is formed on serous surfaces where one of the fibrin constituents is normally found, and in fibrous tissue where there are similar chemical constituents; but, as stated above, not generally on inflamed mucous surfaces, or in epithelial structures. But since it is possible that in these cases the exudation becomes altered by filtration through the tissues, there is no reason to doubt that fibrin, or both its chemical constituents, may be exuded from the vessels. The inflammatory fibrin itself is never *organised*, but often becomes *replaced* by a vascular connective tissue which grows into it, and causes its absorption. This is the process formerly spoken of as organisation of lymph.

**Pus.**—Pus is inflammatory exudation in which the corpuscles greatly predominate, and the intermediate substance is liquid. It is thus difficult to draw a line between pus and softer forms of inflammatory lymph, but the former does not contain fibrin, nor does it coagulate spontaneously. The following analysis of pus may be taken as representing an average specimen:—

Water . . . . .	887.6
Pus-cells and mucus . . . . .	46.5
Albumen . . . . .	43.8
Fat and cholesterin . . . . .	10.9
Sodium-chloride . . . . .	5.9
Other alkaline salts . . . . .	3.2
Earthy phosphates and iron . . . . .	2.1
	<hr/> 1000.0

It is noticeable that pus contains a much larger proportion of fat than any other inflammatory product, or than blood. Its specific gravity is 1030 to 1033. The appearance of pus is well known: it is a creamy fluid, which, when normal or 'laudable' and fresh, has a very faint—not offensive—odour, and no sign of putrefaction. Under certain circumstances, it has a strong ammoniacal or putrefactive odour, and is described as 'unhealthy,' or sanious. When allowed to stand, all pus separates into a liquid portion or 'serum,' and a sediment consisting chiefly of the corpuscles. The serum resembles blood-serum, containing paraglobulin or fibrino-plastic substance, potassium-albuminate (or casein), ordinary serum-albumen, and myosin. Other constituents of pus are protogon, chondrin, gelatin, leucin, tyrosin, and xanthin.

*Pus-corpuscles.*—The corpuscles of fresh, newly formed pus, as seen, for instance, on an inflamed mucous surface, are not distinguishable from leucocytes of the blood, showing active amoeboid movements and continual change of form. Most corpuscles from large collections of pus, such as abscesses, are already dead, being nearly spherical in form, with the appearance of a cell-wall, and showing when acted upon by acetic acid three or more small nuclei.

*Causes of suppuration.*—While the production of individual pus-corpuscles is to be explained in the same way as that of inflammatory cells in general, there are good reasons for thinking that the formation of pus requires, in addition, some more special cause.

The most important cause is undoubtedly the presence of bacteria, especially certain species of micrococci, which are almost always present in collections of pus, large or small; sometimes contained in the pus-corpuscles, sometimes free in the liquid. Ogston found micrococci in every one of 74 abscesses not previously opened; and they can be found, broadly speaking, in all boils, pustules, and the like. The commonest species are *Staphylococcus pyogenes albus* and *S. aureus*, with sometimes *Streptococcus pyogenes*. The latter is the characteristic form in internal suppurations, especially in pyæmia. There can be no doubt that these micrococci produce suppuration, since, when cultivated, they have often been made to produce furuncles and abscesses when rubbed firmly or inoculated into the human skin, even more constantly and certainly than in the case of experimental animals. Since these micrococci are often found (by cultivation) to be present in certain parts of the healthy human skin, as well as widely spread in human dwellings, there must be some favouring circumstances or contributory causes which enable them thus to injure the tissues. Such favouring circumstances are

(1) some previous lesion of the tissues; (2) special conditions, especially bad nutrition, the tenderness of tissue in children, and perhaps idiosyncrasy; (3) certain states of the blood, especially the presence of special substances, such as glucose, and probably ptomaines or enzymes, absorbed from the intestines. Animal parasites, such as hydatids, appear to produce suppuration by their direct action, without the assistance of bacteria.

In the absence of micrococci, suppuration may be produced by other agents; which may also act as contributory causes with the bacteria. Such are turpentine, croton oil, carbolic acid, ammonia, strong solutions of perchloride of mercury, &c.; several of which are powerful bactericides. In addition, the same result has been produced experimentally by sterilised solutions of certain bacterial products, as albumoses and ptomaines, *e.g.* cadaverine; and certain enzymes, *e.g.* the vegetable ferment of jequirity; and doubtless others will be found to have the same effect. It thus appears that chemical substances having powerful solvent or necrotic action on tissues produce suppuration, and do so apparently by softening the walls of the blood-vessels and favouring emigration of leucocytes—which will be mostly dead. The action of micrococci is to be explained in the same way, these organisms producing enzymes which peptonise and thus soften or dissolve the walls of the blood-vessels. The well-known destructive or solvent action of pus is also thus accounted for. Micrococci are doubtless the cause of almost all suppurations, since the other agents mentioned will not be present unless experimentally introduced. The bearing of these conclusions upon antiseptic surgery, which has supplied much of the evidence on which they are based, is obvious. We gain also by this knowledge an adequate explanation of the infectivity of pus; as seen in contagious impetigo of the skin, conjunctivitis, urethritis, &c.

*Granulation-Tissue.*—When a part has been destroyed by inflammation, the lost tissue is replaced by the preliminary formation of a peculiar structure, consisting of a highly vascular connective-tissue framework, containing an excessive number of leucocytes, and also epithelioid or formative cells, with occasional giant-cells. The vessels are of delicate structure, and easily lacerated. This tissue also contains nerves. It has been compared to the cellular structure which composes the embryo, or to *embryonic* tissue, but the great predominance of vessels constitutes an important difference. Such tissue always originates as an outgrowth of the vessels of the inflamed part; it grows into regular connective tissue, and is thus the most important means of replacing the tissue destroyed. When produced on a surface, and growing out in the form of tufts, it

receives the name of *granulations*; and, as is well known, ulcers and cavities become filled up by it. There is no doubt that granulation-tissue may form fibrous tissue, but not epithelial structures or any specialised tissue. The formation of granulations is not necessary to the repair of wounds; and their persistence is evidently a sign of delayed repair; hence some have regarded their formation as antagonistic to healing. Hamilton has ingeniously shown that granulations are, or may be, produced by outward dilatation of capillaries not restrained by the integument.

**VARIETIES.**—The most striking differences between different kinds of inflammation are those depending upon the differences of tissues, and of the situations in which it occurs.

1. *Catarrhal*.—On mucous membranes the exudation is mucous, does not coagulate, and contains only detached epithelial cells, with scattered leucocytes; the process tends to become chronic; but the effect on the body as a whole is less marked than in other forms. This is *catarrhal* inflammation, a term which is with less propriety transferred to inflammations of the skin, the lung, and some glandular organs.

If catarrhal inflammation is very severe it becomes purulent, and the exudation consists chiefly of pus, little or no mucus being produced. This purulent catarrh is especially characteristic of specific inflammations of the mucous surfaces, such as virulent conjunctivitis or gonorrhœa.

2. *Croupous or fibrinous*.—*Croupous* inflammation is that form in which a coagulable exudation is formed upon a mucous surface. In diphtheritic inflammation there is, besides a membranous exudation, some necrosis of the mucous membrane. In these forms the tendency is to acute, not chronic, disease; and the general symptoms are strongly marked. The name 'croup' has also been transferred to certain inflammations of the lung ('croupous pneumonia') and of the kidney, but (in the latter case especially) with doubtful propriety.

The *fibrinous* form may be regarded as the normal or usual form of inflammation of serous membranes and fibrous connective tissue.

On serous surfaces the lowest degree of inflammation is seen in a serous exudation, containing little or no plasma, hardly to be distinguished from simple dropsy; but there is no clear line between this and a coagulable exudation, or fibrinous inflammation. This too, if still more severe, may become purulent; and, as we see in the pericardium or pleura, a purulent may succeed to a fibrinous inflammation. Finally, vascular connective tissue, forming adhesions, is generally produced. In areolar connective tissue, and in the interstitial tissue of various organs, the same stages may be distinguished, known as *inflammatory œdema*, which occurs near a

focus of acute inflammation; *inflammatory hardening*, such as precedes the formation of an abscess; and, finally, either abscess itself or purulent infiltration—the two forms of suppuration in connective tissue.

3. *Parenchymatous*.—It is not so easy to define the kinds of inflammation as affecting the special elements or parenchyma of organs. The lung appears to be an exception to the general rule that epithelial surfaces show the catarrhal form of inflammation, since lobar pneumonia is a type of the fibrinous form; but it should be remembered that the anatomical structure of the air-vesicles more resembles a serous than a mucous surface, having only a single layer of epithelium; and that mechanical injury to the lung always produces a catarrhal form of inflammation.

The name 'parenchymatous inflammation' has been given to those changes occurring in the special tissues of organs independent of their connective-tissue framework. The only factor common to all such appears to be a granular degeneration of the protoplasm of their cells, identical with what is elsewhere called albuminous degeneration, though it may end in fatty or other degeneration. In contradistinction to this, inflammation of the connective-tissue framework is termed *interstitial inflammation*.

4. *Phlegmonous*.—Phlegmonous inflammation is the same as *acute interstitial inflammation*, ending in the formation of abscess.

5. *Indurative*.—Indurative inflammation is that in which new connective tissue is produced in the interior of organs. This is *chronic interstitial inflammation*.

6. *Scrofulous*.—Scrofulous inflammation is that type which occurs in cachectic persons, whose tissues are easily injured and heal slowly. Such persons are subject to chronic inflammations, which, further, involve destruction of tissue, and in which the inflammatory products readily undergo degeneration; unless these characters of destruction and degeneration are present, the name 'scrofulous' has no precise meaning. There can be little doubt that all scrofulous inflammations are really tubercular. *Strumous* is a word better forgotten, being synonymous with scrofulous, and liable to be misunderstood.

7. *Infective*.—Infective inflammations are those produced by the passage into the blood of infective matter, derived from some previously existing inflammation. Pyæmia and inflammations associated with the puerperal state are instances.

8. *Chronic*.—Most inflammations have at first a typical course, reaching their acme and then declining. If the decline is not followed by resolution they become chronic. Others, again, show from the first the character they all along maintain. Chronic inflammations are usually distinguished by the

persistence of that condition of the vessels which permits exudation and cell-emigration, with less hyperæmia and general fever than in the acute form. On mucous surfaces the chronic form differs little from the acute, except in these two respects. In serous membranes chronic inflammation produces fibrous adhesions, with little or no liquid exudation. In the interstitial tissue of solid organs a large amount of new connective tissue is produced by chronic inflammation, which first compresses the special elements, causing them to waste, and then contracts in bulk, so that the organ becomes atrophied, and usually harder and more fibrous. This is the process called 'fibroid degeneration,' as in cirrhosis of the liver. 'Chronic parenchymatous inflammations' are simple degenerations, as seen in the kidney.

**TERMINATIONS.**—The most favourable termination of inflammation is what is called *resolution*, in which the vascular phenomena and tissue-changes decline together, and pass away without leaving any tangible material result. Even in the apparently most perfect cases of resolution, there is little doubt that products of exudation remain when the vascular changes have subsided, and are slowly removed by the lymphatics. Other so-called terminations are *necrosis*, or total death of the part; and partial destruction by supuration or *ulceration*. But if any loss of substance occurs, the inflammatory process cannot be regarded as at an end till the loss is wholly or partially restored by newly formed connective tissue. Where there is no destruction of tissue, but only masses of liquid or solid exudation, the inflammation is not, strictly speaking, resolved till these are removed. Very frequently an acute passes into a chronic inflammation.

**CONSEQUENCES.**—1. *Local consequences.*—If an inflamed part does not simply return to its original state, *atrophy* is the most common consequence. *Hypertrophy* can hardly be said to occur in the part actually inflamed, though it may in adjoining parts, as we see in enlargement of bone from periostitis. False hypertrophy from new formation of connective tissue is common; but, as such tissue contracts, the final result is atrophy. The hardness of this tissue causes *induration* to be put down among the consequences of inflammation.

2. *General consequences.*—The effect of inflammation on the whole body is to produce the condition of *fever*, which is discussed in another part of this work. It will suffice here to say that fever involves raising of the body-temperature, weakening and acceleration of the heart, and disturbance of the nervous system, as well as of all the nutritive processes. It was formerly supposed that local inflammation produced fever simply through the increased production of heat by more rapid tissue-change in the inflamed

part; this process raising the temperature of the blood passing through the part, and thus of the whole body by means of the blood. But this simple explanation is not adequate. It has even been much disputed whether the heat of inflamed parts ever surpasses that of the blood, though the temperature of inflamed external parts is higher than the normal in that situation, and than that of a corresponding part, not inflamed, on the other side of the body. But this is also the case in hyperæmia, in which condition the external temperature never surpasses that of internal organs. Experiments on inflammation have led to very conflicting results: those of Simon and Weber being directly opposite to those of Jacobson and others. But even if we assume an actual production of heat in inflamed parts, this will not be enough, as is shown by calculation, to account for the rise of temperature in the whole body. It has also been supposed that some fever-producing or *pyrogenic* substance passes into the blood from every inflamed part, and causes increased tissue-change, with consequent increased production of heat through the whole body; or else exerts a special action on the heat-centres. Several substances having a pyrogenic property have been isolated. They are either animal ferments (enzymes) or albumoses, produced by the action of bacteria on proteid substances. Finally, it is held that the nervous system has a large, or even the chief, share in the production of fever; the inflamed part acting through the nerves on the cerebral heat-centres controlling the temperature of the body. But none of these theories is definitely established, and the manner, therefore, in which local inflammation produces fever is not yet perfectly understood. The degree in which local inflammations cause fever varies, and does not appear to depend wholly on the mass or the intensity of the inflammation, though both these conditions are partly concerned. Acute inflammations produce more fever than chronic; those of connective-tissue more than those of mucous surfaces; and, most of all, those which end in supuration. In infective inflammations the fever is generally high, but not to be attributed to the local inflammation, being a concurrent effect of the same cause—the infective poison. Besides general fever, the special condition called *pyæmia*, in which other local inflammations result, may be a consequence of primary inflammation; but only when certain special causes are at work. See FEVER; and PYÆMIA.

**ÆTIOLOGY.**—Most inflammations are caused by some injury—either mechanical, as by actual violence; or physical, as changes of temperature; or chemical, by powerfully acting substances, as acids, alkalis, and many more. A most important secondary factor is the condition of the body, whether under- or over-nourished, or in some other

way wrong; and this may probably be still more closely defined as the condition of the blood-vessels. Many parts of the body, as the skin and the stomach, are constantly exposed to injury, but do not become inflamed unless from some internal cause; and, therefore, a change of nutrition may be the apparent or immediate cause. It is also clear that certain inflammations, as herpes zoster, are determined by disturbance of the nerves; and it is very probable that similar nervous disturbances cause other local inflammations. Besides these, there are certain specific causes, namely, infective or specific poisons, which, when introduced into the blood, produce local inflammations. Many local inflammations, external and internal, appear to arise spontaneously, neither irritation nor fault of nutrition being easily traced. But in these, as in others, it is probable that some infective cause is at work, or else that there is a gradual accumulation in the blood of some irritant substance, the effects of which are only seen when the nutrition of the part is lowered. Such, for instance, are the secondary inflammations which occur in chronic diseases, as morbus Brightii. It is also probable, and in some cases proved, that other inflammations which were at one time thought spontaneous, are really secondary, depending upon some previous local inflammation, even without what is called actual pyæmia. Thus the number of such apparently spontaneous inflammations is gradually lessening.

**TREATMENT.**—We shall divide this subject into the treatment of *directly accessible* (chiefly external) inflammations; and that of *indirectly accessible* (internal) inflammations. Treatment will also differ according as the inflammation is acute or chronic; according to the stage, if acute; and also according to the constitution of the patient.

(a) *Directly accessible.*—Directly accessible inflammation in an early stage may be treated by local blood-letting, and by astringents. The benefit of local blood-letting in an early stage is undoubted, and is probably due to its relieving the condition of stagnation, and permitting freer circulation of blood in the part. Of astringents the type is *cold*. When the vascular disturbance, that is, hyperæmia, is great, and the general fever high, cold, produced either by ice or evaporation, is generally the best treatment. In the case of mechanical injuries, for instance, it may be regarded as an ascertained fact that if any injured part be kept cold during the period of reaction, the inflammation is less severe. When the condition of inflammatory exudation is set up, the effect of cold is less marked; and if a temporary benefit is produced, the condition after the application of cold is stopped may be as bad as ever, or perhaps worse. The defect of cold as an antiphlogistic seems to be that, though it reduces

hyperæmia, and, if sufficiently intense, actually checks inflammation, it does not remove the injured condition of the vessels and tissues on which the profounder inflammatory changes depend. Cold is also a powerful nervous sedative, and reduces the nervous irritability of inflamed parts. It is remarkable that an effect like that of cold is produced by solutions of certain *metallic salts*, especially those of lead, zinc, silver, and bismuth. These salts furnish the most certain and direct means of treating an inflammation in parts actually accessible to their action. Hence they are used in superficial inflammations of the mucous membranes, such as the digestive mucous membrane and the conjunctiva, and of the skin. Their use is limited by the difficulty of bringing them in actual contact with deeper inflamed parts. But Mr. Hutchinson has shown that even in severe injuries the antiphlogistic effect of lead is quite equal to that of cold, if it can be made to pass deeply enough into the injured parts. These mineral (and also vegetable) astringents act more potently on the exudative processes of inflammation than on the vascular disturbance. Hence their activity is most valuable when that of cold ends; and they have a striking effect on chronic inflammations which are unaffected by cold. Even *pressure* may be useful as an astringent, as we see in strapping a testicle or an inflamed joint. In applying all astringents, it is of the first importance to be sure that their effect is actually exerted on the inflamed part. In applying ice to the chest for pneumonia or pericarditis, for instance, this result is very doubtful.

In the treatment of even acute inflammations, the precisely opposite application, that of *heat*, is sometimes valuable. Hot and cold have respectively always had their partisans, and have been, the writer thinks, needlessly opposed. There are instances (such as the cure of a whitlow by plunging the finger into very hot water) where the sudden application of heat seems to cut an inflammation short. Probably this is only when the cause is some infective micro-organism. Short of this, heat doubtless increases the activity of most inflammatory processes. But heat combined with *moisture* is the type of an *emollient*, by which the substance of inflamed tissues is relaxed, the blood-vessels dilated, the sense of tension and nervous irritation removed; and though exudative processes are probably encouraged, the *mechanical* condition of inflammatory stagnation is relieved, and resolution is thus hastened. When pus is forming, there is little doubt that heat and moisture (in the form of poultices and fomentations) hasten the process, and increase the amount of pus formed; but since the increased production of pus is of no consequence, and it is more important to hasten the process, this may be the right treatment for suppuration. When

pus is once formed, the same treatment is useful in guiding it in the direction in which it is least hurtful. Finally, it may be beneficial to apply heat and moisture superficially, to relieve deep-seated organs, by stimulating the vascular and lymphatic circulation through the skin. Thus, in applying poultices for pneumonia we do not make the lung or even the pleura hotter, but relieve the overloaded blood-vessels and lymphatics.

Further, in treating all superficial inflammations we must guard against anything which increases the injury, such as movement, further irritation by the air, or by anything contained in the air. Thus, in some skin-diseases, as herpes zoster, secondary inflammation is prevented by an artificial covering of collodion. Antiseptic surgery shows that not the air, but bacteria conveyed by the air or otherwise, are to be feared, and if these are excluded much inflammation is prevented. The benefits of mechanical rest in treating injuries of the limbs need not be more than mentioned; nor that of physiological rest, wherever it can be obtained, in threatened or existing inflammation of any active organ whatever—for example, the brain, the stomach, or the kidney.

In many acute inflammations, however, where we know the course of the inflammation, and that we cannot absolutely stop the process, our best method may be merely to apply palliatives, that is, to adopt an *expectant method*.

*Diet* is also of great importance. There is little doubt that the intensity of all inflammations is lessened by greatly diminishing the food taken. In some acute inflammations, especially fevers, we may pursue a different system, with the view of saving the patient's strength; but it is possible that in the reaction against the starving process, 'the feeding of fevers' may be in the present day sometimes carried too far.

In the treatment of *chronic inflammation* in accessible parts, the first aim will be to check the exudation and cell-migration, that is, to bring the vascular wall into a healthy state. Here we find that metallic and vegetable astringents are most useful. But it may, with the same object, be well to draw more blood into the part, in order that the vascular wall may be better nourished. This is effected by *stimulants*, which are of well-known efficacy in chronic inflammation. Some of these agents are the same as astringents, but in a more concentrated form—nitrate of silver, sulphate of copper, &c.; others, like iodine and cantharides, are irritants. The local action of certain substances which may be called *aromatics* in healing some kinds of chronic inflammation is most remarkable. Thus, tar, either from wood or from coal, and similar bodies are used in chronic inflammations of the skin; copaiba, cubeb, sandal-wood, &c., when excreted by the

urine, act beneficially in chronic cystitis and urethritis. Benzoin, the tars, copaiba, tolu, and many other aromatic and resinous compounds, when excreted by the lungs, act in the same way on chronic bronchitis. The *rationale* of the action of these drugs is difficult to understand, but they are all oxidisable substances which undergo slow oxidation in the air, often accompanied by an ozonising action. Again, it may happen that it is best to sweep the old tissue away, and allow new vessels to be formed, which will probably have healthier walls. This is effected by destructive *caustics*, as nitrate of silver, potassa fusa, chloride of zinc, or even the actual cautery.

In chronic inflammations, internal treatment is often of great service, as we shall presently show.

(b) *Indirectly accessible*.—The treatment of indirectly accessible inflammations, or, what amounts to the same thing, the treatment by internal (general) methods even of directly accessible inflammations, is much less satisfactory than the local.

The first indication is to give the part actual rest, mechanical or physiological. The next is to consider if there is any way of reducing the intensity either of the local reaction, or of the fever. Of such means the chief are general blood-letting; the general application of cold; and certain particular drugs, such as mercurials, antimonials, purgatives, digitalis, aconite, quinine, and a number more.

Of *general blood-letting* we cannot say much here. There can be little doubt that it often lowers the energy of inflammatory processes in an early stage. If it has gone out of use, it is probably because the course of many diseases is now better known, and we do not expect to cut them short; because the list of infective diseases is enlarged; and because the benefits are believed to be outweighed by the supposed drawbacks in diminishing the strength of the patient. Of *cold baths* we need only here say that they are far more potent in checking the condition of fever than in stopping local inflammation. Of *drugs*, digitalis is supposed to act by depressing the heart's action; but in theory this is doubtful, and in practice we see little effect on organic inflammations. Aconite also has little local effect, but does modify the febrile state. Quinine is thought, on theoretical and experimental grounds, to check the emigration of leucocytes, and to kill the pathogenic bacteria. The first belief is experimentally true, if the drug is in a certain degree of concentration; but we have no safe means of introducing it, in this degree of concentration, into the blood. The second is extremely doubtful. Alcohol even has been recommended as an antiphlogistic, on the ground that it lowers the temperature in health. But even this is not constant, and there is no good reason for thinking that it

has this effect in fevers, still less that it checks the inflammatory process. If alcohol be given, it must be on other grounds. Salicylic acid, carbolic acid, and thymol are lauded as destroying micro-organisms; but, as with quinine, we cannot get them into the blood in sufficient concentration, and, if we could, it is probable that other more serious disturbances would be produced.

The conclusion must be that there is no one drug which is capable of controlling local processes of inflammation, though the resulting condition of fever may be modified.

The use of mercurials and antimonials seems to have been affected by the same considerations as blood-letting. The action formerly attributed to them is now doubted, probably on very insufficient grounds, and it is further thought they do harm in other ways. But no adequate explanation has been given of the difference in this respect between the practice of this and the last generation. Purgatives remain as a harmless, unquestionable, but not very potent antiphlogistic method.

In the general treatment of *chronic* inflammations we have more satisfactory principles. Chronic inflammation is, in other words, *imperfect repair*. It is in cachectic persons, or persons with an inherited proclivity (perhaps not yet manifest) to cachectic diseases, that inflammations most tend to become chronic. Hence, the first rule is to improve the nutrition. Many patients with chronic inflammations get well at once when placed in good quarters, and on good food, provided that the digestive functions are first attended to. Next in importance come nutrient tonics, of which cod-liver oil is the chief. There are few chronic inflammations in which it does not do good. Iron is very often valuable; and, if it fails or is contra-indicated, arsenic may be employed. In chronic inflammations of fibrous tissues, iodide of potassium has a real value, not easily explained. In treating other inflammations, either acute or chronic, there are many specific remedies, but these are remedies for the disease, not for its inflammatory features only. For this reason, we do not here speak of mercury, colchicum, salicylate of sodium, and other agents.

Finally, a most important means of treating indirectly accessible inflammations must be mentioned, namely, that by *counter-irritants*, or setting up a rival inflammation. In order to relieve an inflammation, for instance, of the knee-joint, we produce a superficial inflammation of the skin. This is most used in chronic, but applies to some acute inflammations also. Various explanations have been given of the undoubted efficacy of this treatment. Some believe the action is transmitted through the nerves; others that blood is drawn away; others that the lymphatics are stimulated. The writer's belief is that in the most marked cases of benefit from counter-irritation there is a continuity

of the tissue between the inflamed organ and the part where the counter-irritant is applied, and that the action may sometimes consist in drawing away blood; but more generally the benefit results from setting up currents of plasma through the lymphatics and the connective-tissue spaces. It should be noted that in some inflammations where œdema is a marked feature (*e.g.* epididymitis), simple puncture has an unquestionable efficacy which may perhaps be explained in the same way. The substances used for counter-irritation are either vesicants or rubefacients, such as cantharides, ammonia, mustard, or iodine. Dry heat at different temperatures may produce the effect of either of these classes. When redness is produced on the skin, it does not follow that hyperæmia alone results. In fact, the desquamation often shows that a low form of inflammation has been established.

The writer has here endeavoured to give only the principles of the treatment of inflammation. For the treatment of inflammations of special parts, the articles on these subjects must be consulted, as well as the articles on BLOOD, Abstraction of; COUNTER-IRRITANTS; and HEAT, Therapeutics of.

J. F. PAYNE.

**INFLATION** (*in*flo, I blow into).—A term applied, therapeutically, to the method of blowing air or gas into any hollow space. It is employed particularly in connexion with the lungs in the process of artificial respiration (*see* ARTIFICIAL RESPIRATION). It is also used for the purpose of dilating the bowel in cases of obstruction (*see* INTESTINAL OBSTRUCTION). The term 'inflation' was formerly used, pathologically, as a synonym for emphysema of the lungs, but is now more strictly limited to the condition in which the alveoli are temporarily distended with air, from any obstruction in the air-passages. *See* LUNGS, Emphysema of.

**INFLUENZA**.<sup>1</sup>—SYNON.: Epidemic Catarrh; Fr. *Grippe*; Ger. *Influenza*; *Epidemischer Schnupfenfieber*.

The term 'influenza' is said to have been first introduced in 1741, when the disease was prevailing in the North of Italy, and it has been generally adopted in this country; though the more scientific term—'epidemic catarrhal fever'—is often used in systematic works.

**DEFINITION**.—This disease is not to be regarded as simply an unusually prevalent common catarrh, but must be considered as a specific affection, which appears occasionally over wide districts, and at or about the same time; is characterised by marked febrile symptoms; is often attended by serious com-

<sup>1</sup> This article appears in its original form as written by the late Dr. Peacock. It is followed by a supplement which brings the subject down to the present time.—EDITOR.

plications; and causes great and prolonged prostration of strength.

**OCCURRENCE.**—Various epidemics of influenza are on record from the middle ages down to more recent times, and those which have occurred in the last and present centuries have been fully described. The disease is reported to have prevailed in 1729, 1732-33, 1737, 1742, 1758, 1762, 1767, 1775, and 1782, and in 1803, 1833, 1837, and 1847. The following account is chiefly founded upon the disease as it prevailed in 1847; but the description given of its peculiarities corresponds with the accounts of previous epidemics.

**FORMS.**—Influenza may be described as assuming three distinct forms:—

1. That in which it is *simple*, or unattended with any serious complication.

2. When it is *complicated* with serious pulmonary affections, especially bronchitis and pneumonia.

3. When the *disorder of the digestive organs*, which generally exists to some degree in the disease, becomes a more prominent feature; while there are at the same time marked rheumatic symptoms, and the complaint shows a tendency to assume a remittent form.

The description would also be imperfect without reference being made to the *modifying influence* which the epidemic exerts *over other diseases* prevailing at the same time, and especially over the specific fevers.

**1. Simple Catarrhal Fever.**—In this form of the disease the attack is most commonly sudden. The patient experiences a sense of cold down the back and between the shoulders, lapsing into general chilliness or complete rigors, and succeeded by flushes of heat and dryness of the skin, pain in the head, chest, and extremities, and prostration of strength. Generally these symptoms follow some exposure to cold and damp, but occasionally they appear without being traceable to any immediately exciting cause, and more rarely the attack comes on gradually, with a general feeling of indisposition of two or three days' duration.

At first there is dryness of the nostrils and soreness of the throat, with a sense of tightness or constriction of the chest, and a dry, hard cough. As the disease advances, copious defluxion from the nostrils takes place; the throat is often seriously affected; and the cough is more frequent. The expectoration is at first scanty, and consists of a pale glairy fluid; but at a later period there is more copious discharge of opaque mucus. At the same time some degree of difficulty of breathing and soreness at the chest are experienced. The respiration is in most instances accelerated; and, on auscultation, the inspiratory sounds are dry and harsh, especially in the posterior and inferior parts of the chest; and sibilant and sonorous rhonchi may be audible on forced inspiration. In some cases no

marked sounds are heard, but the vesicular murmur is very indistinct. A sense of chilliness, alternating with flushes of heat, is a general symptom throughout the progress of the attack; and there is distressing headache, particularly in the forehead, across one or both eyebrows, as well as pain in the balls of the eyes. These symptoms often undergo considerable remission during the day. There is also commonly much mental depression and nocturnal restlessness. The tongue is usually moist, and covered with a white creamy fur; but occasionally it is morbidly red at the tip and edges, and thickly coated with a whity-brown fur towards the centre and root; more rarely, and especially in the morning, it is dry. In the greater number of cases, entire loss of appetite, with some nausea, and a confined state of the bowels, are present from the commencement; but occasionally diarrhœa is observed at an early period, and not infrequently it comes on during the progress of the disease. Sense of weight, tenderness, and pain in the right hypochondrium are frequently experienced; and there is often some icteroid tinging of the conjunctivæ, or of the general complexion. The urine is scanty and high-coloured at first, but subsequently becomes more free, and deposits some sediment.

Prostration of strength is throughout one of the most marked and distressing features of the complaint, and there is a general feeling of soreness, with dull aching pains in the chest, back, and limbs. The pulse is but little varied in frequency, generally ranging from 80 to 90, and rarely exceeding 100. Though occasionally full, it is uniformly very compressible, and, after the first day or two, feeble. The skin is seldom hot or very dry, or if so it is only at the commencement of the attack, and it soon becomes cool and moist; the complaint usually subsides with free perspiration.

The ordinary duration is from three to five days in the milder cases, and from seven to ten in the more severe. The disease, however, on its subsidence usually leaves the patient for some time much prostrated, and suffering from loss of appetite, inaptitude for bodily or mental exertion, and a troublesome cough. There is a great tendency to relapse.

Not infrequently towards the termination of the attack the transient pains, which are troublesome during its course, increase in severity, and rheumatic affections of an obstinate and painful character supervene. These often assume a remittent or intermittent form, returning regularly at the same hour for several days in succession, and not infrequently affecting one side of the head, or one eyebrow or eyeball, and occasionally the intercostal muscles of one side.

**2. Epidemic Catarrhal Fever with predominant Pulmonary Affection.**—Not infrequently the symptoms of implication

of the ærial mucous membrane or lungs are more marked, there being decided quinsy, laryngeal symptoms, croup, bronchitis, or pneumonia. Of these the most frequent and important are the bronchitic and pneumonic complications.

*Bronchitis.*—This has especially the tendency to assume the acute capillary form. In cases of *acute capillary bronchitis* occurring as a complication of influenza, there is usually in the *early* stage increased frequency and some difficulty of breathing, constriction of the chest, and soreness or stiffness of the throat. The cough, though slight, is troublesome from its frequency. The expectoration, if there be any, is scanty and of a glairy character. The tongue is usually red at the tip and edges, and covered on the dorsum with a creamy mucus or with a whitish-brown fur; occasionally it is morbidly red throughout. The pulse is accelerated, beating generally 110 to 112 or 116 in the minute, and occasionally more frequently. The skin is not usually hot, except it be at the commencement of the attack, and if so it usually becomes cooler in two or three days. With these symptoms there are the marked prostration of strength, the severe frontal headache, the general soreness of the body, and the pains in the back and limbs, which characterise the ordinary cases of influenza.

When the chest is examined in this stage of the disease, the only morbid signs detected are a roughness of the inspiratory sound, particularly when a forced inspiration is drawn; some slight crepitation, audible more especially towards the lower part of each dorsal region; feebleness of the vesicular murmur; and perhaps slight sibilant rhonchus in front. The respiration is, however, quicker and shorter than natural, averaging 28, 32, or 40 in the minute; the dyspnoea is greater than is explained by the obvious physical signs; and most generally there is some lividity of the face.

In the *second* stage all these symptoms are much aggravated. The respiratory acts are performed quickly and imperfectly, the respirations in the minute varying from 30 to 40 or 50. The cheeks are much flushed, and the lips of a somewhat purple colour. Generally there is not acute pain in the chest, but rather a sense of constriction and soreness; the cough, though frequent and occurring in paroxysms, is not usually severe. The expectoration still continues scanty, and consists of small yellowish-white pellets, forming tenacious masses of a peculiar nodulated or botryoidal form, very much resembling, when floating in water, fragments of some of the large oolitic limestones. The tongue is mostly covered with a thick whitish-brown fur, and somewhat dry and often red at the tip and edges; or morbidly red and glazed. The pulse is much accelerated, beating 120, 130, or 140

times in the minute, but it is generally small and very compressible. In some instances, after being low and feeble at the outset of the disease, it acquires a more sthenic character in the second stage. The skin is rarely dry, or much above the natural temperature; and the hands and feet are generally cool. The skin of the extremities is also much congested, so that when blanched by pressure, the colour does not readily return. The prostration of strength also becomes greater; and there is much headache, and often transient delirium, especially during the night. On percussion, the chest does not present any marked alteration of resonance, unless there exists some other disease of the lung; and on auscultation crepitation of a more or less fine character is audible with the inspiration, first in the inferior part of one or both dorsal regions, then spreading rapidly higher up in the back and toward the bases of the lungs in front, whilst sibilant rhonchus is heard in other parts of the chest.

The *third* stage of the disease is marked by the dyspnoea becoming so severe that the patients are compelled to sit constantly upright in bed, or to lean forwards, resting on their arms and elbows; whilst at intervals the respiration becomes very laborious. The lividity of the cheeks, lips, and hands is increased; the eyes become prominent; and the expression of countenance is extremely anxious. The cough is frequent, and of a short abortive character, giving the impression of viscid secretion in the lung, which the patient has not power to expectorate; whilst it is aggravated by paroxysms, which cause pain in the head and increased lividity. The sputum now becomes large in quantity; it is of a greenish-yellow colour, very viscid, contains little air, and is occasionally streaked with blood. The respirations are very frequent, 50 to 60, or even more; and expiration is very laboured and prolonged. The pulse is very feeble, and either extremely quick—140, 150, or 160 in the minute—or intermittent, so as to number only 100 or 120 beats. The tongue is covered with a thick yellowish-white or brown fur, and is generally dry; sordes also form upon the teeth. The general surface of the body becomes cool and bathed in perspiration; and the hands and feet decidedly cold. The delirium is more constant; with the decaying strength the cough declines; the expectoration ceases or becomes slight; and the patient sinks.

With the progress of the disease the physical signs change. The chest in front yields a uniformly clear sound on percussion, while behind there is a general impairment of the resonance. The crepitation gradually extends over larger portions of the lungs, being of a finer character in the parts more recently involved; giving place to subcrepitant and

mucous rhonchi in the situation in which it was first heard; and finally becoming of a gurgling character in the neighbourhood of the larger bronchial tubes. The subcrepitant rhonchus also towards the end of the attack is heard with the expiration as well as with the inspiration; and, if there is some local condensation, bronchial respiration may be developed.

In the cases which terminate favourably, the amendment is marked by the respiration becoming less hurried and laborious; by the expression of countenance being less anxious, and the face less livid; and by the prostration of strength decreasing. The pulse becomes less frequent, the cough less severe, and the sputum less viscid—more of a mucopurulent character, with a tendency to form a homogeneous mass, containing large air-bells. At a later period it becomes thinner, and declines in amount. The mucous and subcrepitant rhonchi are replaced by finer sounds, and the space over which the morbid signs are heard diminishes—these disappearing first in the upper part of the chest, then at the front and sides, and lastly in the dorsal regions. The clearness of the sound on percussion on the front of the chest also passes away, and is often succeeded by a somewhat dull sound; and the respiratory sounds become indistinct. The convalescence is, however, generally protracted; the patient is liable to returns of dyspnoea at intervals; and the cough continues troublesome. After the subsidence of the pulmonary symptoms, the rheumatic pains, if previously present, may become aggravated, or may then first appear and become troublesome, affecting the head, face, or intercostal muscles, and being aggravated towards night.

**ANATOMICAL CHARACTERS.**—On examination after death, one of the peculiar features of this form of influenza is the extremely inflated condition of the lungs, which, in lieu of collapsing when the chest is laid open, in some cases protrude from the cavity. This condition is not limited to certain parts in which there are larger or smaller bullæ, but consists in a general inflation of large portions of the lung. The mucous membrane of the bronchi is reddened, and the injection increases towards the smaller tubes, where the membrane may be intensely red and have a villous appearance. The lung-tissue in the cases proving fatal at an early period has a peculiar dry appearance, but in the later stages it is œdematous. There is also more or less lobular condensation, the condensed parts being depressed below the adjacent inflated tissue, and having a deep purple colour. At a later period the condensed tissue may soften from the presence of pus, and small cavities may be formed in this manner. The bronchial glands are enlarged and softened. The cavities of the heart, especially on the right side, are dis-

tended with firm and more or less decolorised coagula.

**PROGNOSIS AND DURATION.**—The cases of influenza complicated by acute capillary bronchitis are always very serious in their character; and when the disease occurs in persons who have before been out of health, and especially if there be any previous disease of the lungs or heart, or if the subjects be very young or elderly, they prove fatal in a large proportion of cases. The duration of the disease in the cases which prove fatal is from about ten to fourteen days; and when recovery occurs, the patient is ill from a fortnight to three weeks, or longer.

**Pneumonia.**—Of the symptoms in these cases, cough is one of the most troublesome; the expectoration is peculiar, partaking both of the glassy, transparent or opaque character of the sputum of bronchitis, and of the brownish viscid expectoration of pneumonia, with the usual small air-bells. In some cases, however, when there is great prostration, there may be no expectoration. Pain in the chest is generally experienced at the invasion of the disease, of a more or less severe character, but afterwards it is not present to any marked extent, or is only experienced when the patient has a severe paroxysm of coughing. The dyspnoea also is not severe, and there is not much lividity of the face, unless in cases in which there is also considerable bronchitis. The breathing is not usually very rapid, the respirations not exceeding 28 or 32 in the minute. The pulse also is comparatively quiet, beating 80, 90, or 100, and it is usually soft and compressible, or decidedly small and weak. The skin, as in the other forms of influenza, is not generally hot or dry; or, if so at the commencement of the attack, it soon becomes cool and moist. The tongue has usually the whitish-brown covering which has before been described, and is not dry; but sometimes it does become dry and brown. There is often very marked disorder of the digestive organs—sickness and vomiting and diarrhoea; and usually some slight jaundiced tinge of the skin is observed. There is also not infrequently some delirium and stupor of mind; and the prostration of strength is often extreme.

On examining the chest, in addition to the signs of bronchitis, which are always present to a greater or less degree, there are the usual indications of pneumonia. At first fine crepitation is perceived in the seat of the disease; to this more or less marked dulness on percussion, bronchial respiration, and increased resonance of the voice and cough, succeed; and these signs, though generally found in one or both dorsal regions, may be more widely diffused. Notwithstanding, however, the threatening character of the symptoms, it was observed

in the epidemic of 1847 that the disease was not very fatal, and the signs of consolidation disappeared more readily than in most ordinary cases of pneumonia. The resolution was shown by the return of crepitation, though of a coarser character, in the seat of consolidation; and by the gradual diminution of the bronchial respiration, and of the dulness on percussion. After a time the natural vesicular breathing again became audible. The duration of the disease was very similar to that in the cases of acute capillary bronchitis. In this, as in the other forms of influenza, the convalescence was often very protracted; there was the same tendency to recurrence of the symptoms on any slight exciting cause; and the rheumatic pains often continued long to distress the patient.

**3. Catarrhal Fever, complicated with gastro-intestinal affections and rheumatism, and of a remittent character.**

It has already been stated that more or less marked symptoms of gastro-intestinal and hepatic disorder may be present; and that rheumatic pains, having a more or less decidedly remittent character, may be observed. It has further been said that in the cases in which the pulmonary complication assumes the pneumonic form, these symptoms may be generally more marked than in the cases of acute capillary bronchitis.

In some cases, however, the latter symptoms were, throughout, the predominant feature; and as similar observations have been made as to former epidemics, cases of this kind may fairly be regarded as constituting a special form of the disease.

In this form of the epidemic, nausea and sickness generally occurred at an early period of the attack, and often became very urgent symptoms. The matters vomited often had a bilious tinge; and there was generally a marked bilious tinge of the conjunctivæ and general surface of the body, amounting in some cases to decided jaundice. There was often diarrhœa; and sometimes blood was passed in the stools. The pains in the head, back, loins, and extremities, which are present with more or less severity in all forms of influenza, were from the first of a severe character, or increased with the progress of the disease till they constituted a predominant feature. The pain was usually most distressing in the head, especially in the forehead, and in some cases was limited to one temple, one eyebrow, or one eyeball. This was generally of an intermittent or remittent character, coming on at night after more or less distinct remission during the day; and was attended with singing in the ears, distressing restlessness, agitation, and inability to sleep, whilst delirium generally increased during the exacerbation. In some cases it only amounted to a little excitement and incoherence, but in others it was so

urgent as to require the employment of restraint to retain the patient in bed, and sometimes it continued without intermission for some hours. It was, however, remarkable how completely it subsided, as a rule, in the morning. With these symptoms there was usually much tremor of the extremities; and the eye was pale and glassy, though the pupil was frequently contracted.

Early in the attack the pulse was quick and feeble, and of a peculiarly vibratory character, though sometimes it was but little accelerated. At a later period it often became very rapid and feeble, or fell below the natural standard of frequency, being very soft and compressible, and occasionally intermittent. The tongue was at first moist, covered with the usual whity-brown fur, and red at the tip; subsequently it had a tendency to become dry. The breath had a peculiar, offensive, acid odour. Epistaxis occasionally occurred, and sometimes to an alarming amount. The skin was generally moist, and the perspiration had the usual sour rheumatic smell. The urine was at first scanty, but afterwards increased in quantity, and deposited much sediment of urates. Occasionally it was slightly albuminous.

With these symptoms there were evidences of some pulmonary disorder—bronchitis or pneumonia; and very frequently murmurs were heard at the heart, which were not, however, generally persistent.

After the exacerbations the sweating was often very profuse, so that in the morning the patient was found completely bathed in perspiration, and the bedclothes were quite wet. The prostration of strength also was often extreme, so that the hands and feet became livid and cold, and the patient resembled a person in the algide stage of cholera.

Notwithstanding their severity, the cases of this third form of influenza generally did well, though the patients long suffered from the rheumatic symptoms, and only very slowly recovered their strength. As seen from the Registrar-General's reports, there was a great increase in the deaths from 'rheumatism' during the prevalence of the epidemic, which probably referred to cases of this kind.

**4. Modifying Influence of the presence of Influenza on other Diseases.**—In all visitations of influenza of which we have detailed accounts, the epidemic has been attended by an unusual prevalence of other forms of disease, and especially of the specific fevers—and the features of such diseases have been much modified by the epidemic influenza. To this rule the influenza of 1847 affords no exception. As shown by the reports of the deaths in London and the country generally, there was a great increase in the total mortality; this especially showed

itself in the zymotic class of diseases, but obtained also as to diseases of the heart, brain, and digestive organs. An unusual number of deaths were recorded from 'typhus,' under which head were then included the fevers which we now discriminate into typhus, typhoid, and relapsing fever. Of all these forms of disease, cases occurred during the time, though the chief prevalence was apparently of typhoid and relapsing fever. The features of these diseases were generally so much modified by the epidemic influence, and they were so constantly attended by pulmonary complications, that it was often difficult to decide whether any given case was to be regarded as a case of specific fever or of influenza. Various forms of local disease were also prevalent at the time, both as distinct affections, and as complications of specific fever, such as diphtheria, parotitis, otitis, stomatitis, and quinsy, with erysipelas, abscesses, &c.; and these were often attended by great prostration of strength, and added greatly to the danger of the various other forms of disease with which they were combined.

**PATHOLOGY.**—Every phenomenon of influenza points conclusively to the influence of some powerful depressing agent, operating on the nervous system, or entering the blood. The sudden seizure of a large proportion of cases; the extreme prostration of strength from the commencement, and to a degree disproportioned to the amount of local disturbance; the symptoms of disorder of all organs, and especially of the cerebro-spinal system; and the debility which so often succeeds even simple cases of the disease, can on no other supposition be explained. It seems probable also that the affection of the respiratory mucous membrane may be due to the morbid influence, whatever it may be, operating more specifically upon it; but it is extremely difficult to offer even a probable suggestion as to the nature of that influence. The almost simultaneous outbreak of the epidemic in places widely apart; the seizure of a large proportion of the population of a town or district within the course of a few hours; and the sudden illness of individuals or bodies of men visiting a locality where influenza is, or has very recently been, prevailing, and previous to direct intercourse with any persons actually suffering—are all circumstances opposed to the notion of the disease being dependent on infection in the ordinarily understood sense; and might seem to point to the operation of atmospheric influence as the cause. The conditions, however, under which the disease has presented itself in different epidemics render such an explanation impossible. It has travelled over districts without reference to season or climate, and has prevailed in the same locality in all seasons and in almost every variety of weather. It is true that it has often broken

out after great meteorological changes, as in the last two epidemics after sudden and remarkable falls from a comparatively high to a very low temperature; but, on the other hand, epidemics have arisen under other circumstances, and such alternations of temperature are frequently observed without the occurrence of an epidemic of influenza. The disease has also broken out at the same time at different places in which the same atmospheric conditions did not exist, so that the operation of the sudden change can scarcely be regarded as acting even as an exciting cause. There can, however, be no doubt that the more common predisponents to disease, such as defective drainage, overcrowding, impure air, deficient clothing, and insufficient or unsuitable food, powerfully conduce to the prevalence and fatality of an epidemic of influenza.

**TREATMENT.**—Of the treatment of the simpler forms of influenza it is not necessary to say much. The patient should be confined to bed; have a foot-bath; take some form of diaphoretic medicine; and be allowed a mild, unstimulating diet. If the cough becomes troublesome, or if there be pains in the chest, sinapisms may be applied, and some anodyne may be added to the medicine; and for the relief of the subsequent debility, stimulants, tonics, and a nutritious diet may be enjoined. In the cases complicated with pulmonary affections, the same measures may be used, in combination with expectorants and anodynes; and more decided counter-irritation may be applied to the chest. When there is very copious secretion, and the patient cannot expectorate freely, the greatest benefit is often derived from the use of emetics, and they may be employed with advantage in cases in which the patient's strength is too much depressed to allow of nauseating doses of medicine being given. To the use of emetics, the stimulating expectorants—squill, ammonia, &c.—should succeed. For the relief of the cases in which there is nausea and sickness or vomiting, effervescent with morphine, or hydrocyanic acid, may be given. When there is more or less jaundice, small doses of calomel or grey powder, in combination with Dover's powder, are very beneficial; and when there is diarrhoea, the Dover's powder or decided astringents—such as acetate of lead, or tannic acid—may be employed. When the rheumatic symptoms are severe, the greatest relief is obtained by the administration of small doses of colchicum with bicarbonate of potash and opiates; and when the prostration of strength is great, ammonia, in combination with bark, should be given. In the cases in which there is a decided tendency to remissions and exacerbations, bark also may be prescribed, or, in the more severe cases, quinine; and quinine and other tonics should be freely given during convalescence.

All depressing treatment should be avoided. The patients are too much prostrated to admit of its employment. In each of the different forms of influenza it is necessary to administer support very freely, and sooner or later to exhibit stimulants. In the cases in which there is pulmonary or other local complication, the strength becomes more rapidly and more seriously depressed, and stimulants and support are still more urgently needed; and, indeed, it is necessary to have recourse to them at an earlier period, to exhibit them more freely, and to persevere in their use for a longer period in such affections, when occurring during an attack of influenza, than when arising as idiopathic diseases. In the management also of the other forms of febrile affections which are seen during an epidemic of influenza, a more restorative and stimulating treatment is required than under ordinary circumstances, for all such diseases partake of the peculiarly asthenic type of the epidemic.

THOMAS B. PEACOCK.

### The Epidemics of 1889-90-91-92.

**OCCURRENCE.**—No well-marked widespread epidemic of influenza occurred after that of 1847-8 until 1889. The epidemic which commenced in Europe in the autumn of that year closely resembled previous visitations. It was present at Bokhara, in Central Asia, in May 1889; it was first recognised in St. Petersburg about mid-October; in Berlin and Paris towards the end of November; in Spain and Italy early in December; and in the United Kingdom in the last days of December—although in the light of subsequent knowledge it can be shown that cases occurred in London six weeks or two months earlier. On the western shores of the Atlantic it appeared first in Jamaica early in December; in the eastern ports of the United States about ten days later; and in those of Canada after a further interval of the same duration. The general direction of progress would therefore appear to have been from east to west; but to this there were many exceptions. In Africa the epidemic appeared at Alexandria at Christmas 1889; at Cairo a fortnight later; in Cape Colony in January; in Basutoland two months later; in the Shiré Highlands in October; and in Mauritius in August 1890. India, Ceylon, and Japan were invaded in February, New Zealand and Australia in March, 1890. No transference from east to west, moreover, could be observed in each country invaded; the epidemic, on the contrary, appeared to radiate from the seaports or large towns. In this country, for example, London appears to have been the first English centre of the disease in 1889; Hull in 1891. It is probable that Great Britain had not been entirely free from influenza in the interval between the subsi-

dence of the epidemic in the spring of 1890 and its recrudescence in 1891, but the first town that suffered from distinct epidemic prevalence was Hull; the epidemic began there in the second week of March. Sheffield was attacked about three weeks, Leeds and Bradford about five weeks, later. These towns, with the exception of Hull, had undoubtedly suffered from the epidemic in the previous year. The epidemic also reappeared in London, and affected many of the towns and villages in Yorkshire and the midland counties, but was less widespread than in 1890. The epidemic recurred in the autumn of 1891; in Great Britain it prevailed first in the east of Scotland and the south-west of England. It spread from these two districts over the whole country, and appeared early in the north of Ireland. During the winter of 1891-92 also influenza prevailed as an epidemic in every civilised country, and in every quarter of the globe.

**FORMS.**—The three clinical forms described by the late Dr. Peacock were observed during the latest epidemics. The following classification, which has been very widely accepted, will serve to indicate the general characters of the forms recently most prevalent.

**A. Catarrhal**, marked by conjunctivitis, and a tendency to complications affecting the respiratory tract—pharyngitis, tracheitis, bronchitis, broncho-pneumonia, and pneumonia.

**B. Gastric**, marked by sudden onset of vomiting and diarrhoea.

**C. Nervous**, marked by severe headache and backache, and great depression.

The last-named form was that most often met with. The patient after a slight chill is seized with pain in the eyeballs, frontal headache, and pains in the back and limbs; the backache is in many cases very severe, and has been compared by women who have borne children to the pangs of childbirth. The temperature rises rapidly, reaching its maximum, in a large proportion of cases, within twenty-four hours; the rise is generally limited to 2° or 3°, but temperatures of 104° F., and even higher, have been noted. The duration of the febrile period varies from one to nine or ten days, but is usually three, four, or five days. The maximum is, as a rule, only maintained for a few hours. The fall is sometimes rapid: a distinct crisis, accompanied by profuse sweating, occurs, and the temperature declines to the normal, or even lower, in twelve hours. In other cases the fever has a remittent character, with regular daily fluctuations by which the temperature returns to the normal in two or three days. Relapses were more frequently observed in the recent epidemics than in any previous one: after from four to six days of freedom from fever the temperature again rose, and all the symptoms recurred; as many as three such relapses have been observed.

In a few cases in which the symptoms were otherwise well marked, the temperature remained at or below normal throughout.

The pulse-rate rises with the temperature, but the pulse, even when very rapid, remains soft; slight cyanosis is not uncommon during the pyrexia, and death is sometimes due to cardiac failure; a remarkably slow pulse (50 or less) is occasionally observed during defervescence. During the acute stage the patient experiences great depression and lassitude—symptoms which persist after the pain and pyrexia have disappeared, and are often aggravated by insomnia; in some cases convalescence is prolonged for weeks, or even months, and occasionally well-defined melancholia develops. The prostration is accompanied by anæmia. Acute meningitis, peripheral neuritis, and myelitis may be mentioned among the rarer complications, while neuralgia of various regions is frequently observed, and sometimes persists for long periods. Loss of the sense of taste is a not uncommon symptom, and frequently continues beyond the period of convalescence. Occasionally, either at the onset or during defervescence, the patient passes into a condition of stupor which may be mistaken for catalepsy. The pains in the limbs are sometimes accompanied by a distinct arthritis. Epileptic symptoms of a mild form (*petit mal*) are not infrequent.

The symptoms observed in the gastric form are such as those described by Dr. Peacock under his third form. Children in particular suffer from this form: a sudden attack of vomiting is followed by diarrhœa, the stools being not infrequently bloodstained. The degree of prostration is often out of proportion to the severity of the pyrexia or diarrhœa. The icteric tint of skin, the epistaxis, and the albuminuria noted by Dr. Peacock have again been observed, and a fuller description of the symptoms would merely involve a repetition of what he has written.

The same remark applies to cases with complications of the respiratory tract. Catarrh of the conjunctivæ, nose, and pharynx is present in a very large proportion of these cases, and bronchitis and broncho-pneumonia frequently appear to be merely extensions of the catarrhal process traceable to imprudent exposure. But in some cases pneumonia seems to be the earliest manifestation of the disease. This pneumonia, though lobar in distribution, is probably always catarrhal in type; it is associated with the presence of the streptococcus pyogenes, and ought perhaps to be regarded as a complication occurring very early in the disease; it is frequently accompanied by pleurisy, and occasionally results in pulmonary abscess or in pyo-pneumothorax. The temperature curve is modified essentially by these complications, which tend to prolong the period of pyrexia; when broncho-pneu-

monia is present, the fall which precedes convalescence is slow and irregular.

Among the very numerous occasional symptoms or complications to which attention was directed during this epidemic, reference may be made to the following. A rash, in some cases erythematous, in others a scattered papular eruption, is occasionally observed. Herpes, sometimes very widespread, and an affection of the cornea apparently of an herpetic character, have also been recorded. Purpura, sub-conjunctival hæmorrhage, and hæmorrhages from the stomach, lungs, or uterus appear to have occurred in an unusual proportion of cases in some localities. The statements of different observers as to the condition of the spleen are conflicting, but it was probably enlarged in half, or in more than half, the cases. Inflammation of the middle ear has been frequently noted.

*Morbidity and Mortality.*—One attack of influenza does not afford more than a very imperfect protection against a second. The proportion of the population affected by the epidemic has been very variously estimated: in St. Petersburg it was put at two-thirds, in Berlin at one-third, in London at 13½ per cent. Among the soldiers of the Home District during the first eighteen days of the epidemic (after which it very rapidly declined) the percentage admitted for treatment was 10·57, while one-third of the persons employed in the General Post Office suffered. Mr. Smeë has estimated that 8 per cent. of the total population of Europe were attacked during 1889–90. The mortality ascribed to influenza directly was not large, but on this as on previous occasions the occurrence of an epidemic of influenza has had a very remarkable effect upon the death-rate of a town. In London, for instance, the death-rate rose from 20·3 in the week ending December 28, 1889, to 32·4 in the week ending January 11, 1890; in 1891 the death-rate in Sheffield rose from 21·7 in the week ending April 4 to 70·5 in the week ending May 2; in Paris at the height of the epidemic the number of deaths from all causes was nearly trebled. An analysis of the vital statistics of the towns and countries affected shows that the excessive mortality produced by influenza is due mainly to a large increase in the number of deaths attributed to diseases of the respiratory organs, and secondarily to an increase in those attributed to diseases of the circulatory system. In London the number of deaths registered in the four weeks of 1890 during which the epidemic was at its height was 2,258 above the average; the deaths due to diseases of the respiratory organs were 1,791 above the average; while those due to diseases of the circulatory system were 318 above the average. This excess of deaths due to respiratory diseases was made up as follows: Bronchitis, 911; pneumonia, 465; phthisis, 337;

other diseases of the respiratory organs, 78. Bronchitis and pneumonia have already been enumerated among the commonest complications of influenza; the increased number of deaths from phthisis was due partly to the prostration which accompanied influenza, but mainly to the broncho-pneumonia which it produced.<sup>1</sup>

**MORBID ANATOMY AND PATHOLOGY.**—The morbid changes found after death from influenza are of a very varied character, and are in the main to be attributed to the complications by which death has been brought about. Those due to influenza itself are of a character common to all forms of acute infective disease—namely, parenchymatous degeneration of the liver, kidneys, and spleen, of the muscular substance of the heart, and of the minute vessels. The last named may lead to capillary hæmorrhages into the viscera or nervous centres. The spleen has been found much enlarged and almost diffuent. Numerous bacteriological examinations have shown that in pneumonia complicating influenza the diplococcus pneumoniæ is present in some cases, the streptococcus pyogenes in others. In the bronchial sputum these two microbes have also been found, while in the secretion of the nasal cavities the staphylococcus aureus and albus and the capsule-bacillus of Friedländer, as well as two species of bacilli capable of producing pneumonia in rodents, have been detected. The micro-organisms thus far enumerated must be regarded as the causes, not of influenza, but of its respiratory complications. Klebs has stated that the blood during the febrile stage contains flagellate protozoa in very large numbers, either free in the plasma, or adhering to or within the red blood-corpuscles; he compares them to the monads of malaria. His observations have not been confirmed.

More recently (January 1892) a bacillus, previously unknown to bacteriologists, has been asserted to be the specific exciting cause of influenza. Pfeiffer, who found it in all the patients (thirty-one) he examined, states that in uncomplicated cases it exists alone, and in enormous numbers, in the bronchial secretions, and is capable of penetrating into the peribronchial tissue, and even to the surface of the pleura. Canon found it in the blood during the febrile stage, and in larger number during defervescence. The bacillus, which is very minute, being smaller than that of mouse-septicæmia, grows at temperatures above 28° C. on glycerine-agar, forming small colonies looking like drops of water. These colonies do not run together, but remain dis-

tingent. The bacillus, which is immobile, also grows in *bouillon*, forming in time a white woolly mass at the bottom of the vessel.

**ÆTIOLOGY.**—The history of the epidemic now under consideration strongly supports the view that influenza is propagated mainly, if not entirely, by human intercourse. The evidence in favour of this has been fully stated by Dr. Parsons.<sup>1</sup> The epidemic has not travelled faster than human beings could travel, but it has travelled faster during this epidemic than at earlier dates, when means of communication were less rapid. It has in each country appeared first in towns; and the towns earliest affected have been the capital, or seaports, or frontier towns having trade with countries already affected. There has been no steady progress from east to west, either over the world or in particular countries; and neighbouring communities have often suffered at considerable intervals of time. The recent epidemic has not afforded indisputable instances of sudden outbreaks without antecedent cases; but, on the other hand, a large amount of evidence has accumulated proving that the disease has been introduced into each district by individuals coming from places where the epidemic already prevailed, and that the first cases in the new localities have occurred in persons brought into intimate relations with the new arrivals. The interval between the first case of the disease in a community, and the next developing in relation with it, has been found to vary from two days, or less, to seven or eight days. A considerable number of instances has been recorded in which an attack of the disease has followed a short interview with a patient already suffering; the usual interval in these cases has been two to three days. If the earlier cases were overlooked owing to their being few in number and of a benign character, it would appear as if the disease had suddenly broken out, and not that it had merely increased in severity and in the number of its victims.<sup>2</sup> The influence of human intercourse is also seen in the following facts: (1) Persons living in suburbs going daily to business in the town have generally been the first to suffer, other members of the households, and persons employed in the suburbs, being attacked later; (2) persons employed together in large numbers in enclosed spaces have suffered in larger proportion than those em-

<sup>1</sup> These paragraphs have been re-written since the publication of Dr. Parsons's exhaustive report (*Local Government Board Report on the Influenza Epidemic of 1889-90*, by Dr. Parsons); but the writer had previously become convinced of the correctness of the view therein set forth.

<sup>2</sup> If it be assumed for any disease that it has an incubation period of two days, and that each patient communicates the infective principle to two others, then the number of persons attacked on the twenty-first day would be over one thousand, and on the twenty-seventh day over eight thousand.

<sup>1</sup> The Registrar-General states that, in 1890, the number of deaths in England and Wales directly attributed to influenza was 4,523, but he estimates the number directly and indirectly due to it at 27,000, which is equivalent to a death-rate of almost 1 per mille.

ployed few together or in the open air;<sup>1</sup> (3) in institutions in which the inmates were brought much into association, the spread of the epidemic has been more rapid and extensive than in those in which the inmates were more secluded from one another.

The fact remains, however, that it is not always possible to trace communication between an infected area and that in which a fresh outbreak occurs; and the acceptance of the view that influenza is, as a rule, transmitted by human intercourse, does not necessarily involve the assumption that the virus cannot be propagated outside the human body.

The opinion that influenza may be conveyed by direct infection from the domestic animals, more especially the horse, found support in the prevalence of the disease called influenza among horses in England for some time before the epidemic outburst, and in its reappearance in 1892. Similar observations have been made during previous epidemics, but on other occasions influenza has prevailed as an epizootic without any coincident epidemic; persons employed in attending on horses suffering from influenza have not suffered earlier or more than other members of the community, and influenza has occurred among stablemen without any coincident epizootic. Under the name 'influenza,' however, several diseases of the horse are probably confounded; and though recent evidence is opposed to the view that the equine disease can be communicated to man, the possibility of such an origin cannot be absolutely denied.

The sudden onset of the disease, the short duration of the febrile paroxysm, and the occasional occurrence of relapses, have been quoted in support of the theory that influenza is allied to malarial intermittent. The mode of spread of the epidemic along commercial routes, its rapid dissemination, and the absence of any constant antecedent meteorological conditions, militate against the view that it is a miasmatic air-borne disease.

The resemblance of the disease to dengue (*see* DENGUE) has been pointed out by many writers, some of whom have held that the recent epidemic was dengue. In various parts of the Levant an epidemic of dengue preceded by a few months the recent epidemic of influenza, which there presented the same clinical features as in Northern Europe. Dengue, however, is a disease of warm climates and of hot seasons; it spreads much less rapidly, and an epidemic persists in a locality much longer; further, though individual cases of the two diseases may resemble each other

<sup>1</sup> For instance, among men employed by the London and North-Western Railway the proportions were very different: 4·7 per cent. of the drivers and firemen, and 11·5 per cent. of the clerks, were attacked; of 122 seamen employed by the same company 6 were attacked; of 709 hotel servants 100 were attacked.

very closely, the general clinical picture is different. In dengue the fever is generally higher, an exanthem is the rule, the pains in the limbs are more distinctly localised in the joints, and the complications so commonly observed with influenza are rare in dengue.

As to the mode of origin of epidemic influenza, and the circumstances which favour its wide dissemination, nothing has been certainly ascertained. It has been imagined that under certain favourable conditions it may be evolved from 'a common cold,' and in support of this hypothesis it has been pointed out that the early cases in each country during the recent epidemic were of a mild type, and that the latest were often the most severe. The nature of these favourable conditions, however, has not been defined, and it can only be said that they have nothing to do with temperature, moisture, or wind, and are, in fact, entirely unknown.

It has been asserted that influenza is endemic in various parts of the Russian empire and in China, and it is the fact that the direction in which the epidemic spread on this, as on former occasions, is not incompatible with the view that it had its origin in Eastern Asia.

TREATMENT.—But little can be added to the observations made under this head by Dr. Peacock. No specific has been found, and the treatment adopted has been mainly symptomatic. Phenazone and phenacetin were used with some success to subdue the severe headache and muscular pains; salicylate of sodium or salicin seemed to be of value, especially in cases presenting rheumatic symptoms; restlessness and insomnia have been treated by preference with bromide of ammonium. The danger of using depressant remedies is universally recognised. Quinine has been extensively used, both as a prophylactic and in the early stage of the attack; its value, whether as a prophylactic or as a remedy, is probably very small. The preparation most generally used was the ammoniated tincture, and the moral effect of moderate doses of this not unpalatable drug has probably been distinctly favourable during the first days of alarm and panic.

The main point in successful treatment is a recognition of the truth that influenza is a serious malady, and not to be compared to a 'common cold.' A patient who fights against the disease in its early stage, or who returns to work too soon after the subsidence of acute symptoms, is far more likely to suffer from the severe pulmonary complications, more especially pneumonia, than one who submits to treatment early, and continues to take precautions against cold and over-exertion until his strength is completely regained. There is, probably, no one circumstance which would tend so much to diminish the mortality from influenza as attention to the rule which has been formulated by some—

forty-eight hours in bed after the temperature has become normal.<sup>1</sup>

If the view be accepted that epidemic influenza is, in nine hundred and ninety-nine cases out of a thousand, communicated by infection from the sick to the healthy, then the propriety of protecting delicate and aged persons, and patients suffering from phthisis or other pulmonary disorders, is obvious. The view in question is so highly probable that it would appear to be proper to act upon it.

DAWSON WILLIAMS.

**INHALATION, Ætiology of.**—See DISEASE, Causes of.

**INHALATIONS, Therapeutic Uses of** (*inhalo*, I breathe in).—Inhalation is a method of applying remedial agents to the respiratory tract, whereby these substances in a gaseous or atomised form are brought into contact with the mucous membrane of the nose, mouth, pharynx, larynx, and bronchi, and may even penetrate to the epithelium of the air-cells. Inhalation dates from the days of Hippocrates and of Galen, and has been more or less in vogue in all ages; but only lately has it been proved that the inhaled material passed through the finest ramifications of the bronchi into the pulmonary alveoli, and in some instances became absorbed by the capillaries, thus entering the general circulation. The examination of the lungs of colliers, grinders, and others engaged in dusty occupations has shown that the inhaled dust can be detected in the lung-tissue, where it induces chronic pneumonia. The experiments at the Académie de Médecine proved that medical sprays are equally penetrating.

**METHODS.**—The modes of inhalation vary with the drug used, depending mainly on the temperature at which it volatilises, and also on the medicinal effects aimed at.

Chloroform, ether, bichloride of methylene, and nitrite of amyl evaporate at ordinary temperatures, and only need to be diluted with air to be safely inhaled. Calomel and sulphur are sublimed at high temperatures in special apparatus; but the majority of drugs are best vaporised through the medium of hot water or steam, or reduced to fine spray by passing compressed air through their solutions.

Many forms of inhaler are in use, but in selecting one for warm inhalations the requisites are—(1) that it can be used without difficulty by the patient; (2) that a temperature of 130° to 150° F. can be steadily

maintained; (3) that the steam be thoroughly impregnated with the medicament; and (4) that the inhaling tube be fitted to the nostrils as well as to the mouth, so as to ensure a sufficient supply of the inhaled vapour. Lee's steam-draught inhaler is especially useful for projecting a full current of steam into the throat and air-passages. The force and temperature are alike controlled by the sliding valve on the inhaling pipe. When no inhaler is at hand, a jug with a wide mouth, and half filled with hot water, may be used, a towel being placed round between the mouth and nose of the patient and the opening of the jug, to prevent the escape of the vapour into the air.

**VARIETIES AND USES.**—Inhalations are employed chiefly in diseases of the pharynx, larynx, and air-passages, and may be classed as *sedative*, *stimulant*, and *antiseptic*.

*Sedative.*—Steam is soothing to the throat, when this part is dry, inflamed, or irritable. In incipient laryngitis and croup, as well as in irritable bronchitis, the inspiration of steam from specially arranged kettles is very grateful, the moist vapour promoting secretion and expectoration.

Jets of steam are used in hospitals and bath establishments as vehicles for the inhaled drugs, and are directed into the patient's mouth.

As sedatives the vapor coninæ and the vapor acidi hydrocyanici of the pharmacopœia are recommended in cases of laryngeal or pulmonary irritation. A sedative inhalation made of one part of chloroform and two or three of rectified spirit—of which one teaspoonful may be added to a pint of water, at 60° to 100° F.—is much commended in hay fever and laryngeal spasm. A combination of chloroform and conium, in the proportion of 5 minims of the former to 1 drachm of the succus conii, in half a pint of boiling water, is useful in phthisis and some forms of asthma. One drachm of liquor potassæ should be added, for this promotes the volatility of the conine, as in the official vapor coninæ. It must be remembered that chloroform is very powerful when given with steam as an inhalation. One drop of chloroform in a pint of water at 150° may produce giddiness.

*Stimulant.*—Stimulating moist inhalations can be prepared with various volatile oils. Oils of pine and of cubebs are useful stimulants in cases where there is much secretion from the throat and air-tubes. Thus 2 drachms of oil of pine or oil of cubebs may be mixed with 60 grains of light carbonate of magnesium in 3 ounces of water; and of this mixture 1 drachm may be used in a pint of water at each inhalation. One drachm of the compound tincture of benzoin, in half a pint of hot water, is often useful in chronic bronchitis and laryngitis. Oil of turpentine or the vapor olei pini sylvestris of the pharmacopœia makes an excellent

<sup>1</sup> It has been asserted that recent vaccination affords some protection against influenza. The experience of Sheffield, where a severe epidemic followed shortly after an epidemic of small-pox had led to a very general resort to revaccination, appears to be sufficient to disprove this theory, which, in fact, seems to rest upon no solid groundwork of facts.

stimulant inhalation in cases of dilated bronchi.

*Antiseptic.*—Antiseptic inhalations are used where the object is to correct a fetid secretion, as well as to stimulate the secreting membrane to fresh action. In fetid bronchorrhœa, in gangrene of the lung, in fetid abscess, and in pyo-pneumothorax, benefit is derived from the vapor creasoti, the official vapor iodi, or from glycerine of carbolic acid (from 2 to 3 drachms to the pint of boiling water), or again from oil of thymol, prepared like the other essential oil vapours, with light magnesia and hot water (60 grains to the 3 ounces). Carbolic acid vapour can be well diffused in a room by mixing the acid with water in Lee's steam-draught inhaler, or by dropping the acid on a hot plate of metal as in Savory & Moore's carbolic vaporiser. Creasote or eucalyptus oil, mixed with an equal quantity of ethylic alcohol, is often employed in the form of drops (10 to 20 drops at a time) on a small sponge placed in a perforated zinc respirator. The respirator thus charged is worn by the patient for one hour morning and evening, with a view to improving the state of the lungs. After a free morning expectoration the respirator-inhaler should be used, and instruction given to inspire deeply by the mouth and expire through the nostrils with the mouth closed.

*Antispasmodic.*—The fumes of burning nitre-paper are employed as inhalations in cases of pure spasmodic asthma uncomplicated with bronchitis. The paper—prepared by soaking white blotting-paper in solution of nitrate of potassium (30 to 40 grains in an ounce of water)—is sometimes washed over with tincture of benzoin, and this, in certain cases, may be an improvement; but in ordinary forms of asthma the nitre-paper, burnt till the patient is enveloped in smoke, will usually relieve the asthmatic paroxysm. The fume of a grain of powdered opium volatilised on hot metal has been praised as a remedy to cut short nasal catarrh; as well as smoking solid opium in the Chinese fashion through a pipe, in spasmodic asthma. Cigarettes, pastilles, and powders containing stramonium, belladonna, and other antispasmodics, are well known as remedies in spasmodic asthma.

*Atomised inhalations of spray* have of late years come into deserved repute as valuable aids to the treatment of chronic diseases of the throat and lungs. The principle of the hand-ball and steam-spray atomisers is that, if two capillary tubes are placed at a certain angle to each other, one dipping into a fluid, while through the other a stream of air is driven by heat or compression, a vacuum is formed in the first, causing the liquid to pass out in the form of fine spray. In using these instruments, the operator should seek to blow the spray into the

patient's throat at the time when an inspiration is being taken, as thus the spray will obtain a free entrance through the larynx into the trachea. It is doubtful if much spray enters the air-tubes; some certainly does, as has been stated above, but the cold sprays do not appear to afford so much relief to affections of the lungs as the warm inhalations. In cases of tumidity of the larynx, a spray containing 10 grains of alum to 1 ounce of distilled water may be used. In place of alum, 2 grains of sulphate of iron, 5 grains of sulphate of zinc, or 5 grains of dry chloride of iron, in 1 ounce of water, may be employed. For antiseptic purposes, 5 minims of sulphurous acid, or 1 minim of carbolic acid, to 1 ounce of water, or a like quantity of solution of iodine, may be used. In putrid sore-throat and diphtheria the writer has seen excellent results from iodine, either inhaled in vapour or applied in solution. Three and a-half drachms of lactic acid in 10 ounces of water form a solution which, thrown as spray into the throat, is of use in diphtheria; it seems to dissolve the exudation. As a styptic and hæmostatic spray 50 to 200 grains of tannic acid are employed, dissolved in 10 ounces of water, but for relaxed throat a weaker solution is useful. For sedative purposes a solution of bromide of ammonium, or one containing half a grain of acetate of morphine to 1 ounce of water, may be employed. Diluted ipecacuanhawine spray is said to be very efficacious in relieving the dyspnoea of chronic bronchitis and emphysema. This spray in a few instances may induce vomiting, but this accident may be obviated by diluting the wine with a considerable proportion of water. All these spray solutions should be filtered through paper, otherwise they may contain small particles which clog the aperture of the atomiser. At the Continental spas it is usual to medicate large chambers by means of sprays and vapours, in which patients can sit for hours breathing the artificial atmospheres; and in this way various mineral waters, such as those of La Bourboule, Aix-les-Bains, and Caunterets are locally applied.

*Insufflation* is a method of applying powders to the larynx. A vulcanite tube curved at a suitable angle is carried over the tongue to the laryngeal aperture, and then the powder, which has been introduced through a small opening covered by a slide in the tube, is blown into the larynx and trachea. The blowing is effected sometimes by an elastic ball at the end of the tube, at other times by the mouth of the operator. In laryngeal phthisis  $\frac{1}{10}$  to  $\frac{1}{4}$  of a grain of hydrochlorate of morphine, mixed with sugar of milk, may thus be blown into the larynx, and often with great comfort to the patient. A grain of iodoform and a grain of boric acid are often added to the morphine powder.

JOHN C. THOROWGOOD.

**INJECTION** (*injection*, I throw in).—**SYNON.**: Fr. *Injection*; Ger. *Einspritzung*.

**DEFINITION.**—Injection is the act of introducing a fluid into any part of the body, by means of a syringe or similar apparatus. The word is also employed to designate the fluid so introduced.

**VARIETIES.**—Injections are chiefly used in the treatment of disease; but reference must also be made to the method as it is practised by anatomists for the preservation of 'subjects' for dissection; and for the purpose of filling the blood-vessels, lymphatics, ducts, cavities, spaces, &c., preparatory to microscopical examination.

The therapeutical application of injections comprehends the following measures:—

1. *Hypodermic* or *subcutaneous* injection, in which the fluid is injected under the skin. **See** HYPODERMIC INJECTION.

2. Injections into the natural *canals* or *open cavities* of the body; for example, the external ear, the Eustachian tube, the nose, the nasal duct, the stomach, the rectum (*see* ENEMA), the urethra, the bladder, and the vagina and uterus. The various forms of injections just indicated will be found fully discussed under the diseases of the several organs.

3. Injections into *shut sacs*, normal or morbid; such as the tunica vaginalis, the serous cavities, the synovial cavities, the sheaths of tendons, and cysts and chronic abscesses. The fluids used in this class of injections are generally stimulant, such as a solution of iodine. **See** HYDROCELE.

4. *Intravenous* injections, the fluid introduced into the circulation being either blood (transfusion), saline solution, milk, or some other kind of nutritive material. **See** TRANSFUSION.

J. MITCHELL BRUCE.

**INOCULATION** (*in*, into; and *oculus*, a bud, a graft).—As usually understood, inoculation is either an operative procedure or an accidental occurrence, by means of which morbid materials are brought into direct contact with the minute vessels of the skin or of a mucous membrane, or with those of the subcutaneous or submucous tissue, so that they are readily and speedily absorbed, the result being that they originate certain definite and specific diseases, varying with the nature of the material thus introduced into the system. In short, inoculation, as commonly practised or observed, is a mode by which the contagia of certain specific diseases are directly conveyed from one animal or individual to another; and some of these affections can only be thus transmitted, while others are capable of being communicated in other ways, but in this way most effectually and with the greatest degree of certainty. The most familiar examples of diseases thus transmitted are vac-

cinia, small-pox, syphilis, and hydrophobia. It need hardly be mentioned that vaccinia can only be conveyed by inoculation, and it is for the purpose of inducing this disease that the method is ordinarily intentionally practised, so that the terms *vaccination* and *inoculation* have come to be popularly regarded as synonymous (*see* VACCINATION). Under certain exceptional circumstances inoculation of the contagious matter of syphilis, small-pox, or anthrax is employed, with the deliberate purpose of originating these affections. The inoculation of the modified virus of rabies, according to Pasteur's method, is generally recognised as a most valuable plan of treatment, with the view of preventing the development of hydrophobia in those into whose system the poison has gained access, either owing to the bite of a rabid dog or in other ways (*see* HYDROPHOBIA). The employment in this way of tuberculin, for the supposed cure of phthisis and other forms of tubercular disease, according to Koch's method, or of tuberculinin, must also be mentioned here, but without comment (*see* PHTHISIS; and TUBERCLE). Inoculation has been much practised of late years in experimental pathology, by which the effects upon the system of the introduction of septic liquids have been demonstrated; as well as of certain solid morbid products. **See** CONTAGION; IMMUNITY; and INFECTION.

With regard to the modes by which inoculation is effected, it need only be said here that, when it is practised as an operation, the material is usually introduced into the subcutaneous or submucous tissue, by means of a lancet or other pointed instrument, or, in certain cases, with the aid of a hypodermic syringe. Sometimes the surface of the skin is merely scarified, so that the epidermis is more or less destroyed; or this is removed by some blistering agent, and the contagious substance then applied to the exposed cutis. Accidental inoculation may take place in connexion with any abrasion, wound, or ulcer on the skin or on a mucous surface; or by the bites of animals, as in the case of hydrophobia.

FREDERICK T. ROBERTS.

**INSANITY.**—**SYNON.**: Fr. *Folie*; Ger. *Geisteskrankheit*; *Geistesstörung*.

Insanity is popularly known as disorder of mind: as physicians, we know it to be disorder of the highest organs of the nervous system which unite in the performance of that function recognised and spoken of as mind. There can be no disorder of mind without disorder of brain: as physiologists and pathologists we have to study and treat the latter, and for this reason the legislature enacts that certificates of insanity shall be given by medical men, and by them alone, and that to their care shall be committed those who are insane.

As a preparation, then, for the study of

insane mind, the learner ought to acquire a knowledge of healthy mind—the healthy function of a healthy brain. He must know what are the structures which combine to make up that which we call the cerebrum, and contribute to its healthy working, and must trace the growth and development of this working from the earliest days of infancy to the time of adult life. He will perceive that the growth of mental function is as gradual as that of bodily power; that in some it may be more rapid than in others, like that of the body; that it may be arrested in its development, or stunted and deformed; and that it may by imperfection of the organs, as blindness or deafness, be impeded or stopped. In all particulars it will be found subject to the laws which regulate the growth of the body generally.

Before examining brain-function, it will be necessary to become acquainted with the various structures which form the cerebrum. The brain of man, and indeed of all vertebrates, is made up of nerve-cells and nerve-fibres; of a substance or *stroma* in which the cells are imbedded, called neuroglia; and of blood-vessels and lymphatics. The nerve-cells are gathered into convolutions or *centres*; and by means of the nerve-fibres communicate with the organs of special sense, with the sensory ganglia and spinal cord, with each other, and with the convolutions of the other hemisphere. Modern science is endeavouring to throw light upon the nature and uses of these convolutions. The seats of the highest intelligence, they at the same time appear to be centres of voluntary motion, and of the outward manifestation of intellectual action. See CONVOLUTIONS OF THE BRAIN AND CORTEX CEREBRI, Lesions of.

Concerning the physiology of the blood-supply of the brain there is still great doubt and controversy. Anatomically we know that from the internal carotid and vertebral arteries, combining in the circle of Willis, there pass to the brain-substance the anterior, middle, and posterior cerebral arteries. The terminations of these ramify in the pia mater, and thence send many small vessels to the grey matter, and fewer, but larger, ones to the white, the supply of blood to the former being much the more plentiful. That, however, which chiefly concerns the student of insanity is the vaso-motor system of nerves which controls and regulates the blood-supply. Modern research appears to render it more and more certain that the condition of insanity, at any rate in its early and acute stage, is mainly one of increased blood-supply. On what does this depend? To this question no precise answer can as yet be given. Investigations are still being made as to the nerves which dilate and contract the vessels; but with regard to this subject, and the so-called inhibition of nervous influence, much more must be ascertained before

the pathology of insanity can be definitely fixed. The lymphatics of the brain are also, according to some observers, largely concerned with the disturbance of mental function, if it should happen that by being blocked up or impeded they fail to carry off the waste products of the organ. They are contained in perivascular lymph-spaces, lying between the outer fibrous coat of the blood-vessels and a hyaline membrane or sheath of pia mater which separates them from the brain-substance.<sup>1</sup>

Even concerning the neuroglia, controversy exists. Is it merely connective tissue, or is it nerve-structure? Authorities lean to the former view, and the increase of it in diseased brains seems to point to a growth of lower structure at the expense of higher.

Passing to the functions of the brain, the phenomena comprised under this name are of two kinds. There are the various movements excited by the stimulation of the different brain-centres, on which the experiments of Dr. Ferrier have thrown new and interesting light. But these are not the phenomena of mind. The latter can only be studied by observation of a totally different character. For mind implies sensation, feeling, consciousness; and as we have no consciousness of any feelings but our own, it is evident that here objective observation is insufficient, however unwilling physiologists may be to admit it. Yet the subjective examination of ourselves is as inadequate by itself to explain the phenomena of diseased mind, as objective observation is to make known the nature of mind in general. We must use the subjective method as our key, and by means of it open up and interpret the phenomena of mind; we may then objectively examine the mental characteristics, the growth and development, the diseases and decay of mind in all human beings, children or adults, idiots or insane, and by tracing thus the effects of injuries and disease, we arrive at a knowledge of the differences between sound and unsound mind.

By observation of the movements brought about by nerve-function, we perceive that they follow the application of a stimulus, and that many actions take place in response to stimuli which we call reflex, instinctive, or automatic, before those begin which are the product of mind in its highest sense. For mind grows slowly and gradually. The first movements of the child, reflex or instinctive, are the result of stimulation of nerve-centres, but are only slightly, if at all, attended by consciousness. This becomes appreciable later, and with it the commencement of *memory* and *ideas*, as the feelings roused by stimulation are stored away in the mind, to be reproduced as some new stimulus is presented to them. The various brain-

<sup>1</sup> Dr. Batty Tuke. Morisonian Lectures. *Edinb. Med. Journ.*, Dec. 1874.

centres are in this manner stored with ideas, the result of experiences derived from without, and by means of the nerve-fibres they are united in groups till a most complex system is evolved. The sensations are developed into more and more complex feelings, till the highest emotions of civilised man are reached; and in a similar manner the intellectual processes grow from mere perceptions and cognitions to the highest trains of thought. Mind may be said to be made up of feelings and the relations between feelings, for the intellectual element of mind is the relational element, and it will be found that we cannot locate in two parts of the brain the emotions and the intellect, as some physiologists have proposed; for no kind of feeling, sensational or emotional, can be wholly free from the intellectual element; and on the other hand it very rarely happens that any act of cognition can be absolutely free from emotion.<sup>1</sup>

For the growth of healthy brain and mind all the conditions of physical health are necessary. If at birth there is inherited defect or accidental injury, or if at any subsequent period development is arrested or perverted, idiocy or imbecility is the result. There will be an imperfect recording of the experiences of life, an inability to learn, and a deficient power of bringing into relation one with another the various groups of nerve-centres which make up the brain, and are the seat of mind.

If, however, growth and development have proceeded normally, and normal mind is the result, what are the conditions of insanity, or disorder of mind? What are the conditions of healthy and unhealthy brain-function?

I. We know by experiment on nerves and nerve-centres, and by observation of the objective phenomena of motion, that a discharge of nerve-force or nervous fluid—call it what we will—is liberated when a centre is stimulated, and that this ramifies according to its quantity in various directions throughout the system. When there is health and vigour the supply will be large, and every channel both in the brain and body will be duly supplied. But in the healthiest nervous system there must in time be a cessation of this discharge, for the supply will be exhausted, and repair and replenishing must take place. And unless this is done, nerve-function will be impaired or cease. For the repair of the higher nerve-centres sleep is necessary, for only during sleep is the repair of the waste effected, and we commonly, nay, constantly, find that mental disturbance is preceded by loss of sleep.

In some cases want of sleep may depend on the actual time allowed for it being insufficient. Though not a very common cause of insanity, yet it sometimes is found in per-

sons who are very hard-worked, or who, by religious exercises and services, deprive themselves of sleep. In the great majority, however, want of sleep is the result of a pathological condition of the brain, or in connexion with disorder of the liver or other remote organ, conditions which by appropriate treatment the physician seeks to remedy as the first step towards the cure of the insanity.

Not only may there be a want of repair and replenishing of the nerve-force expended, but there may be also a defective generation and supply of it. Through anæmia, or exhaustion from acute diseases or long-continued illness, the nerve-centres fail to generate from the blood the power necessary for their due operation. The discharge liberated does not ramify throughout the nervous system, calling into action every part of the brain, and penetrating to every portion of the muscular structures; thus the failing supply is manifested in the gloom of *melancholia* and the slow and torpid movements which accompany it; and when it is reduced to a still lower ebb, there may be not enough to carry on even the semblance of mind: the patient presents the appearance of utter fatuity seen in *dementia*, whether primary or secondary, and either sits motionless and lost in the condition termed *melancholia cum stupore* [Fr. *mélancolie avec stupeur*; Ger. *Schwer-muth mit Stumpfsein*], or executes the purposeless and automatic movements of acute dementia [Fr. *Démence aiguë*].

The defect of nerve-force may bring about insanity in more than one way. A sudden strain or shock may make an unwonted demand upon the nervous supply. Incessant thought, especially of a painful kind, may use up the reserve of force; this is not renewed, and insanity is the result, the increased molecular discharge not being duly compensated by an increased supply from the nutritive sources. Secondly, the supply may fall so far below the standard, that it is not sufficient for the ordinary demands of everyday life, and so, without any mental cause, but simply from nerve-inanition, the stage of depression appears.

II. It may be, however, that there is not so much a defect of nerve-force, as an unstable condition of the cerebral centres, which is manifested by a rapid and continuous discharge. Such a discharge in a more sudden and violent form we are familiar with in the phenomena of epilepsy. In insanity it may vary from undue hilarity and excitement up to the incessant movements and vociferation of acute delirium; and, like epilepsy, it may terminate in exhaustion, coma, and death. The supply of nerve-force in such cases is often abundant. The patient goes without sleep for days and nights, and yet recovers. The higher brain-centres lose the power of control, owing to the violence of the dis-

<sup>1</sup> Herbert Spencer, *Principles of Psychology*, vol. i. p. 473.

charge, and the lower and more automatic centres thus liberated are stimulated to overaction, which is manifested in noisy and violent delusions, and so on to mere delirious incoherence. And, after the discharge has ceased, there may be a period of dementia and complete obliteration of mind, before the exhausted brain begins to recover. Persons who are in this condition of unstable nerve-equilibrium may be thrown off their balance by mental causes—by shock, or grief, or losses. Their brain may also be affected by disorder of the other organs of the body *sympathetically*. In all such patients there is a predisposition, often inherited, to nervous instability and rapid discharge, and such 'causes' as puberty, pregnancy, or childbirth bring about an explosion. Here, too, there is marked cerebral hyperæmia, producing an abnormal nutrition and corresponding abnormal function.

III. Disturbance or defect of mind may be caused by incomplete development of any part concerned in mental action, or by the presence of anything within the cranium which interferes with the healthy life of the various organs. There may be tumours of different kinds, or abscess. Insanity may also follow blows on the head; and deterioration of brain-tissue is frequently caused by the action of alcohol and other poisons. In these cases we have not a functional disturbance which may suddenly arise and as suddenly pass away, but a gradually developed organic lesion, which usually advances, producing serious and permanent results. The degeneration of the organ and its tissues through age will also produce corresponding results.

**SYMPTOMS.**—What are the symptoms of insanity, and how far do they correspond to the pathology of brain-disturbance as set forth in the above remarks?

The first symptom usually noticed in a person becoming insane is an alteration in his *emotional* condition. Either he is more quiet and dull than usual, or, on the contrary, more restless, irritable, excitable, or hilarious. The dullness may vary from a mere disposition to sit still, and neglect his work or amusements, to actual gloom and despondency. The restlessness and excitement may also vary in degree, and be accompanied by gaiety or outbursts of anger and of violence. The change may be more or less concealed, according as the patient can or cannot control himself. Those of his own family or people about him may notice it long before others, and this stage will vary in duration, often lasting some time before delusions or other marked symptoms are discoverable.

The *acts* of the patient will correspond to his altered feelings. His relations towards the outer world will be changed. In his torpor or gloom he will look on everything despondingly, will be unable to perform his duties, will not care for amusements, will sit

at home inactive, or take up morbid fancies about his health. On the contrary, he will act rashly in matters of business, embark in foolish speculations, take no heed of time or appointments, spend money recklessly, indulge in debauchery or frivolous pursuits, show causeless anger to those about him, or exhibit silly and childish hilarity when matters of grave moment are pending.

Here we have a *slight insanity*. The higher brain-centres are but slightly disturbed, and can still exercise a considerable amount of control over the lower and less specialised centres. It may be difficult to say that any one act or feeling is of itself indicative of insanity, but the patient is a changed man, and the term *moral insanity* is specially applicable to this condition.

Such an insanity may remain and be permanent; more frequently, however, it either passes off, the patient recovering, or it advances in one or other of the directions already indicated. The gloom will increase; corresponding delusions will present themselves, with appropriate acts and conduct; and the patient will drift into *melancholia*. Or he will become more and more hilarious, angry, excited, or suspicious, and a state will arise to which for want of a better we give the name of *mania*. Though delusions are not usually found in the stage of alteration, they are seldom absent in the second and more advanced period, and generally correspond to the feeling of the sufferer, so much so that his appearance and humour, so to speak, frequently enable us, without previous information, to arrive at the delusions under which he is labouring. This stage is often called *intellectual insanity*, or insanity with delusions. Patients labouring under one or other of these forms vary in the degree of insanity, from a condition in which they are able to talk coherently on many topics to one of complete incoherence or delirium. To the former the name of *partial insanity* is often applied.

Another condition is that of fatuity or childishness in various degrees. It may come on rapidly, the patient passing at once into this state, which is then called *acute* or *primary dementia*; or it may be the result of years of insanity or brain-disease, and is termed *chronic* or *secondary dementia*. In such people we find loss of memory, inability to revive the relations of feelings and ideas—not emotional disturbance, but rather an absence of all emotion.

As regards the bodily symptoms, almost every variety of insanity is ushered in by sleeplessness; sleep being deficient or altogether absent, perhaps for days, according to the acuteness of the attack. This indicates a disturbance of the brain-circulation, which is also shown by heat and pain of head, injection of the conjunctivæ, and throbbing of the carotids. There may be excessive vascular

action even when the insanity is the reverse of what is sometimes termed 'sthenic,' and the general condition of the patient is one of anæmia rather than hyperæmia.

It has been said that the mental symptoms of insanity are accidental, that they do not indicate the pathological conditions, but depend on the state of the body, and that no classification of the forms of the disorder ought to be based on them. But it is perfectly certain that the brain of a man suffering from melancholia differs altogether from that of one in acute mania. In the former there is a scanty generation of nerve-force, which is insufficient to reach the remote channels and plexuses of the brain. There is not a general incoherence and confusion of ideas, and the patient can converse rationally on many topics. His feelings are those of pain and not of pleasure, because pains in general are more intense than pleasures in general,<sup>1</sup> and the former are called into consciousness by the feeble nerve-currents, while the latter are not. In what is termed mania we may have every shade of feeling displayed, from gloomy and suspicious irascibility to great hilarity. Feelings of anger and resentment are called up without the controlling power of reflection and judgment, such as would prevail were the whole brain at work, and the higher faculties and feelings co-operating and in relation with the lower. This, again, may be due to a want of nerve-force, or more frequently to an undue expenditure of it, as may be seen in the irritability of an over-tired child. What we may call the more automatic and less complex feelings of fear and self-love are evoked, while there is not force and pressure enough to supply the rarer and more remote qualities of comparison and reasoning, which require the combination and union of the highest portions of the brain. If there is an increased and accelerated blood-flow, great hilarity and self-satisfaction may be the result, yet with perfect incoherence of ideas, owing to all the relations of the brain-centres being interrupted by the tumultuous or impeded circulation, and the excessive nervous discharge. And in dementia the very opposite may be witnessed. The supply of force is reduced so low that there is an absence of all ideas; memory, at any rate of recent events, is lost, and only the well-marked occurrences of earlier life are recalled.

That the mental symptoms are to be by the pathologist altogether disregarded, and that they are of no assistance in the appreciation of the pathological condition, is a most extraordinary assertion. These symptoms do not depend on the state of the body generally, but on that of the brain, and as accurately indicate the condition of the latter as the breathing indicates that of a lung.

<sup>1</sup> See Herbert Spencer, *Principles of Psychology*, vol. i. p. 602.

The brain in insanity is the *pars affecta*, the unsoundness is the symptom; and when we see in the same individual at one time mania and at another melancholia, we may be sure that the condition of brain at the one period is not the same as at the other, though originally one cause may have lighted up the malady.

DIAGNOSIS.—Accuracy of diagnosis is specially important in insanity, owing to the legal and social results which flow from it, and because the restrictions on liberty which may be necessary for proper treatment cannot be resorted to until a diagnosis has been conclusively established. The direction of the investigation will be different, according as the condition may be the result either of incomplete development, or of disablement or perversion by disease. Where *imbecility* is in question, it may often be necessary to take several opportunities of examining the patient. This condition always implies intellectual defect, though great moral depravity is often the predominant symptom. It is therefore necessary to ascertain whether any or what kind of occupation has been attempted, and what amount of incapacity has been shown, and whether the individual has proved capable of profiting by such education as he has received. Adults in ordinary circumstances ought at least to be able to read, write, and count. The decision rests on whether such capacity has been shown as is required in the ordinary conduct of life. To form a correct opinion in cases of *acquired insanity* is often a very difficult matter. The relatives of the patient often obstruct rather than aid the inquiry. They are generally divided in opinion, and this may be partly an aid and partly an obstruction; but great care is required to avoid taking part at first with either side. Before seeing the patient it is proper to make what inquiry is possible into the hereditary history of the family, and the nature of the diseases or injuries from which the patient may have suffered, including any previous attacks of insanity. The ordinary habits, disposition, tastes, and occupations must be ascertained, and also the present habits, disposition, tastes, occupations, amount of sleep, and general bodily health. The mental symptoms, such as suspicions, delusions, or loss of memory, which have suggested the allegation of insanity, must also be inquired into; and special attention must be paid to any indication of the change of conduct or disposition which is so characteristic of the advent of mental disease. A reference to the articles descriptive of the forms of insanity will show the importance of all these points. In the whole investigation care must be taken to avoid accepting mere *ex parte* statements, and as much information as possible should be obtained by the inspection of letters or other documents written by the patient. Sometimes the con-

duct of the patient renders it difficult to obtain an interview, and care must be taken not to overstep legal limits in the attempt. If the physician has to see the patient in the presence of other persons, it is a necessary precaution to make certain that he clearly understands whom he is to examine. In obtaining the interview a certain amount of stratagem may sometimes be resorted to; but it is best that the physician should be introduced by a friend in his true character as a doctor, and on no account should any false statement be made. As a mere matter of expediency it will be found that any deviation from this produces more evil than good. If the interview takes place in the patient's home, valuable information may frequently be obtained by observation of the order or disorder which prevails. The condition of the furniture, the state of the patient's clothing, his manner towards the rest of the household and their bearing towards the patient, ought to be noted. The patient's physiognomy, the condition of the pupils, and any gesticulations or convulsive or tremulous movements, must be observed. In conversation it is well to get as soon as possible to the subject of the patient's health, as it relieves many necessary questions of their offensive character. Eccentric ideas ought not to be combated more than may aid in making the patient disclose them fully; and everything tending to show the presence or absence of delusions, irrational suspicions, or loss of memory, ought to be elicited. Many other points will probably be suggested by the course of each inquiry; and the importance to be attached to them, as well as to those just mentioned, must be decided by a careful study of how far they are included or excluded by the known symptoms of any of the various forms of insanity.

**ÆTIOLOGY.**—We now pass to the causes of insanity, which are usually spoken of as *predisposing* or *exciting*; in many cases both combine in the causation of the disorder.

The great *predisposing* cause is an inherited disposition to neurotic disorder—one difficult to estimate and, indeed, to discover, owing to the care with which it is concealed, but the importance of which cannot be questioned. It has been adverted to in the second section of the pathological conditions, and will again be noticed. See INSANITY, Varieties of.

When we speak of such predisposing causes as *sex*, *age*, and *condition of life*, it is evident that they can only be called causes in the sense of being concurrent conditions, in some of which a man or woman is more likely to become insane than in others.

What are the relations of *sex* to insanity? Referring to statistical tables, we find that in the Report of the English Commissioners in Lunacy for the year 1892 there were under treatment in asylums at the end of the year 1891, 29,293 males and 34,698 females.

There were admitted during the year (not reckoning re-admissions or transfers) 8,233 males and 8,690 females, while in the same period there died 3,560 males and 2,924 females. The preponderating number of females under treatment is probably due to the fact that the mortality among them is far less than among males, and consequently they accumulate in asylums. The difference in the number of the two sexes who become insane is probably not material, but it seems that the males are the larger number, or, looking at the difference in the mortality, they would fall below the number of the females further than they do.

With regard to *age*, we find that the tendency to insanity increases with the development of brain and mind. In the first decade of life it is rare. In the second, which includes the period of puberty, it is more common, but not so much so as in the next. The period between 25 and 40 years is that in which the greatest number of cases arise, and is that of the highest development and working power. After this the number declines in each decade, as before it rose. With the age the character of the insanity varies. In youth it is displayed in violent and paroxysmal mania, sometimes in acute dementia, and cataleptoid states. There is great motor disturbance, and emotional rather than intellectual aberration. Rarely are the young melancholic. In the prime of life there is active mania with delusions and intellectual insanity, and at this period we meet with the most acute forms. Later, melancholia prevails; while in old age weakness of mind, passing into fatuity and second childhood, indicates the general decay of the brain and nervous system.

As to the *condition of life* in which insanity is most frequently found, there is not much to be said. That it occurs more frequently in civilised than in barbarous countries may be assumed without recourse to figures, because in the former mind and brain are more complex, and therefore more prone to disorder. It is, however, the failures and vices of civilisation that bring about the great mass of insanity. And of these the chief is *poverty*, with all its attendant physical evils of insufficient food and wretched dwellings, and moral evils of anxiety and degradation. Next to poverty, and closely bound up with it, is *drinking*, which among the working classes plays a fearful part in the causation of the disease. Among the predisposing causes of insanity included by the International Congress of Alienist Physicians in 1867 are, besides those already enumerated, consanguinity; great difference in the age of the parents; influence of the soil and surroundings; convulsions or emotions of the mother during gestation; epilepsy and other nervous affections; pregnancy, lactation, menstrual periods, critical age, puberty, venereal excess,

or onanism. We may add to these, damage received at birth owing to difficult parturition.

The *exciting* causes of insanity are usually divided into *moral* and *physical*. Among the former we may reckon domestic trouble and anxiety, mental shock, over-work, religious excitement, political excitement and war, and disappointment. Concerning such little need be said. They may vary in duration, some quickly bringing about insanity, others persisting for years before that result is reached. For the most part, those who are affected by such causes are already predisposed by hereditary taint, by a neurotic temperament, or by being at one or other of the critical periods of life.

Among the *physical* exciting causes are some which are both exciting and predisposing—for example, intemperance and epilepsy. These may be the immediate precursors of an attack; as well as agents causing a predisposition to the disorder, by being repeated through a series of years. Other physical causes are parturition, menorrhagia, amenorrhœa, and various other ovarian and uterine ailments; acute febrile diseases and chronic illness, producing exhaustion; constitutional diseases, as gout, ague, or syphilis; disease of heart and vessels; exposure to great heat or cold; lead and other poisons; anæmia; blows on the head, and organic affections of the bones of the cranium, or the various parts contained therein.

**CLASSIFICATION.**—Almost every writer on insanity has suggested a special classification of its forms, and the majority have founded their suggestions either on the ætiology or on the symptomatology of the disease. Of those based on symptoms no classification is simpler than Griesinger's: (1) states of mental *depression*; (2) states of mental *exaltation*; (3) states of mental *weakness*. He placed general paralysis and epilepsy apart as mere complications of insanity. His groups, therefore, correspond broadly with the old divisions of *Melancholia*, *Mania*, and *Dementia*. The ætiological classification most widely known is that of Morel. He divides the forms into six: (1) *Hereditary* insanity, including congenital nervous temperament, moral and impulsive insanity, imbecility, and idiocy; (2) *Toxic* insanity, including conditions caused by insufficient or injurious food, poisons, or noxious air or water; (3) *Hysterical*, *epileptic*, and *hypochondriacal* insanity; (4) *Idiopathic* insanity, dependent on disease of the brain or its membranes; (5) *Sympathetic* insanity; and (6) *Dementia*, or the condition of terminative enfeeblement. For any systematic study of the subject, it is obvious that some symptomatological grouping, based on the characters of the mental manifestations, must be necessary. It has, however, been maintained that it is impossible, either on this or on a purely ætiological basis, to found

groups which have more than an artificial relationship to one another. And there is some truth in this criticism. But, though the ties which bind the psychological groups together may be in a sense regarded as artificial, it is found in practice that the associated conditions exhibit a considerable amount of intimate natural connexion.

At the International Congress of Alienists in 1867 the following classification was laid down, intended to combine the ætiological and symptomatological methods: 1. *Simple Insanity*, comprehending mania, melancholia, monomania, circular insanity, moral insanity, and the dementia following these forms. 2. *Epileptic Insanity*. 3. *Paralytic Insanity*. 4. *Senile Dementia*. 5. *Organic Dementia*. 6. *Idiocy*. 7. *Cretinism*.

The first class, it will be observed, comprises all the varieties which may be regarded as merely functional; the others are mostly associated with permanent structural lesions.

No classification which has been proposed can be regarded as altogether satisfactory. This is partly owing to the fact that the true nature and limits of insanity itself have been very imperfectly recognised. The essence of the condition is, of course, the manifestation of disease through some deviation from the healthy standard of mental action. It is a condition of mental unhealth analogous to bodily unhealth. But we must not allow ourselves to imagine that there is a class of morbid mental manifestations which are independent of the condition of the physical frame. The truth is, that there is no pathological condition of the individual in which both mind and body are not affected; but in some diseases the mental symptoms come into prominence, in others the physical. The notion, not yet altogether exploded, that there is something in insanity altogether distinct from bodily disease, arose from the belief so long prevalent that mental action is independent of physical conditions, and from the fact that the study of insanity has been and still is too much dissociated from the study of the rest of cerebral pathology. Acting upon the broader and truer views, attempts have been made by Schroeder van der Kolk, and others, to introduce a more natural system of classification. Such attempts have proceeded on the recognition of all mental symptoms as phenomena whose nature cannot be ascertained without a full consideration of the physical symptoms of disease by which they may be accompanied. Dr. Skae proposed a classification based on the belief that every mental disorder bears a relation to some bodily disease—acute or chronic—analogous to what the delirium of fever does to the fever in whose course it is manifested. The detailed list which he offered was, however, admittedly imperfect; and it is likely that any satisfactory classification on this principle will only be arrived at after a much closer

study of the mental symptoms of disease than has yet been given to them. But Skae deserves the credit of having given the most powerful impulse to the purely medical, as opposed to the metaphysical, mode of studying insanity. In furtherance of this, some progress has been made by Clouston, Batty Tuke, and others, who have published careful monographs of some of the more prominent forms. If by such means we can group together conditions which are similar, not only in their mental but also in their physical characters, we obtain units which may ultimately contribute to the building up of a more perfect system, and can never be altogether disregarded by classifiers in future. Most of the attempts which have been made to describe such groups must at present be regarded rather as valuable suggestions than as well-established clinical and pathological species. For the purposes of this work it has been considered best to describe, in the present article, the various phases of insanity which have been regarded as of special importance, without regard to the principles which underlie their conception; and afterwards to discuss the various well-marked forms of the disease under their several heads, alphabetically arranged.

**PROGNOSIS.**—The general prognosis of insanity will depend (1) on the duration of the existing disorder. Perhaps the best established fact of all is, that the chances of recovery diminish in direct proportion to the duration of the malady, and that it is consequently of the utmost importance to place a patient early under adequate and appropriate treatment. If a twelvemonth elapses without appreciable improvement, the chances are decidedly unfavourable. If delusions or hallucinations remain fixed and unchanged at the end of a year, especially if there be hallucinations of hearing, the prognosis is bad. The chief exception is where there is marked melancholia. Patients will recover from this after long periods; whereas such recoveries are seldom found in insanity when depression is absent. (2) When the cause of the insanity has been of long duration, the prognosis is less favourable than when it is a passing or accidental cause. (3) Is the prognosis unfavourable in hereditary insanity? So much of the so-called simple insanity is hereditary, that we must admit that recoveries from it are not infrequent, for it is from this simple insanity that recoveries chiefly take place. Hereditary insanity is developed by very slight exciting causes, and thus the prognosis is often favourable, and recovery takes place; but relapse is to be feared, and the prognosis in a second or third attack is not nearly so good. In this hereditary insanity, too, we frequently meet with the cases of recurring and 'circular' insanity, the progress of which is most unfavourable. Both Ray and

Griesinger have remarked that the prognosis in hereditary insanity is favourable only where the individual has previously been of normal mind. When he has always been eccentric or semi-insane, and undoubted insanity at last manifests itself, the prognosis is very bad. (4) The more acute the symptoms, the greater the cerebral disturbance and insomnia, the more favourable is the prognosis, if the case is recent. Conversely, the prognosis is bad when there is little bodily disturbance, where sleep is present, the appetite normal, and the secretions unaffected, especially if persistent delusions or an entire moral change are found. (5) As all deviation from the ordinary mental state and disposition is indicative of insanity, so any return to it is a favourable sign, however trifling the circumstances may be. (6) Improvement, however slow, is a good sign if it be progressive. So long as this goes on, recovery may take place; but many patients improve up to a certain point, and then go no farther. (7) The age of the patient must be considered. Young people recover in greater numbers than those advanced in life. The latter recover if their insanity be melancholia; but, if it be mania, with hallucinations and delusions, and obscene conduct and ideas, recovery is rare, especially if the memory is impaired, and signs of approaching dementia are present. (8) All periodicity in the disease, such as exacerbations and remissions on alternate days, is unfavourable.

**TREATMENT.**—Only a few general remarks on treatment need be offered here. Our objects should be to restore to health the disordered brain, to cause the incessant waste to cease, to promote a storing and not an expenditure of nerve-force. The brain must be nourished by healthy blood. The quantity of the latter when in defect must be increased; when its quality is in fault it must be improved; when the blood-flow is in excess it must be checked; while all causes of disturbance reacting upon the brain from other organs of the body must be removed.

It is not to be forgotten that powerful effects are produced throughout the nervous system, both in the lower and higher centres, by what has been termed 'inhibition.' By the diversion of nervous action from one channel to another, considerable influence may be exercised.<sup>1</sup> Emotional excitement may be diverted into motor or intellectual channels; or, by other emotional stimuli, may be counteracted or arrested. Intellectual or ideational troubles may be diverted by emotional longings, or by counteracting intellectual pursuits. And for all this certain adjuncts are necessary. Painful emotional distress, with the idea of impending ruin, is perpetually fostered by the sight of the loved

<sup>1</sup> Dr. Lauder Brunton, On Inhibition. *West Riding Asyl. Med. Reports*, iv. 179.

faces of wife and children : the patient must therefore be removed from them. Outbursts of anger are constantly directed against those most familiar, and delusions correspond. These must be abolished by residence among strangers. Great as is the need of medicinal treatment in many cases, it is not so universally demanded as is the removal of the sufferer to fresh surroundings. We try again and again, in apparently the most promising cases, to effect a cure at home, and we fail. The necessity for early treatment in insanity is dwelt upon by every writer ; and the treatment, when insanity is once fairly established, only begins after the patient is removed.

The first questions to be solved are how the removal is to be effected ; and to what place the patient is to be removed. In the majority of cases, especially in the case of the poor, no doubt can arise—an asylum is the only place open to them, because the friends being poor cannot afford any other plan of treatment, and are compelled to have recourse to the public asylums of the land. An asylum is necessary for many because the patient is dangerous to himself or others, or would incessantly struggle to escape from a less guarded dwelling. But there are some patients who may be cured out of an asylum. Some recover from acute but transitory attacks of delirious mania very rapidly, much as do the sufferers from delirium tremens ; and, if measures of safety can be taken, we may watch such for a few days and perchance they may recover without removal. Many persons at the very outset of insanity may be cured by removal and judicious treatment, if their friends will only open their eyes and acknowledge the threatening evil, and not wait, as they so often do, till compelled by circumstances to interfere. Such patients must not be sent abroad or out of reach, must not go alone, or without able or skilled companions. They may go from place to place, or to a friend's or medical man's house. Fresh scenes and faces, and the cessation of work or worry, will often effect a cure. But they must be people able to walk out in public thoroughfares, and to live in houses under ordinary precautions. Where they cannot walk in public, and cannot live in a house without its being converted into a prison, they ought to go to an asylum, where there are grounds for exercise, and where facilities for escape are not always suggesting attempts. Patients' friends constantly make a mistake : they keep the patient out of an asylum at the time asylum treatment would cure him, and send him there when all hope of cure is over, and when, as a chronic lunatic, he could be just as well taken care of outside one.

With regard to medicinal treatment little need here be said. The medicines appropriate to each form of insanity will be spoken

of under the different heads. An important group consists of sedatives and hypnotics ; and to those formerly in use, as the bromides, opium, chloral, and Indian hemp, there have been added various new drugs, as hyoscyne, hyoscyamine, paraldehyde, and sulphonal—the last two the most valuable of all, in the writer's experience. Speaking generally, it may be said that in the depressed and melancholic cases the preparations of opium are of the greatest service, not only for procuring sleep, but for allaying the terrible feeling of gloom which prevails in the daytime, and especially on first waking from sleep. The efficacy of opium here is not that of an hypnotic only. It acts as a stimulant during the waking hours, and for this it has no rival. Next to it in melancholia paraldehyde deserves to be placed. In the opposite class, the excited or maniacal, opium rarely does good and may do harm, preventing instead of producing sleep. In place of it the bromides with or without chloral may be given, or hypodermic injections of hydrobromate of hyoscyne. The nauseous taste of paraldehyde makes it difficult to administer ; but sulphonal is tasteless, and, if mixed with food, will not be discovered. Besides these, the chief medicines required are tonics, the preparations of iron being the most valuable. Constipation must be carefully prevented.

Good and abundant food is an essential in the treatment of the insane. Stimulants are required in many cases, particularly the depressed and anæmic forms ; but in the opposite, though often useful, they in some cases produce or increase excitement, especially in the early stage. With the food tonics should be given ; and those best suited are, in the writer's experience, the mineral rather than the vegetable, and chief of all the preparations of iron and arsenic.

G. F. BLANDFORD.

**INSANITY, Morbid Anatomy of.**  
**METHODS OF EXAMINATION.**—In order to examine any brain specimen thoroughly it is advisable to employ two methods : (1) freezing, cutting, and staining fresh portions with aniline black ; and (2) hardening for some weeks in one or other of the well-known solutions—such as Müller's. The first is the essential process, the second is mainly useful as confirmatory of its results. The teasing-out method is valuable for the observation of cell-structure. Full directions on this subject will be found in Dr. Bevan Lewis's *Manual of Examination of Human Brain*. By the freezing method sections can be submitted to the microscope in two hours, unchanged by the action of reagents, and, if necessary, can be at once photographed ; by the hardening process larger areas can be examined, certain lesions are made more prominent than by freezing, and sections can be permanently preserved.

The morbid anatomy of the different structures will now be systematically reviewed.

1. **Calvaria.**—The calvaria is often found thickened, dense, heavy, or thinned. In recent cases of syphilitic disease and in general paralytics, it has been found inflamed, both tables thickened, the cancellæ enlarged, and the lacunæ full of granulation tissue. The dense skull is generally found in old-standing cases of chronic terminative dementia, and in epileptics. The inner table may present local indurations.

2. **Membranes.**—In treating of the membranes we would premise that in our opinion it is anatomically correct to speak of only two, the *dura* and the *pia mater*. No doubt exists as to the minute anatomy of the former; it is a thick, inelastic, fibrous membrane, containing vessels and lymphatics, and lined on its inner surface by flattened epithelium. The *pia mater* is a double membrane, the outer layer of which (the so-called *arachnoid*) covers the brain without folds, and bridges the sulci; the inner is applied to the surface of the convolutions, and dips into all the fissures and sulci. Between these two layers a rich plexus of blood-vessels and lymphatic spaces ramifies, the layers being intimately connected by large numbers of trabeculæ over the free surface of the convolutions, but being widely separated in the sulci and fissures, and in the basal cisterns. Thus we have a visceral and a parietal layer of the *pia mater*, neither of which is in itself, as is often stated, a vascular membrane. For the purposes of differentiation the former will be spoken of as the *arachno-pia*, the latter as the *visceral pia*. The space between the *arachno-pia* and the *dura* is now termed the *sub-dural* space (formerly the *arachnoid cavity*), and the spaces between the *arachno-pia* and the *visceral pia* will be termed the *pia-matral* spaces instead of the *sub-arachnoid* cavity. Thus from without inwards we have—

(a) *Dura mater*; (b) *Sub-dural space*; (c) *Arachno-pia*; (d) *Pia-matral spaces*; (e) *Visceral pia*; and (f) *Brain-matter*.

#### **Lymphatic System of the Encephale.**

In order to have a clear conception of the results of morbid action in the membranes and brain-matter, the lymphatic system of the encephale must be studied. The *visceral pia*, whether on the surface of the convolutions or in the sulci, sends with each vessel a prolongation inwards surrounding it, and forming the *hyaline sheath* or *perivascular canal*, which can be traced to the ultimate ramifications. Every cell of the grey matter is encapsulated by a fine membrane, first described by Obersteiner, which is a 'spur-like' prolongation of the hyaline sheath. Starting from the cell, we have thus a capsule connected by a fine channel with the hyaline sheath of the vessel, which debouches into the *pia-matral spaces* (the so-called *sub-arachnoid space*), and these

communicate directly with the basal cisterns—the cerebello-medullary, the inter-peduncular, that in front of the optic chiasma, the Sylvian, &c., with the ventricles, and with the wide *pia-matral space* in the spinal canal. The *pia-matral spaces* communicate with the *sub-dural space* by means of the *Pacchionian villi*. These organs, which are prolongations of the *arachno-pia*, protrude not only into the lymphatic spaces of the *dura*, but also into the longitudinal sinus, being separated in both instances by a fine membrane from the fluid contained in each class of vessel. This constitutes the main lymphatic system of the encephale, which, finding vent through all the foramina of the skull and vertebral column, maintains direct communication with the extra-skeletal lymphatics. But, besides this main system, there are reasons, chiefly founded on the observations of Dr. Bevan Lewis, for believing in the existence of what he calls a *lymph-connective system*. Scattered throughout the grey and white matter, but more specially arranged along the vessels, and in the outer layer of grey matter of the convolutions, are found cellular structures of two kinds: the one small, consisting of a readily stained nucleus with slight protoplasmic surroundings; the other large, consisting of a nucleus not easily stained in health, and a considerable protoplasmic body, sending out poles or processes of two kinds. One set of processes is numerous and is distributed throughout the neuroglia between vessels, cells, and fibres; the second is a single process, and runs directly to the perivascular lymphatic. Whether this is or is not a tube has not been yet determined, but the strong probability is that it is not canalicular. These spider cells, which were first described by Deiters, and go by his name, are held by Dr. Bevan Lewis to be the *scavenger cells* of the brain. He maintains his theory as to their function mainly on the ground that, whenever there is any special demand for the removal of the products of disease, as in inflammation, these bodies increase in size, their processes become distinct, which they are not in health, and present all the appearances of increased activity. Of this fact there can be no doubt, and there are many pathological analogies, e.g. the behaviour of the connective-tissue corpuscles of the lung in connexion with pigment, which give a general support to the theory. His has described an *epi-cerebral lymph-space* between the *visceral pia* and the brain-substance. Its existence is open to grave doubt. It is not demonstrable in healthy animals, and difficulty arises in endeavouring to explain how the fluid it is said to contain can reach the lymph-system in the *pia-matral spaces*.

(a) *Dura mater.*—The most frequent lesion of the *dura mater* is a morbid degree of adhesion to the skull; this is specially marked in general paralytics, and is the result of sub-

acute inflammatory processes commencing in the subjacent tissues. It has occasionally, though rarely, been found thickened. Osteophytes have been noted in puerperal cases; bony plates have been recorded, and the falx has been found ossified.

(b) *Sub-dural fluid*.—The sub-dural fluid may be considerably increased, in which case the pia as a whole is much affected. Adhesions between the dura and the arachno-pia are rare.

(c) *Arachno-pia*.—*Opacity* of the arachno-pia is generally found to some extent in sane subjects after middle age, and is specially well marked in habitual drunkards. Considering that subjacent to it lie the principal lymph-conduits of a very active organ, it is not to be wondered at that some evidence should be left of the products of waste in the meshes of this delicate membrane even in healthy subjects. In many forms of insanity, e.g. acute idiopathic mania and melancholia, and in general paralysis, opacity is noticeable at a pretty early stage. In three cases of acute idiopathic melancholia, the subjects of which committed suicide within six weeks of the incidence of the condition, the writer found well-marked pearly or milky streaks, not implicating the whole layer, but showing themselves along the course of the veins; thus a purple and congested vein showed a narrow white band on each side of its exposed surface. This early indication is caused by transudation through the walls of the veins, the blood in whose main trunks, debouching into the sinus at an angle contrary to its current, is, particularly under conditions of disease, liable to be retarded. In 60 per cent. of insane subjects the arachno-pia shows indications of diseased action. It may be gelatinous-looking, due to enlargement and distension of the pia-matral lymph-spaces by serous fluid; thick and opaque in consequence of the infiltration of exudation; and we occasionally find the parietal widely separated from the visceral layer, the trabeculæ having been stretched or destroyed. These appearances are specially well marked in cases which have been the subjects of prolonged attacks of excitement, whether in the form of mania or melancholia, and who have lapsed into terminative dementia. It may be noted here that, although mania is manifested by exaltation and melancholia by depression of feeling, both are expressions of excitement. The writer has met with instances where this membrane was not more affected than it generally is in sane persons, but these were for the most part cases in which the insanity had been of a true hereditary type, evidenced by simple delusional conditions uncomplicated by excitement.

Between the dura and the arachno-pia, so-called 'arachnoid cysts' or 'pachymeningitic' *false membranes* are often found. Both terms are erroneous: they are not

cysts, they are not formed from any membrane, nor are they the result of inflammation. They are entirely due to extravasations of blood, probably produced during periods of cerebral excitement, and proceeding from the dammed-up vessels of the pia (see MENINGES, CEREBRAL, Diseases of). Such false membranes may be very extensive, occupying the whole superior and lateral aspects of a hemisphere, or they may be confined to a limited area; they may be tough and thick, or very thin, and so delicate as to render it difficult to lift them whole from the pia; or the so-called pachymeningitis may be only evidenced by staining of the dura mater by blood-pigment. Thin blood-clots have been occasionally found in the cavity of the pia, and between the visceral pia and the surface of the convolutions; but these were never so thoroughly organised as the false membranes between the arachno-pia and the dura. In the substance of the brain itself the writer has found in two cases very small clots about a line below the surface. In both these instances the cranium had been submitted to traumatic injury; in one of them vacuoles were shown containing blood-pigment.

The amount of fluid in the cavity of the pia is often greatly increased; we may measure as much as 12 ounces, but the more frequent quantity is from 2 to 6 ounces. Such large accumulations of fluid are generally found in cases where there has been brain-wasting, and is therefore compensatory. But it must be distinctly understood that, in recent and acute cases, fluid, whether in the cavity of the pia or in the sub-dural space, is the direct result of inflammatory processes and is in no sense compensatory; on the contrary, it may produce brain-atrophy by pressure. This is particularly well marked in general paralysis, in rapidly fatal cases of which condition there may exist considerable atrophy of one hemisphere, with extensive effusion into the sub-dural and pia-matral spaces. General paralysis is the result of sub-acute inflammation, either commencing in the pia and extending gradually inwards along the course of arteries and veins, or affecting the two regions synchronously. Under these circumstances the production of exudation is a necessary consequence. But in the case of idiopathic insanity the original seat of irritation is the grey matter. By idiopathic insanity it is intended to indicate the insanity which follows on the action of so-called 'moral' causes: grief, worry, anxiety, prolonged study, &c.—any emotion or mental action productive of prolonged excitation of the cells.

It is a well-ascertained fact that mental action is accompanied by functional *hyperæmia*, which is of course a necessary condition for functional activity. Functional hyperæmia is produced either by inhibition of the vaso-motor centre in the medulla, or

by stimulation of the vaso-dilator centres, or possibly by their combined action. The expression of Crichton Browne may be accepted as a postulate: 'the amount of blood-supply to the brain and its parts is determined, not by vascular domination, but by the functional activity of the nervous tissues.' Functional hyperæmia is apt to lapse into pathological hyperæmia if the excitation or emotion is unduly prolonged, and develops more or less rapidly what is equivalent to the first stage of inflammation. The first microscopic indication of this condition is the presence of leucocytes, together with a very fine granular material, the broken-down nuclei of leucocytes and epithelium, and of small yellow masses of blood-pigment between the walls of the vessels and the hyaline sheath. The writer has noticed these appearances within six weeks of the incidence of acute idiopathic melancholia. The perivascular lymphatics become blocked by these morbid materials; bulge out unduly; and are distinctly dilated by exudation fluid, which cannot find its normal course of exit. Under these circumstances the outflow of lymph and of effete matter from the cell-capsule becomes impossible, or is very much impaired; fluid accumulates within it, and the cell suffers. In health the *reaction of the cerebro-spinal fluid* is alkaline; under conditions of disease it becomes acid, in consequence of the accumulation of the acid products of cell-waste. Thus the cell lies bathed in a poisonous fluid, and its functional activity becomes necessarily impaired or perverted. Roy holds that acidity of the fluid is sufficient in itself to produce and maintain hyperæmia. A certain proportion of this exudation finds its way to the pia-matral spaces by the sheaths, which become more or less seriously affected; but another portion of it becomes diffused throughout the substance of the convolutions, interfering with the normal relations of fibre and connective tissue. The brain becomes more or less oedematous. In the case of idiopathic insanity the inflammatory process extends outwards, and the pia mater becomes implicated. Following implication of its lymphatic apparatus we have *accumulation of fluid* in its cavities, which may extend to the sub-dural space. In the comparatively rare condition of inflammatory adhesion between the arachno-pia and dura, local accumulations occur on the superior aspect in both cavities. In whatever manner local accumulations occur, corresponding atrophy of the subjacent brain-substance is the result produced by pressure. The diffusion of the fluid products of inflammation commencing in the pia or in the grey matter is circumscribed inferiorly by the resistance offered by the connective tissue; and, as in an abscess, local inflammatory tension may exist in a hemisphere without implicating remote areas. These facts were fully illustrated by observations made in two cases which, during

1889, were trephined for the relief of symptoms of general paralysis. (See *Brit. Med. Journ.*, Jan. 4, 1890.) In one case the dura mater bulged and filled the hole after removal of the disc on the right side; on the left there was little or no bulging. The patient died seven months after the operation, from pneumonia; on examination, the right hemisphere was found much atrophied, with a large quantity of pia-matral and sub-dural effusion. The bulging points necessarily to pressure, as any merely compensatory fluid could exercise no such power.

*General softening* of the brain is due to cedema.

**Blood-Vessels.**—The blood-vessels, in addition to the changes already mentioned as occurring around them, are found tortuous, dilated, thickened, occluded, or aneurysmal. It is of importance to bear in mind that many of the arterioles of the cortex are terminal; that is, they do not anastomose, and are distributed to limited areas (Duret). No conclusion can be arrived at with safety as to the degree of vascularity by the appearance of *puncta vasculosa*, as the number and distribution of these spots are dependent on various accidental circumstances. Tortuosity is rarely met with. It has been reported as extreme in the vessels of the pia. Within the brain-substance the writer has noted vessels twisted and lying in a space, the lumen of which was much larger than normal; the sheath in fact was so much enlarged as to hold a corkscrew arrangement of the vessel. This was observed in epileptics. Increased vascularity is noticed in recent cases symptomatised by acute mania and acute melancholia, and in the early stage of general paralysis. But anæmia is more common in cases of long standing, and in many instances is due to the diseased or congested states of the vessels, to thickening of their walls, or to atheromatous deposits; in other cases to the condition of the general system. Thickening of the walls may be due to arteritis, which in specific cases may cause complete occlusion; and increase of the muscular coat, especially in chronic alcoholism, is of pretty frequent occurrence (B. Lewis). The opinion of this most careful observer must be held in respect; but it may be stated that, as regards other regions, the general opinion is that thickening of the muscular coat is more apparent than real, and that the appearance of hypertrophy depends on thickening of the fibrous tissues. Miliary aneurysms are not infrequent. Local atrophies are produced by implication of the terminal vessels by one or other of the morbid processes just spoken of. Adhesion of the visceral pia to the brain-substance is due to inflammatory changes in the vessels, and to consequent proliferation of the layer of Deiters' cells existing on the surface of the convolutions.

**Nerve-Cells.**—The morbid changes in the nerve-cells are most marked in the frontal lobes, and in the superior convolutions of the parietal lobes, which regions are in fact those mainly implicated in insanity. In the dependent portions of the hemispheres, in the occipital lobes, and at the base of the brain, morbid changes of any kind are rarely noticed.

The special morbid conditions of the nerve-cells are: (a) Pigmentary, granular, or fuscous degeneration; (β) Hypertrophy; (γ) Calcification.

(a) A certain degree of *pigmentation* is almost invariably found in the cells of the fourth layer of sane adults. The writer has examined these structures in twelve cases taken casually from the mortuary of an infirmary, and found it more or less observable in all cases over thirty years of age; but it was specially well marked in cases dying from pneumonia or bronchitis. The writer has examined the brains of demented where no evidence of pigmentation could be found. But an extreme degree of pigmentary, fuscous, or granular degeneration is a very common condition in many forms of insanity, particularly senile mania and general paralysis. Major distinguishes three stages: (1) The cells lose their sharply defined contour and become swollen or inflated; the axis-cylinder and apical processes usually remain intact, but the protoplasmic processes disappear, and the cell becomes rounded off; the nucleus becomes swollen, more or less round or oval, and displaced, while it and its nucleoli are more easily stained than in the normal state. (2) A deposit of granules takes place at the base of the cell, and gradually permeates its whole structure. (3) The cell goes on to destruction, breaking down and shrinking, leaving the nucleus surrounded only by a mass of granules, apparently of a fatty nature, forming a gap in the cerebral tissue previously occupied by the swollen cell; still later the granules entirely disappear, leaving the nucleus free. He has not observed the nucleus actually undergoing disintegration, but often no trace of it is to be found in the mass of granules left by the degenerated cell. Bevan Lewis's views on these changes are fully stated in his *Text-Book of Mental Diseases*.

(β) *Hypertrophy* of the cells of the fourth layer has been observed in senile atrophy and general paralysis. As the name implies, they are large, and swollen in appearance, often presenting unpigmented granular masses in their interior.

(γ) *Calcification* of the cells by the deposit of phosphate of lime within their walls has been observed, according to Blandford.

The cells are found *vacuolated*, and the nucleus may present a clear diaphanous spot.

In almost all cases of old-standing terminative dementia the protoplasmic poles are

either lost or materially diminished in number. This, along with the changes brought about by inflammatory processes and œdema, interferes with, or destroys, the relation of specialised areas.

**Neuroglia.**—The connective tissue of the brain undergoes inflammatory changes of a subacute or chronic nature, with the results of which we are familiar as more or less diffused *sclerosis*. In many forms of insanity the Deiters' cells along the course of the vessels and the outer layer of grey matter show indications of great activity. They, as well as the smaller cells, become increased in number, and the poles assume an amount of distinctness unnoticeable in health, staining deeply, and being traceable for many fields of the microscope. A meshwork is formed, destroying or displacing the normal nerve-fibres, and in the grey matter causing such a degree of homogeneity of structure as to make it stain as if it was composed of one tissue. In general paralysis, throughout the white matter the smaller cells are immensely proliferated, and along the course of blood-vessels the increase may be so great as to almost entirely hide them.

*General sclerosis* has only been observed in one case, which is fully detailed in the *Journal of Anatomy and Physiology*, May, 1873. In a hydrocephalic epileptic idiot (whose brain weighed sixty ounces) the hemispheres differed in weight; the left being 23½ ounces, the right 30¼ ounces. In the heavier or hypertrophied side, the nerve-fibres were found lying in fasciculi consisting of from four to six strands; these fasciculi were widely separated from one another by a clear, finely fibrillar plasma, in which nuclei existed, somewhat larger than normal.

*Disseminated or partial sclerosis, or grey degeneration*, is a lesion frequently met with in the brains of old-standing cases of insanity, especially in general paralysis. Its most frequent seat is the white matter of the motor tract; less frequently it is met with in the hemispheres. In the pons Varolii, medulla oblongata, and spinal cord of epileptics, patches of this disease are of common occurrence and in an extreme degree. When a fine section of nerve-tissue affected by this disease is examined by the naked eye, circumscribed opaque patches can be seen. In coloured sections these tracts are strongly tinted. As a rule, they are found contiguous to a vessel whose nuclei are much proliferated, and around which considerable proliferation of the nuclei of the neuroglia exists. Under the microscope, the nerve-fibres are seen to be partially or completely atrophied; the axis-cylinders and sheaths are destroyed; and the field is occupied by a finely molecular and fibrillated material, embedded in a cloudy homogeneous plasma. In this matrix the proliferated nuclei exist, somewhat enlarged, sometimes slightly granular in appearance;

but around the implicated spot they are to be seen in much greater quantity, and not actively diseased. The atrophied nerve-fibres occasionally project raggedly into the grey matter, where they are lost. Rokitansky believes this to be essentially a primary increase of the neuroglia; Leyden thinks it occurs secondarily to the atrophy of nerve-fibres; while Rindfleisch and others are of opinion that the first stage is marked by proliferation of the nuclei of the vessels, which is followed by an increase of the neuroglia and the development of a morbid plasma, which is, in all probability, modified neuroglia.

**Secondary Lesions.**—The two most important secondary lesions are *miliary sclerosis* and *colloid degeneration*. The former was first described by the present writer and Dr. Rutherford, and was ascribed by them to changes in the nuclei of the neuroglia. This theory must be abandoned; and the opinion of Dr. Bevan Lewis that it and colloid degeneration are due to myelin changes appears to be correct. The term does not properly indicate the pathological condition, but it has held place so long as to be generally accepted as indicating a certain lesion. Miliary sclerosis appears as unilocular, bilocular, or rarely multilocular spots, of a somewhat luminous pearly lustre, confined to the white matter, and consisting of molecular material with a stroma of exceedingly delicate colourless fibrils. 'In the immediate neighbourhood of these patches we find the nervous tissue in a state of parenchymatous degeneration, which, resulting in a destruction and atrophy of the essential elements, becomes the site of a *genuine sclerosis*. . . . The patch undoubtedly consists of altered myelin exuded in droplets from the medullated fibres and coalescing more or less completely' (Bevan Lewis). Colloid bodies are produced by analogous conditions.

J. BATTY TUKE.

**INSANITY, Varieties of.**—In this article various forms of insanity will be described under separate heads. DEMENTIA, GENERAL PARALYSIS OF THE INSANE, MANIA, and MELANCHOLIA will be found in other parts of the work.

**1. Alcoholic Insanity.**—The conditions included under this head must not be confounded with what is called dipsomania. In the latter affection the indulgence in alcohol is a symptom, and not necessarily a cause; while here the insanity is always a direct result of some form of alcoholic poisoning. It is met with in three forms—namely, *acute alcoholic insanity*, *chronic alcoholic insanity*, and *delirium tremens*.

*Acute alcoholic insanity* seldom occurs except when there is an hereditary tendency to mental disturbance, or when the cerebral energies have been impaired by excesses,

traumatic injury, or over-work. Where all these predisposing causes exist, it may not require a large dose of alcohol to bring on an attack. The most frequent form of the affection is violent maniacal delirium, known as *mania a potu*, with a tendency to homicidal acts. In some cases the mental disorder takes the melancholic form, and it becomes necessary to guard specially against the strong suicidal tendency which generally characterises it. Unless the brain has been weakened by repeated attacks, both forms are curable and generally of short duration. The treatment is the nourishing, non-stimulating regimen detailed in the articles on MANIA and MELANCHOLIA.

*Chronic alcoholic insanity* is one of the results of chronic alcoholism, and there is no condition which better illustrates the 'solidarity of the psychical and somatic functions of the nervous system' and the interdependence of their morbid manifestations. The physical symptoms are fully described in the article on ALCOHOLISM; the mental symptoms are generally present from the beginning, though not always prominent enough to attract special attention. The sleeplessness, so characteristic of commencing mental disorder, is an early symptom; then restlessness and depression, with suicidal tendency, sometimes passing rapidly into complete dementia, but generally passing gradually through a course of moral and mental degradation, which progresses step by step with the symptoms of failure of physical nervous power. The affection presents many points of resemblance to general paralysis of the insane, and is in some cases only to be distinguished from it by obtaining evidence of alcoholic poisoning, and by the persistence of the mental depression, which is seldom more than a transitory symptom in the general paralytic.

*Delirium tremens* is described fully under the heading of ALCOHOLISM; but it is proper to note here that, after the acute symptoms of that disease have passed away, there is sometimes left behind a state of subacute insanity of a characteristic nature. At first suicidal symptoms are apt to appear. Suspicions of poisoning, fear of impending evil, and hallucinations of hearing are also frequent. The treatment required is constant companionship of a trustworthy attendant, exercise, fresh air, and change of scene, with attention to every ordinary means of restoring the functions to a healthy state. Under proper treatment the prognosis is favourable.

JOHN SIBBALD.

**2. Amenorrhœal Insanity.**—Mental derangement is often accompanied in females by suppression of the menses. But in many such cases the insanity cannot be called amenorrhœal, as the cerebral and uterine disorders may only be associated as both symptomatic of some debilitating cause

affecting the whole system. Then the mental condition is usually the depression produced by anæmia. But there is a mental derangement directly resulting from sudden suppression of the catamenia, to which this distinctive name is not inaptly applied. Here the insanity takes the maniacal form. It is sometimes ushered in with urgent febrile symptoms, in which case the mania assumes the acutely delirious character. Where general febrile disturbance is not prominent, the mental condition is more simply maniacal, and sometimes does not get beyond mere irritability with delusions. The pathological condition must in either case be regarded as mainly a hyperæmia of the brain.

**TREATMENT.**—This must be directed towards the restoration of the arrested discharge. If the patient be seen at the commencement of the attack, the hip-bath and a gentle purgative may recall it. If the menstrual period has passed, the attention of the practitioner must be devoted to the relief of the more urgent symptoms. If the symptoms of cerebral congestion are distinct, leeches will probably be found useful, locally—behind the ears, or to the womb. Regular action of the bowels should be secured, but active purgation should be avoided. The food ought to be easy of digestion, and care should be taken not to let it be deficient in quantity. If the mental excitement is great and long-continued, it will necessarily produce considerable exhaustion, and the condition of the patient after recovery from the mental excitement will depend very much on the extent to which the strength has been supported during the continuance of this strain. The re-appearance of the catamenia generally implies recovery of mental health; but cases occur where prolonged amenorrhœa leads to a chronic maniacal condition, ultimately passing into hopeless dementia.

JOHN SIBBALD.

**3. Choreic Insanity.**—There appears to be an intimate connexion between the pathological basis of chorea and a certain disturbance of the mental functions. The physical and the mental symptoms, however, do not necessarily correspond in intensity. Sometimes, where the convulsive symptoms are very severe, the mental condition is merely one of dulness, apathy, or irritability. In children it shows itself generally in a maniacal restlessness, accompanied by delirium of a peculiarly automatic character. It is frequently associated at all periods of life with the rheumatic condition, and hence it has by some authors been called rheumatic insanity. It generally commences with sleeplessness and delirious excitement of a remittent character, which is sometimes accompanied by violent convulsive effort. As the excitement passes off, delusions of suspicion are apt to arise, and these are strangely associated with

an apathetic mental condition. In the acute form the prognosis is favourable, recovery generally taking place in from four to eight weeks. The chronic form is apt to pass into dementia.

JOHN SIBBALD.

**4. Epileptic Insanity.**—See EPILEPTIC INSANITY.

**5. Feigned Insanity.**—Insanity may be feigned in order to escape the obligation of duty, or the consequences of crime. If manifested at a time when its recognition might be a benefit to the individual, it becomes necessary to test its reality. It must not be rashly inferred in any case that insanity is feigned; for it sometimes results from the excitement consequent on a sense of guilt or the shock of a false accusation; or it may arise coincidentally but independently of such conditions. The best preparation for making a satisfactory examination in such a case is a familiar acquaintance with the appearance and conduct of persons undoubtedly insane. A person feigning insanity must, to be successful, simulate some known form of the disease; and as each form presents a more or less definite group of symptoms, an impostor is apt to reveal the truth by omissions or by additions inconsistent with the part that he attempts to play; but the mistake generally made by the impostor is to over-act the part. The inquiry may, of course, assume various aspects. A person may pretend to have been insane *at the time a certain act was committed*. Here it is to be remembered that the insanity, if real, would not probably have been confined to the time of the commission of the crime; and some evidence of premonitory symptoms previous to the act would probably be found. In such cases it is proper to regard want of motive as so far an indication of insanity; but when the supposition of a sudden mental perturbation is put forward, some reason would have to be shown for its occurrence; and some history of the occurrence of cerebral injury, or of previous attacks of insanity, epilepsy, or other cerebral affection ought to be forthcoming. A person feigning to be insane *at the time he is examined* must endeavour to present symptoms of either mania, melancholia, monomania, dementia, or imbecility. When the symptoms arise suddenly, simulation of the maniacal condition is generally attempted. The exertion which this entails will, however, generally compel an impostor to exhibit symptoms of fatigue, and even to sleep, when the true maniac would exhibit persistent excitement. The raving also when feigned may be recognised as hesitating and premeditated. Forgetfulness, which is generally assumed by the impostor, is an infrequent symptom of mania, except when it occurs in the course of general paralysis, and this is a disease whose other symptoms could scarcely be simulated. If melancholia or monomania

be feigned, the chief facts to be borne in mind are that such conditions, when real, are very unlikely to arise suddenly where there are no symptoms of bodily disease to account for them; and that they are usually characterised by a tendency to conceal peculiarities, or at least not to push them ostentatiously forward. Dementia never occurs suddenly without evident cause. Cases sometimes occur in which insanity is only partially feigned. Young criminals frequently try to exaggerate the signs of the intellectual weakness which is so generally mingled with their moral depravity, in hopes of obtaining a relaxation of discipline, or a change from a prison to an asylum. Such cases are often full of difficulty. The principle which ought to regulate our action is to avoid the continuance of punishment when disease or mental deficiency renders it useless. Before deciding upon the reality of any doubtful case of insanity, all the physical conditions of the individual, such as the amount of sleep, the state of the pulse, skin, tongue, and digestive system generally, the conduct and the state of health immediately preceding the signs of insanity, should be ascertained. The effect of remarks made within hearing of the suspected person should be observed; one who proclaims his own insanity should be distrusted. And the medical history of the family and of the individual should be inquired into, with a view to disclose anything which might have caused insanity or predisposed to it.

JOHN SIBBALD.

**6. Gastro-Enteric Insanity.** — The emotional condition is well known to be to an appreciable extent dependent on the state of the *prima viæ*; and where the nervous system is predisposed to derangement, certain affections of the liver, stomach, and bowels seem sufficient to produce insanity, and to stamp it with a special melancholic character. In addition to the mere depression caused by anæmia, there is associated with such affections a peculiar anguish of mind, and tendency to self-accusation, which is often of the most distressing nature. Refusal of food is frequently a prominent symptom. The intellectual perversion is often slight, and seldom so prominent as in other forms of acute insanity. Relief of the bodily symptoms is generally accompanied by a return to sanity. The affections which have been most frequently observed to produce this form of melancholia, are hepatic congestion, irritation and catarrh of the mucous membrane, constipation, stricture, or other causes of distension of the viscera, and pressure upon the stomach or intestines by tumours in the epigastric region. Schroeder van der Kolk described the mental symptoms as being always due to affections of the colon; but disease of other portions of the canal—as the rectum and anus—also produces them.

JOHN SIBBALD.

**7. Hereditary Insanity.**—SYNON.: Fr. *Folie Héréditaire*; Ger. *Erbliche Geisteskrankheit*.—This implies insanity symptomatic of hereditary weakness of the brain and nervous system, generally coming on without the intervention of appreciable exciting cause.

The nervous system seems to be peculiarly liable to be involved in the effects of hereditary degeneracy, and this is frequently evinced by the occurrence of mental symptoms. The ages at which these are developed, and the character which they exhibit, depend both on the nature and on the strength of the hereditary disposition. The forms of insanity, however, which seem to be most directly the result of hereditary influence, generally make their appearance at those periods of life when either rapid structural development takes place, special functional activity is first exhibited or is ultimately arrested, or upon the advent of senile decay. The ordinary exciting causes of insanity may also affect persons at these periods, and in such cases the resulting disorder will be stamped more or less distinctly with the impress of its origin. But where these forces do not come powerfully into play, it is found that hereditary insanity exhibits a special character according to the period at which it is developed. We have thus an *insanity of pubescence*, a *climacteric insanity*, and a *senile insanity*. Idiocy and imbecility (which will be found discussed under the head of the former) are also frequent results of hereditary weakness, showing itself during foetal life, or during the period of dentition; and the mental derangements which often affect women at parturition, and during the processes which precede or follow it, seem to occupy a position in the pathological scale intermediate between the hereditary and the accidental forms of insanity.

*Insanity of pubescence* is a condition not infrequently met with, and one which it is very important to recognise early in its true character. Much unintentional injury is frequently done by the patient being at first regarded as a delinquent and treated accordingly, instead of receiving the careful management suitable to the disease. The affection is characterised by great disorder of the emotional and moral nature, which is evinced by restless though seldom violent excitement, perverted eroticism, acts of purposeless mischief, and exhibitions of inordinate vanity. Any marked change of disposition during the passage from youth to adult life ought to be regarded as probably pathological in its nature, and must be carefully watched. Towards the completion of the period of adolescence there is a special liability to acute maniacal attacks, characterised by excitement of the sexual or religious emotions. The physical condition is indicated by capricious appetite and symptoms of

anæmia. The treatment required is rest to the mind, both in its intellectual and emotional energies, with cultivation of everything likely to develop physical vigour. The diet should be abundant, but chiefly milk, eggs, and farinacea. Butcher's meat should be used sparingly, and alcohol prohibited. If the hereditary tendency is not exceptionally strong, the prognosis is not unfavourable.

*Climacteric insanity* occurs in males between the ages of fifty and sixty, and in females between forty and fifty. Its general character is a form of melancholia, gradual in its development, manifesting itself in loss of sleep, fear of undefined evil, religious despondency, hallucinations of the senses, refusal of food, and frequently in a suicidal tendency. Excitement and exaltation occur rarely, and are generally of short duration. The mental disorder is frequently accompanied by very marked emaciation, and the tendency is always to anæmia. The treatment required at the commencement is cessation of mental labour and change of scene. During the whole progress of the disease the diet should be full and nourishing, and the digestive functions often require to be stimulated to healthy action. In the majority of cases the prognosis is unfavourable, and where recovery does not take place within one or at most two years the course is generally towards dementia.

*Senile insanity* is essentially a form of dementia, which comes on gradually in persons who have passed through the earlier periods of life without disturbance of their mental health, but who break down in old age. Its principal features are loss of memory, slight excitements, whimsical likings and dislikings, querulousness, and a gradual decadence into fatuity. It is subject to occasional remissions, which are sometimes very short, as when caused by the stimulus of acute febrile states. But these may be so prolonged as to amount practically to recovery. Not infrequently the breakdown of the nervous system pursues a rapid course, and in such cases there is often a marked similarity in the symptoms to those of general paralysis. Both mental and physical conditions in the advanced stages of each are sometimes practically indistinguishable. The diagnosis will depend on whether the characteristic first stage of general paralysis has been observed at the commencement, or only a gradual loss of physical and mental power. Paralytics, moreover, are seldom of advanced age.

JOHN SIBBALD.

**8. Impulsive Insanity.**—Violent acts are committed under an insane impulse by many patients whose insanity is plain and acknowledged. They may be done under the influence of delusions or hallucinations, but the term *impulsive insanity* is com-

monly applied to a disorder manifested not by delusions and similar symptoms, but by acts of violence to which a patient is driven by blind, uncontrollable, and morbid impulse, whereby the will and the reason are overpowered for a longer or shorter time. These are for the most part acts of *suicide* or *homicide*; and in connexion with the latter, great controversy has arisen as to the responsibility of persons committing them. Here, as in moral insanity, there are no delusions; frequently no change will have been detected in the individual prior to the act, nor will there be observers of it. And it is a fact that the impulse may be satisfied and exploded in the act, and having thus found a vent may be felt no longer, at any rate for a time. It may be as sudden as an epileptic fit, and may, like the latter, bring relief to the brain. Indeed, there is a strong connexion as well as analogy between the two disorders, and, as Dr. Maudsley says, instead of a convulsive movement there is a convulsive idea.

In estimating such acts as these we must not only consider the act itself and the manner of its performance, but must also closely inquire into the past history of the perpetrator and his progenitors. For all this we may or may not have opportunity. If the act is one of attempted suicide, and the individual is kept under observation, we may have no difficulty in diagnosing insanity. If it is one of homicide, and the criminal is in prison for, perhaps, only a week or two, opportunity of ascertaining the history of his family may fail, nor will he himself be under skilled observation. Moreover, the period just after the committal of an impulsive homicide will very probably be the one in which fewest symptoms of insanity will be noticeable. The period immediately preceding it will be that which most closely demands a scrutiny, but we may not be able to get any information if the accused has been moving from place to place, has been among strangers, or has had those about him who were obtuse and unobservant. Many acts of suicide and homicide would be prevented if the friends did not shut their eyes with pertinacity to the strange and altered looks and conduct of the patient. But in this impulsive form, although there may not have been enough to warn those about a man to restrain him, there may have been symptoms which are sufficient subsequently to indicate to a physician the presence of mental disorder. There may have been attacks of *grand mal* or *petit mal*; there may have been former attacks of insanity, periods of, perhaps, slight depression, which, though they attracted little notice at the time, may indicate an insane diathesis. The sufferer may have been sleeping badly or have taken less nourishment than usual.

The medical witness will, in the case of

homicide, carefully examine and pay special attention to the following points:—

(a) The nature and character of the act must be noted. The presence or absence of motive may often assist us. When the victim is near of kin and dearly loved, suspicion of insanity will at once arise. When, on the other hand, it is a perfect stranger, never before seen, where there has been no previous meeting or quarrel, the same suspicion will arise. The method of the act may guide us somewhat, but not so much. There may be premeditation, though generally there is not.

(b) The demeanour of the prisoner after the act may assist us. Were there, or were there not, attempts to conceal it, or to escape detection and arrest, and, if so, what was the nature of them? What was said in explanation? It sometimes happens that there is complete unconsciousness or forgetfulness of what has occurred, and we may then strongly suspect the presence of epilepsy.

(c) We must closely inquire into the family history, and shall often find that, in cases of impulsive homicide, the family of the accused has suffered with insanity. And where this is so, we may also find that from youth the accused has been deficient and weak in intellect, or odd and eccentric. The weak-minded, in fact, may be grouped in a special class of homicides. As there is a weak-minded moral insanity, so there is a weak-minded impulsive homicidal insanity, the sufferers from which have not infrequently been hanged, their insanity not having been sufficiently marked to absolve them from legal responsibility. Fits in childhood may contribute to this state. And throughout, at the age of puberty, or in adult life, there may be slight but sure indications of the insanity that has been inherited, which are displayed in an impulse to mischief, homicide, or suicide, even as in others the tendency is shown in ordinary attacks of insanity.

(d) We must look very closely for symptoms or a history of epilepsy. Such indications as nocturnal micturition or a bitten tongue may guide us to the truth; while in acknowledged epileptics it may happen that the homicidal attack takes the place of the ordinary convulsion, and without the occurrence of the latter there may be a period of unconsciousness and unconscious action lasting, perhaps, for days. See EPILEPTIC INSANITY.

The occurrence of one homicidal attack of a strange or anomalous character may make us fear its recurrence; and when we have to examine a criminal who has committed one act of this kind, it is important to inquire whether he has ever at any former time done any sudden act of violence of a similar description. For this reason such patients should not be released from an asylum, except under great and special precautions.

G. F. BLANDFORD.

9. **Legal Insanity.**—Lawyers regard insanity from a different point of view from medical men. A medical man applies his mind to its study so as to ascertain how far he may infer from the evidences of mental action the existence of morbid conditions which he may hope to alleviate or remove. It is therefore his duty to be acquainted with such symptoms as give the earliest indication of the approach or development of these morbid processes. For it is during their initiatory stages that he may most successfully intervene with the resources of his art, to check their progress or to ward them off. He therefore identifies with the existence of disease every deviation from the healthy mental standard which indicates the necessity for medical treatment or advice. A lawyer, on the other hand, takes note of insanity only in so far as it affects the safety of person or the preservation of property. The question which he seeks to determine is whether a person is justly responsible for certain acts which he has committed, or is competent to perform certain acts which he may be called on to perform. It is evident, therefore, that the legal view of insanity must naturally be much more limited than the medical. ‘A lawyer, when speaking of insanity,’ says Sir James Stephen, ‘means conduct of a certain character; a physician means a certain disease, one of the effects of which is to produce such conduct;’ and though this has been adversely criticised, it seems to show correctly the directions in which the two views diverge. They might perhaps be as fairly indicated in other words if we say that the lawyer has to deal with the nature or quality of certain acts, while the physician has to deal with the condition of certain persons. No satisfactory general definition of legal insanity has been given. In the earlier ages of our legal system none but the most outrageous cases of insanity were recognised. Bracton in the thirteenth century defined a madman as ‘one who does not understand what he is doing, and, wanting mind and reason, differs little from brutes.’ Sir Edward Coke, though he recognises different classes, according as the insanity is congenital, permanent, or temporary, only admits that a person is insane when he is *non compos mentis*, or has wholly lost his memory and understanding. Sir Matthew Hale, in the seventeenth century, was the first to recognise the existence of less extreme forms of insanity. ‘Some persons,’ he said, ‘that have a competent use of reason in respect of some subjects, are yet under a *particular dementia* in respect of some particular discourses, subjects, or applications; or else it is partial in respect of degrees, and does not excuse persons who commit capital acts in this state.’ He also said that it is ‘very difficult to define the invisible line that

divides perfect and partial insanity,' and 'that most persons that are felons of themselves or others, are under a degree of partial insanity when they commit these offences.' The recognition of these gradations, reaching even to the mutual overlapping of crime and insanity, indicates as much breadth of view as could be expected at a time when the very judge who recognised them passed sentence of death on persons convicted of witchcraft. We cannot doubt, indeed, that at that period the ignorance of the nature of insanity was such that many lunatics were executed for this offence. The recognition of the necessity of taking legal account of degrees of insanity less severe than 'furious madness' or idiocy may be said to have commenced at the beginning of the present century. The mode in which unsoundness of mind comes into relation with law at present may be looked at most conveniently from three points of view: 1. Where a person suffers from such unsoundness of mind that it is necessary for his welfare, or the safety of the public, that his liberty should be restricted, by his being placed in an asylum or subjected to similar restraint. 2. Where a person suffers from such unsoundness of mind that he is incapable of managing himself or his affairs. 3. Where irresponsibility for crime, on account of insanity, is pleaded in a court of law. It will be found more convenient to consider these three relations of insanity and jurisprudence separately. See LUNACY, Law of; CIVIL INCAPACITY; and CRIME, Irresponsibility for.

JOHN SIBBALD.

**10. Moral Insanity.**—SYNON.: Fr. *Manie sans Délire*; *Folie Raisonnable*; *Monomanie Affective*; Ger. *Gemüthswahnsinn*.

Under the names of *moral insanity*, *emotional insanity*, *impulsive insanity*, *affective insanity*, has been described the disorder of certain patients, which is manifested by insane actions and conduct rather than by insane ideas, delusions, or hallucinations. Such persons are sometimes said to be of whole and perfect intellect, though unsound in the moral and emotional part of their brain. They come under the notice of medical men not so much for purposes of treatment as for diagnosis. Their conduct being chiefly displayed in foolish or violent acts, they require to be restrained, and the question arises: Is this conduct badness or madness? are they responsible for it or not?

Dr. Maudsley gives *moral insanity* and *impulsive insanity* as two subdivisions of *emotional* or *affective insanity*; and, as the symptoms are certainly different, we cannot do better than consider them under separate heads. But certain observations are applicable to both. In neither will there be found delusions; and as delusions are, in the opinion of some, especially lawyers, necessary to the

establishment of legal insanity and irresponsibility, these patients, according to them, are not legally insane. Another remark is that this moral or impulsive insanity does not constitute a definite and well-marked disease, like acute delirium or general paralysis. Every patient may at one time or other be 'morally insane'—that is, may not have reached the stage of delusions, or may have recovered from it, and every patient may commit 'impulsive' acts of violence, whether his insanity is displayed in other ways or not.

By *moral insanity* is to be understood a disorder of mind shown by an entire change of character and habits; by extraordinary acts and conduct; extravagance or parsimony; false assertions and false views concerning those nearest and dearest, but without absolute delusion. Such a change may be noticed following any of the ordinary causes of insanity. It may follow epileptic or apoplectic seizures, or may be seen after a period of drinking. Its approach is gradual, as a rule, rather than sudden, and the extraordinary character of the acts may not at first be so marked as subsequently. Friends wonder that a man should say this or that, or should do things so foreign to his nature and habits, but some time may elapse before they can convince themselves that such conduct is the result of disease, and the acts may be such that many will look upon them even to the last as signs merely of depravity. Such insanity, of course, varies in degree. When it is well marked, and the conduct is outrageous, there will be no difficulty in the diagnosis. But it may be less marked; it may consist of false and malevolent assertions concerning people, even the nearest; of little plots and traps to annoy others, in which great ingenuity and cunning may be displayed. And there will be the greatest plausibility in the story by which all such acts and all other acts will be explained away and excused. It would seem sometimes as if a universal badness had taken possession of the individual, yet a badness so inexplicable that it can only be looked upon as madness. Where we can ascertain that this condition of things is something which has come over the patient, being formerly absent, and that he is altogether changed, we may suspect insanity. But much examination and opportunity for examination may be needful before we can sign a certificate, for such people are often very acute, and quite on the alert. They have no scruples about falsehood, and will deny or justify everything with which they are taxed; and where the insanity is manifested in conduct, the medical man may never be a witness of it, and is obliged to receive on hearsay that which the patient strenuously denies. Careful inquiry, however, will probably reveal the origin and cause of the change. There

may have been a period, though short, of acute insanity—as acute mania or melancholia—which passed away and left this as a permanent condition; or it may be the precursor of a more advanced stage of the disorder, one marked by the ordinary symptoms, as delusions and hallucinations. If the change has been rapid and progressive, if the sufferer has become more and more outrageous and eccentric, it is likely that in a short time unmistakable insanity will be displayed; but some cases progress slowly, and steps for restraint have to be taken before anything like delusion is to be found. It may be necessary to prevent a man from squandering all his property—a common symptom in this variety—or from wandering from home and absenting himself no one knows where, or keeping low company. And when a man previously quite sober suddenly takes to drinking, the question may arise whether this is not the effect of insanity. Great difficulty may be found in proving the latter, but unquestionably it is often the case. Here, however, if the habit is indulged in, the patient will most likely get rapidly worse, and then restraint will be more easily enforced.

Moral insanity may be the precursor of general paralysis; it may also be the sequel and result of a more severe insanity; it may be the outcome of apoplexy, or of a blow or other damage to the brain. It may be one of the alternating states of the so-called *folie circulaire*. Here a period of depression alternates with one of excitement, gaiety, self-glorification, or irascibility, and the latter may be one closely resembling that usually called moral insanity, and evidenced by exaggerated conduct and absurd acts. It may follow a simple attack of epilepsy, or may be the precursor of such attacks, being a part of that epileptic condition known as masked epilepsy. See EPILEPTIC INSANITY.

The one constant and marked feature of this insanity is the absence of delusion; but we are not on this account to argue that the intellect is sound. There is often great acuteness and cunning displayed by such patients, yet along with the cunning there may be the most silly and foolish conduct. Often there is great acuteness shown by those who have delusions, but because of the latter we say their intellect is disordered. Yet it is proof of disorder of the intellect if a patient spends his capital as though it were income, defends and justifies the most outrageous acts, and cannot be made to see that they are outrageous. Close examination will probably reveal the fact that there is very considerable intellectual lesion in these cases. There is a want of the power of attention and concentration of ideas on a subject. A patient commences a story of his grievances, and in two minutes is far away from his theme, and is boasting of his virtues or conduct, and no amount of bringing back will enable him to

give a definite and succinct account of what he has to complain of. Such rambling is a marked symptom of this insanity, and a strong indication of a weakened intellect.

There is one more form of moral insanity which is, perhaps, the hardest of all to diagnose and estimate. It is the congenital moral defect occasionally met with in persons who have been from birth odd and peculiar, and incapable of acting and behaving like other people. They can hardly be called idiots or imbeciles, for they may exhibit a considerable amount of intellect and even genius in certain special directions. We shall generally find that they are the offspring of parents strongly tainted with insanity, epilepsy, or alcoholism, and many in childhood are the subject of fits, chorea, or other neuroses. They are incapable of being instructed like other boys and girls; are often frightfully cruel towards animals, or their brothers and sisters; and seem utterly incapable of telling the truth or understanding why they should do so. Here there is no change; we cannot compare the individual's condition with a former one, but we can only estimate him by the average of mankind. These are the persons who commit crimes and become the chronic inmates of prisons, and it is most difficult both for medical and other prison-officials to say how far they are responsible, and how far not. Careful and special education is needful for them, and this they may obtain if they are born of well-to-do parents; but a vast number are to be found amongst the ranks of the lower classes; the offspring of intemperance and poverty, they swell the numbers of the criminal classes in no inconsiderable degree.

G. F. BLANDFORD.

**11. Pellagrous Insanity.**—This is a form of insanity associated with pellagra, and not met with in Britain. It is characterised by mental symptoms usually indicative of anæmia—great depression, frequently with tendency to suicide, passing on to chronic dementia. It is most frequently met with in some parts of Italy.

**12. Phthisical Insanity.**—It has been generally observed that there is a special mental character associated with pulmonary tuberculosis. This consists frequently in a peculiar cheerful hopefulness, which has received the name of *spes phthisica*, and which seems strangely out of harmony with the unmistakable signs of an inevitable fate by which it is accompanied. But there is also a state of mental depression which has been observed in intimate association with the disease. The peculiar hopefulness is most frequently met with in the acute and active forms of phthisis, and it is often so irrational and persistent as to amount to an insane delusion, growing as it does in strength while the evidences of its baselessness

accumulate. In the last stages of the malady the religious and general emotional exaltation is often extreme, and actual delirium sometimes occurs. An opposite mental condition may be met with in chronic phthisis, more especially in that form of it which has been called latent. Throughout the course of the disease there is then a prevailing depression and distrustfulness, though the physical symptoms are neither so distressing nor so obvious as in the acuter forms. The mental symptoms sometimes precede the physical. Languor and depression, mingled with waywardness, are characteristic of the initial stage. This is usually accompanied by general functional debility, which is often attributed to mere disturbance of digestion and nutrition. The skin is habitually pale, and the circulation feeble. In many cases the physical signs of pulmonary disease when present are apt to be overlooked, and occasionally they escape observation for years. Where the mind is much affected, the ordinary symptomatic cough, expectoration, and dyspnoea are often absent; and this is the case sometimes where physical exploration reveals the existence of extensive vomicae and other characteristics of advanced disease. As the further stages are reached the mental condition becomes less one of depression and more one of distinct enfeeblement. Occasional fits of considerable irritability and great excitement occur. Dr. Clouston, who first drew special attention to this condition, says of the patients that 'there is a want of fixity in their mental condition. There is a disinclination to enter into any kind of amusement or continuous work; and if this is overcome, there is no interest manifested in the employment. It might be called a mixture of subacute mania and dementia, being sometimes the one and sometimes the other. As the case advances, the symptoms of dementia come to predominate; but it is seldom of that kind in which the mental faculties are entirely obscured, with no gleam of intelligence or any tendency to excitement. If there is any tendency to periodicity in the symptoms at all, the remissions are not so regular, nor so complete, nor so long as in ordinary periodical insanity. If there is depression, it is accompanied with irritability and the want of any fixed depressing idea or delusion. If there is any single tendency that characterises these cases, it is to be suspicious.' Where any chronic form of insanity is associated with phthisis, the chance of complete restoration to sanity is very small.

In the affection distinguished by emotional exaltation no special treatment is called for. The only indication of treatment in the other type of disease is the necessity of keeping up a full supply of nourishment to the brain, by the use of nutrient enemata or otherwise. Where the mental condition is much dis-

turbed, a trustworthy attendant ought to be employed, and the general treatment should always be tonic and stimulating. Removal to an asylum ought in most cases to be avoided.

JOHN SIBBALD.

13. *Puerperal Insanity*.—SYNON.: *Insania Gravidarum*; *Insania Puerperarum*; *Insania Lactantium*; Fr. *Folie Puerpérale*; Ger. *Puerperalmanie*.

DEFINITION.—Insanity developed during pregnancy, parturition, or lactation.

It has been the custom of authors, till a recent period, to include under this heading the forms of insanity which occur in females during the periods of utero-gestation, the puerperal state, and lactation. It is now frequent to find the term 'puerperal' restricted to the derangements which come on at the time of delivery, or within a short period thereafter. Whatever names we adopt, it is impossible to consider the insanity occurring at these different periods quite independently. The condition of the woman during the whole process, from the time of conception till the child is weaned, represents one of those physiological crises during which congenital or acquired weakness of constitution tends to show itself.

The *insanity of pregnancy* generally takes the melancholic, though sometimes the maniacal form, and seems to be due to an incapacity in women of neurotic tendency to bear the special strain of the reproductive process. The delusions which characterise the disorder are generally exaggerations of the anxieties and whims so frequent in pregnancy. The family affections are weakened or perverted. In the severer forms the suicidal impulse is strongly developed. Mental disorder seldom commences before the third month of pregnancy, and most frequently about the sixth month. Except in the severer forms the prognosis is generally favourable; but recovery seldom occurs till the termination of the gestation. Asylum treatment should be avoided when the circumstances of the patient permit of suitable nurses or companions being employed at home.

*Puerperal insanity* in its more restricted meaning is frequently understood to include all derangements occurring at parturition, or within six or eight weeks after it. But it is better to limit the term to what occurs during the first three weeks, as the form of disorder which commences after that period is generally different in character.

ÆTIOLGY.—Prominent among the causes of puerperal mania are all states of debility, either induced before parturition by want, intemperance, disorders of nutrition, or rapid succession of pregnancies, especially if lactations and pregnancies are carried on simultaneously; or it may be the result of weakness induced during parturition by hæmor-

rhage or exhaustion. It is liable to occur in primiparæ, when the subjects are either exceptionally young or exceptionally advanced in life. Irritation arising in the pelvic organs, intestines, or mammæ also tends to produce it. Any inordinate mental excitement or depression is apt to bring it on. In a large number of cases it will be found that there is a hereditary neurotic tendency.

**SYMPTOMS.**—Attacks of maniacal excitement sometimes occur during actual parturition. They are usually of very short duration, and seem to be directly dependent on the intense suffering which may accompany the pains. The most-frequent period of their occurrence is when the head of the child is passing either the *os internum* or the *os externum*. The more serious phase of the disorder is in its acute stage a variety of acute maniacal delirium. It usually commences within a week or ten days after delivery. Generally it is preceded by sleeplessness, and the patient manifests more or less apprehension of coming evil. Sometimes, however, she awakes delirious from what had been regarded as a healthy slumber. When the attack has commenced, sleep is always either very imperfectly obtained, or is altogether absent. The pulse is thready and quick; the skin often, but not always, dry and hot; and the head throbbing. The eyes are bright, and the face generally pale, with occasional flushing. The expression is usually indicative of alarm or suspicion on the part of the patient. The tongue is dry and furred, and the secretions of milk and lochia are either suppressed or diminished. The bowels are sometimes loose, but constipation is the usual condition. The appetite is uncertain; sometimes it is abnormally large. Not infrequently the sense of taste is perverted, and the patient suspects the presence of poison in her food, and persistently refuses it. The breath is often offensive in odour. Sometimes the mental excitement does not reveal itself in language, and the patient may be obstinately taciturn from the commencement. But there is usually a great increase of loquacity, gradually progressing from the beginning, and passing into incoherent raving. Sudden impulsive acts of violence frequently manifest themselves in this disorder, and in these the patient often attempts to destroy herself, her child, or persons for whom she has usually the most affectionate regard. She generally appears to be dissatisfied with those in attendance on her, and often entertains delusions as to their identity. In many cases the mental condition bears a strong resemblance to that of delirium tremens, especially when the patient has undergone privations, or has been intemperate during pregnancy.

**DIAGNOSIS.**—The only conditions for which puerperal insanity may be mistaken are the typhoid delirium of puerperal fever or py-

æmia; and the violent excitement frequently symptomatic of meningitis. In these cases the febrile condition precedes the development of the delirium, while in puerperal mania the mental symptoms show themselves from the first. In meningitis the pupils are generally contracted, and the headache is peculiarly intense; while in puerperal mania the pupils are usually dilated, and the headache is not a very prominent symptom.

**PROGNOSIS.**—According to the published statistics of this disease, recovery may be expected in about 70 per cent. of the cases, and a fatal termination need not be anticipated in more than 5 per cent. But as these figures are in a great measure obtained from asylum statistics, and other sources in which only the severer and more persistent forms have been taken into account, it may be fairly assumed that the true estimate would be much more favourable. The duration of the insanity may vary from a few days to a year, after which time the proportion of recoveries becomes extremely small. The great proportion of recoveries takes place during the first six months. The most favourable symptoms, in addition to amelioration of the mental symptoms, are increase of bodily weight, and restoration of the catamenia.

**TREATMENT.**—The transitory mania which sometimes accompanies the severer pains of labour does not require special treatment; but anæsthetics may be given as a preventive measure in cases where there is a known liability to such excitement. Special care ought also to be taken after such attacks to avoid injury by premature bodily exertion, unnecessary social intercourse, disquieting news, or any kind of mental or moral strain. In the treatment of the graver form of puerperal mania, the chief objects are to remove all sources of irritation, to restore the patient's strength, and to obtain repose. If there is any accumulation of fæces, a smart purgative should be at once administered. If the lochial discharge is scanty, an injection of warm water, containing carbolic acid or some other antiseptic remedy, should be given *per vaginam*. The condition of the bladder should be ascertained, and this organ should be relieved, if necessary; care should also be taken not to allow the breasts to be over-distended. In every case the patient should from the first receive as much light and nourishing food as her digestive powers can bear. Where food is refused, custard must be given largely and frequently. Nutrient enemata or suppositories should be administered, or even the œsophagus tube used if necessary. In those cases in which violent excitement does not come on suddenly, an attack may sometimes be warded off or cut short by relieving the sleeplessness which is one of the early symptoms. A full supply of easily digested nourishment is often the best

hypnotic. Chloral hydrate, or perhaps sulphonal, will be useful. Opium ought to be avoided, and hyoscyamus and belladonna cannot be relied on. Both in the first stage, and when the disease is more advanced, everything must be done to promote tranquillity. The room should be darkened, and stillness maintained as far as possible, for the attention of the patient is very easily excited, and both vision and hearing are preternaturally acute. Constant supervision is necessary, however, on account of the tendency to sudden impulsive violence; and the patient should see her infant as seldom as possible. After the maniacal condition has fairly declared itself, the child should be removed altogether, as its presence is sometimes productive of great excitement in the patient, and must always be regarded as attended with risk. In cases where the excitement is somewhat of a hysterical character, bromide of potassium, given in twenty-grain doses at intervals of four hours, may produce good results. Cooling applications to the head sometimes soothe irritation, and induce sleep. Digitalis in small doses, and warm baths, have proved useful. Alcoholic stimulants should generally be given with the food; their effects being carefully watched, and the quantity varied accordingly. Bleeding and every kind of depletion should be avoided. The nursing and attendance should, if possible, be entirely entrusted to strangers, and the patient should not be permitted to see any members of her family. It will sometimes be necessary, especially in the case of poor persons, to resort to asylum treatment; but this ought not to be done if it can be avoided, and with patients in good circumstances it ought never to be necessary in the acute stage of the disease. When the disease becomes chronic, tonics, such as quinine and iron, ought to be given; and a plentiful supply of judicious nourishment should then, as all through the illness, be carefully administered. Patients liable to this disorder should not be allowed without consideration to nurse their children.

The *insanity of lactation* is symptomatic of causes which come into play after the puerperal period, and it ought perhaps to be looked on as symptomatic merely of prolonged anæmia. Acute maniacal symptoms of short duration may occur; but the characteristic condition is melancholia, ushered in by headache, tinnitus aurium, flashes of light before the eyes, and other signs of debility. A suicidal tendency sometimes appears. The treatment required is to wean the child, and generally to save and increase the strength of the patient. The prognosis is favourable in the majority of cases.

JOHN SIBBALD.

14. **Syphilitic Insanity.**—Among the results of constitutional syphilis, affections of

the nervous system are not uncommon; and when the disease affects the brain, the mental symptoms that arise are found in the majority of cases to present a marked similarity in their character. In such cases the mental disorder is generally preceded, as in so many forms of insanity, by distressing sleeplessness. This is followed gradually by increasing depression of mind. Religious anxiety of a peculiarly hopeless character frequently shows itself. Exaggerated self-accusings are earnestly uttered by those who have previously been unusually callous as to the results of their actions. Hypochondriacal delusions are not uncommon, and are frequently associated, in the mind of the patient, with the fact of the syphilitic infection. The feeling of alarm which accompanies these symptoms sometimes develops into a violent excitement, which may be called maniacal. If the symptoms be associated with any of the ordinary signs of syphilitic poisoning—the dry, scaly skin, sore-throat, and the sallow lean face; and especially if any of the characteristic eruptions are present, the mental disease may be expected to yield to treatment by mercurials and iodide of potassium. The mental symptoms which have been here described seem to occur without the existence of any important structural lesions in the encephalon. The development of gummy products within the cranium is frequently evinced by symptoms similar to those of general paralysis. Headache of very persistent character, giddiness, vertigo, and epileptoid fits occur, accompanied at first with mental depression. During the progress of the disease attacks of acute delirium are not unusual. Sometimes extravagant delusions, such as are frequent in general paralysis, are exhibited; but generally the progress of the disease is characterised by a gradual falling into dementia. The prognosis in such cases must be regarded as unfavourable, but considerable improvement frequently follows the administration of antisyphilitic remedies.

JOHN SIBBALD.

**INSECT PARASITES.**—Insect parasites are of two kinds, external and internal. The former are described under several affections of the skin (*see* PEDICULUS); the latter may be classed with the entozoa. *See* ENTOZOA; CHIGOE; and ŒSTRUS.

**INSENSIBILITY** (*in*, not; and *sentio*, I perceive).—This word signifies either loss of consciousness, or merely loss of sensation in a particular part of the body. *See* CONSCIOUSNESS, Disorders of; and SENSATION, Disorders of.

**INSOLATIO** (*in*, in; and *sol*, the sun). A synonym for sunstroke (*see* SUNSTROKE). Insolation is also used to designate a method of treatment, which consists in exposing the patient to the rays of the sun.

**INSOMNIA** (*in*, not; and *somnus*, sleep).—Want of sleep, or sleeplessness. See SLEEP, Disorders of.

**INSPECTION** (*inspicio*, I look upon). The technical name for the examination of a patient by the sense of sight; also for examination of a body *post mortem*. See PHYSICAL EXAMINATION; and NECROPSY.

**INSTILLATION** (*in*, into; and *stilla*, a drop).—The method of applying remedies to a part in the form of drops. Instillation is chiefly employed in connexion with the eye.

**INSUFFICIENCY** (*in*, not; and *sufficio*, I am sufficient).—A synonym for incompetence. See INCOMPETENCE.

**INSUFFLATION** (*in*, into; and *sufflo*, I blow).—This term is used in the same sense as inflation (see INFLATION). It is also a name given to a method of applying remedies in the form of powder to the throat and respiratory passages, by blowing them through a tube into these parts. See INHALATIONS, Therapeutic Uses of.

**INTEGUMENTS, Diseases of.**—See SKIN, Diseases of.

**INTELLECTUAL INSANITY.**—See INSANITY.

**INTEMPERANCE, Effects of.**—See ALCOHOLISM; and DISEASE, Causes of.

**INTERCOSTAL NEURALGIA.**—Any of the dorsal nerves may be the seat of neuralgia, not differing materially in its symptoms from neuralgia affecting other mixed nerves, but especially important from a diagnostic point of view. The pains are paroxysmal; usually affect the region of distribution of the anterior division of one or two of the dorsal nerves; and are confined to one side, most frequently the left.

**ÆTIOLGY.**—The female sex, neurotic heritage, and weak general health predispose to intercostal neuralgia. As determining causes may be mentioned blows; the action of cold; local injury to the nerves from the growth of thoracic aneurysm or tumour; and disease of the vertebræ. Exhaustion from over-suckling, menorrhagia, or leucorrhœa; irritation from cracked nipples; and pregnancy, are all occasional but important causes of this form of neuralgia. The pain met with in the chest in early cases of phthisis is not infrequently due to intercostal neuralgia. It is probable that, in a large proportion of cases, the neuralgic pain is due to neuritis of one or more intercostal nerves.

**SYMPTOMS.**—Pain is complained of at some part of one side of the thorax or abdomen, most often in the region innervated by the sixth, seventh, eighth, or ninth intercostal

nerves, and much more frequently in the front or side than behind. It is occasionally found in the axilla and inner side of the arm. The pain may be intermittent, occurring in paroxysms, varying in number from a recurrence every few minutes to only two or three such during the twenty-four hours; or there may be persistent pain of a dull character, interrupted at varying intervals by darts of a very sharp kind, which may sometimes be referred with precision to the course of the neighbouring nerve. The pain is described as 'tearing,' or resembling such injuries as a 'stab of a knife,' or 'boring with a red-hot iron.' The acts of coughing or sneezing, as well as any rapid movements of the body, are apt to increase the distress, but the pain is also independent of these disturbances, and will attack without any such provocation. The pain is sometimes more of a wearing than acute character, and the rest will often be destroyed by it. Painful points are sometimes to be discovered in the following situations: 1. Over a spinous process corresponding to the emergence of the affected dorsal nerve from the intervertebral foramen. 2. At the side of the chest or abdomen, where the lateral branch becomes subcutaneous. 3. Near the sternum or at the margin of the rectus abdominalis muscle, at any part down to the pubes, where the termination of the nerve supplies the skin. The skin in the neighbourhood of the tender points is sometimes so hyperæsthetic that the pressure of the clothes is painful. In epileptics and other highly neurotic patients, intercostal neuralgia is often associated with palpitation of the heart, and the pain is usually referred in a vague manner to that organ. Close examination will show that it is in the chest-wall, and tender points may generally be discovered. The affection is not accompanied by fever. The paroxysms of pain may produce fainting and vomiting. They often cause dyspnoea, with an anxious expression of face, from the inability to draw a full breath without starting the pain.

**COMPLICATIONS.**—Intercostal neuralgia is sometimes accompanied by herpes zoster. The pain usually precedes the appearance of the eruption, but it is occasionally coincident only, and sometimes comes after it; more often than not it outlasts the eruption, possibly for a long period. In certain cases actual pain lasts but a few days, but is succeeded by an intolerable itching, which is described as being under, not in, the skin. This sensation is said to be felt less in walking than when at rest.

Not infrequently neuralgia of some other nerves, either at a distance, as the fifth, or anatomically near, as the brachial plexus, occurs as a complication of intercostal neuralgia. This is especially likely in cases happening in the period of bodily decay. It is then, too, that the affection may occasion-

ally be accompanied by attacks of angina pectoris.

**DIAGNOSIS.**—Absence of pyrexia, as shown by the use of the thermometer; the intermittence of pain, and its occurrence irrespective of respiratory movements, although liable to be precipitated by them; and the results of physical examination, serve to distinguish intercostal neuralgia from pleurisy, a condition with which it is very apt to be confounded, on account of resemblance in the stabbing character of the pain. It should be remembered, however, that exceptionally no rise in temperature may accompany the painful stage of pleurisy.

From muscular rheumatism it may be discriminated by the presence of the small and characteristic tender points; by tenderness of the spinous processes on pressure; and by the pain being found to be not dependent upon movements. The same features serve to distinguish it from myalgia, especially that form which often comes from long-continued use in an unaccustomed manner of some muscle attached to the ribs, as when a person unused to carpentering handles the saw energetically for a long time.

Physical examination and the presence of pyrexia will preserve from the error of confounding the dull pain often noted in pneumonia with that of intercostal neuralgia.

Pains of a stabbing, plunging, or electric-shock-like character are often experienced in the intercostal spaces in the course of tabes, and it is important not to confound this disease with a simple attack of intercostal neuralgia. The distinguishing points are the occurrence of similar pains coincidentally or alternately in other parts of the body, especially in the lower extremities; the absence of patellar tendon reflex; and the characteristic gait (if present)—all which mark tabes. *See* **TABES DORSALIS**.

**PROGNOSIS.**—As in the case of other forms of neuralgia, that of the intercostal nerve can hardly be said to be attended with danger, though it must be allowed that in some very rare instances the severity of the pain appears to have actually destroyed life. It is apt, however, to be of troublesome duration, dependent on the cause, lasting for periods of weeks or months, and liable to recurrence.

**TREATMENT.**—Search should be made in other branches of the same nerve, and in the distribution of neighbouring nerves, for any source of irritation which it is possible to remove. Constipation of the bowels should be treated by three grains of blue pill, followed by some Friedrichshall or Hunyadi János water, repeated on two or three occasions. Quinine should be given in doses of from five to ten grains twice a day; and if there should be a state of anæmia, iron should be added. Phenazone in doses of from ten to twenty grains may be given two or three times a day, if necessary. Exposure to cold and

damp must be avoided; whilst the surroundings generally should be favourable to improving the nutrition and tone. If the pain be very acute, and sleep prevented, morphine may be injected hypodermically in the neighbourhood of the affected nerve, commencing with a dose of a twelfth of a grain, and increasing this, if necessary, to a quarter of a grain in the twenty-four hours. This dose should not be repeated, however; and it is better to be satisfied with a repetition of the smaller dose, if required. Small blisters (size of half-a-crown) may be applied to the neighbourhood of the spinal column, near the point of emergence of the affected nerve, one succeeding another as it heals. The continuous voltaic current, from about ten to twenty cells, Leclanché or Stöhrer, may be applied, one sponge on the spine and the other upon the painful points in turn. There is no better application than flexible or anodyne collodion for the herpes zoster which sometimes complicates intercostal neuralgia. *See* **NEURALGIA**. **T. BUZZARD.**

**INTERLOBULAR** (*inter*, between; and *lobulus*, a little lobe).—Situated in the tissue between the lobules of any organ. A good illustration is *interlobular emphysema*, in which air occupies the tissues between the lobules of the lungs.

**INTERMITTENT** (*intermitto*, I leave off for a time).—A temporary cessation or suspension, either of a function, for example, of the action of the heart, when the pulse is said to intermit; or of a disease, such as neuralgia or ague, when the symptoms cease for a certain time. *See* **PULSE**; and **INTERMITTENT FEVER**.

**INTERMITTENT FEVER.**—**SYNON.** Ague; Fr. *Fièvre Intermittente*; Ger. *Kaltes Fieber*.

**DEFINITION.**—A fever of malarial origin, characterised by the sudden rise of temperature during the paroxysm; by the equally sudden fall at its termination; and by the regularity of the times of accession and apyrexia.

**ÆTIOLGY.**—Intermittent fever belongs to the class of malarial or paroxysmal fevers. It is the most typical, and the most common of the class. The human system once subjected to the phenomena of a regular attack of ague in any of its forms, may be for the remainder of the life of the person so affected liable to a repetition of the attack, without his necessarily having been exposed afresh to the action of its cause. This tendency is very commonly shown in those who, after long residence in hot and malarial climates, are exposed to the influence of cold, and especially easterly winds, on their return to temperate climates in the springtime of the year. The more the person has suffered from the blood-changes and visceral degenerations described in the article **MALARIA**, the more prone is he

to such recurring attacks. Malarial fevers differ from most others in an important particular: they are not communicable from person to person.

**ANATOMICAL CHARACTERS.**—In the article MALARIA the pathology of intermittent fever is fully discussed, including the question of its parasitic origin. The spleen is enlarged; and in pernicious agues proving rapidly fatal, it is found greatly softened—often reduced to a state of deeply pigmented pulp. Death is common in malarious countries from rupture of the spleen, the result of blows or kicks, often of no great severity. The liver is usually somewhat congested and pigmented, and in cases of long standing it enlarges like the spleen, with the same increase in the connective tissue. In the algide cases hereafter described, when blood is driven in large quantity into the abdominal viscera, the digestive mucous membrane of the stomach and duodenum is congested and softened. The heart is invariably soft and flaccid, pale, sometimes of a dirty yellowish colour—degenerative changes induced by the combined action of diseased blood and high temperature.

**Blood-changes.**—Blood-changes of a remarkable kind are invariably present in all malarial fevers (see MALARIA), and often occur with astonishing rapidity. Army medical officers serving in the Mauritius during the epidemics of malarial fever which prevailed there some years ago, noted that the sufferers were often reduced to a state of complete anæmia in a few hours; the same is the case in Algeria. In such cases dropsical affections supervene, often rapidly, and military medical officers there record cases 'proving suddenly fatal from œdema of the glottis.' The blood is changed before any of the usual symptoms of an attack are present—it becomes dark in colour; the serum which separates has a dark-brown colour; and, when exposed to the air, the coagulum, which is large and loose, does not assume its usual bright-red colour. The white corpuscles are greatly increased in number; and the red corpuscles do not evince their usual tendency to run together in rouleaux.

**State of the urine.**—The urine contains a large amount of free acid, and retains a highly acid reaction for many days in the hottest weather. In the intervals it is often alkaline. When the paroxysms cease, the watery part of the urine diminishes; and it assumes a deep orange colour, depositing also an abundant sediment of urate of ammonium. This change is often observed by intelligent patients, who learn to appreciate its favourable significance. The late Dr. Parkes has shown that at the first elevation of temperature the urea increases; this lasts during the cold and hot stages; then it decreases, falling below the healthy average. Colin and other French authors note the enormous excretion

of urea in malarial fevers both in Italy and Algeria.

**VARIETIES AND SYMPTOMS.**—Three forms of ague have long been recognised—namely, the *quotidian*, which recurs in twenty-four hours; the *tertian*, in forty-eight hours; and the *quartan*, in seventy hours. The term *double tertian* is used when the paroxysm recurs regularly on alternate days, the attacks being alike in severity and duration. Other more rare forms of the irregular type have been described, as the *triple tertian*, with two paroxysms in one day, and one the next; the *double quartan*, with a regular fit one day, a slight one the next, and a complete intermission on the third day. The *quotidian* is the most common; the *quartan* the rarest of all—a rule which seems to hold good wherever malarial fevers prevail. The *quartan* type has been noted from early times for the tenacity with which it clings to its victims.

**Premonitory symptoms.**—These are much the same as in all febrile disorders—namely, pain in the back and lower extremities, languor, lassitude, gastric irritation, loss of appetite, nausea, and sometimes vomiting; occasionally with frequent calls to micturate, the urine being pale and highly acid. Then follow in succession the three stages, the *cold*, the *hot*, and the *sweating*; succeeded by what is technically known as the *interval* or *apyrexial* period, which lasts for a number of hours, varying according to the type of the disease.

**Cold stage.**—The patient experiences a sense of coldness in the back; then rigors set in, at first faintly, becoming quickly more distinct, until the teeth chatter, and the patient feels cold all over, and demands to have clothes heaped on him; the skin shrivels, the nails become blue, and he experiences a sensation of intense discomfort. This feeling of cold is, however, only a 'subjective symptom,' for if a clinical thermometer be placed in the mouth or rectum, even before distinct rigors set in, it will indicate a rise in the temperature of from two to three degrees. The skin, from contraction of the superficial vessels, is indeed colder than natural, but from the first the temperature of the blood is above the normal. The phenomena of the cold stage are gastric irritation, a foul tongue, a rapid pulse, and quickened respiration, with a feeling of coldness not confirmed by the thermometer. A sharp pain in the rectum is sometimes complained of just as the cold stage sets in; attributed by Dr. Lauder Brunton to obstruction to the flow of blood through the hæmorrhoidal veins.

**Hot stage.**—As this sets in, the patient grows warm all over, the face flushes, the pulse rises in frequency and volume, the skin grows hot, and the patient becomes restless, seeking ease to his aching head, back, and limbs in frequent change of posture. The tongue in this stage is usually dry, often bile-

tinted; and the bowels are constipated. The temperature rises to 105°, 106° F., and sometimes higher.

*Sweating stage.*—At first beads of perspiration appear on the brow and face, and the hands become moist; and soon, to the immense relief of the patient, his whole body sweats freely, the temperature begins to decline, and the paroxysm is at an end. The average duration of the paroxysm is from five to six hours, but in severe cases it may last for twelve hours. When sufferers have been exposed to malaria in such places as the mangrove-swamps of Africa, 'they will be reminded of what their systems have imbibed in the way of surroundings by the sickly scent of the swamps thrown off in the secretion through the skin' (Waller). Both officers and soldiers who came under the care of the writer on their return from the last expedition to the Gold Coast, observed the same fact in their own persons: some were severely nauseated by the unpleasant smell, recalling the stench of the places where the poison was taken into their systems.

*Temperature.*—The rapidity with which the temperature in ague rises to 105°, 106°, and sometimes even 107° F., and the equally sudden manner in which it falls when the sweating stage begins, is a very notable and characteristic fact of great diagnostic value. According to Wunderlich, nothing like this is to be seen in any other disease, with the exception of cases of relapse in typhoid, or the febrile paroxysms in acute tuberculosis and pyæmia. As soon as the sweating stage begins, the temperature declines, at first slowly, then as rapidly as it rose; when the deferescence is complete, it will be found one or two points of a degree below the normal, where it remains during the period of apyrexia. If the paroxysms be cut short by quinine, we may still detect at the hour of expected attack a distinct rise in the temperature, although none of the other symptoms of a paroxysm may occur, and the patient may be hardly sensible of it.

*Pernicious agues.*—This term is much used both by French and Italian authors, who apply it to cases both of intermittent and remittent fevers, in which certain symptoms are developed, such as delirium, coma, an algide condition—in a word, any serious complication placing the life of the patient in peril. So far at least as intermittent fever is concerned, this pernicious form appears to be more common in the intensely malarious regions of Italy than in India. Such was the experience of the writer, in whose large sphere of observation such grave complications were almost entirely confined to the worst forms of remittent fever, contracted in places notoriously dangerous. The writer is strongly impressed with the belief that the 'pernicious' symptoms, the mental incoherence and delirium, the coma, and the

'algide condition' so much dwelt on by some Italian physicians of the old school, were often the result, not so much of the disease as of the lowering treatment to which they subjected their patients.

The algide form of 'pernicious' intermittent is sometimes described by writers on the diseases of India as 'ague of adynamic type,' and it was frequently seen and well described by the French military surgeons both in Algeria and in Rome, during the long occupation of that city by the French army. The surface is cold, but, unlike the cold stage of an ordinary ague, the patient is unconscious of the low temperature of the surface. The internal temperature is high, and of this he is aware. There is an immense accumulation of blood in the abdominal viscera, with great thirst. The expression of the patient is tranquil, and his intelligence is intact. 'Il se sent mourir,' says Maillot, 'et l'abattement est tel qu'il se complait dans cet état de repos; sa physionomie est sans mobilité; l'impassibilité la plus grande est peinte sur le visage' (Maillot, Colin).

*COMPLICATIONS.*—Intermittents may be complicated by attacks of various diseases of greater or less severity—attacks often governed by climatic causes, by the habits of the individual patient, or by the fact that he has suffered from one or other of such diseases on previous occasions, such as pneumonia, bronchitis, asthma, dysentery, diarrhoea, or epilepsy. It is an old observation that the last-named disease, even in so-called 'confirmed epileptics,' sometimes disappears when the victim becomes the subject of an attack of ague. One very striking example of this kind the writer has seen.

Pneumonia is certainly the most formidable complication met with in intermittent fever. Invalids returning from India or from other hot and malarial climates to high latitudes, unless they are carefully protected by suitable clothing, are prone to suffer from this disease. The rapidity with which consolidation of the lungs takes place in such cases is very remarkable. It is not an uncommon thing to see five or six cases of this kind out of one party of invalids landed at Netley from India, if they have been exposed to cold weather on entering the Channel. If pneumonia is double, recovery is rare, the patients either sinking at once, or dying after a longer or shorter illness from pneumonic phthisis. Pneumonia of this type is a common and fatal disease among the malarial-poisoned civil population of Rome during the winter months; and the French military surgeons record its prevalence in the French garrison there at the same season, and also among malarial invalids sent back to France both from Algeria and Rome (*Recueil des Mémoires Méd. Mil.*, t. ii., 2<sup>e</sup> série, p. 268). When pneumonia occurs as a complication of any form of malarial fever, it is one of the

gravest import—a fact pointing to the necessity of careful exploration of the chest. Scorbutus is a formidable complication. It is common, although often overlooked, among European and native soldiers in India.

**DIAGNOSIS.**—An ordinary intermittent presents no difficulties. The well-marked nature of the paroxysms; the sudden rise of temperature and its equally sudden decline; the splenic enlargement; the discoloration of the skin; the urinary changes described above; together with considerations relating to the place where the disease was contracted; and, above all, the therapeutic test—that is, the power of quinine in preventing the recurrence of the attacks—ought to clear up all doubts.

**PROGNOSIS.**—This, in ordinary agues, so far as immediate danger is concerned, is highly favourable: death from uncomplicated ague is very rare. The direct mortality from ague, at all events among the European races affected by it, is small; the indirect mortality from the malarial cachexia, occurring either *per se* or as a complication of other diseases, is very great. In complicated or so-called pernicious agues, the prognosis will depend on the extent to which important organs are involved—cerebral, pulmonary, or gastric; much on the stage the disease has reached before the patient comes under treatment; and much, very much, on the nature of that treatment.

**TREATMENT.**—Keeping in view the fact that every paroxysm of intermittent fever, particularly in a hot climate, is a step, however short, on the road to the cachectic condition described in the article on MALARIA, the importance of breaking the recurrence of the paroxysms will be apparent. This, then, is the first indication of treatment; the second is hardly less important, namely, to improve the condition of the blood, and by judicious treatment—therapeutic, dietetic, and climatic—to prevent further degeneration of organs, and, so far as may be, to restore those affected to their normal condition.

If, as the writer firmly believes, there be such a thing as a specific disease, intermittent fever is specific. Like all such, it has a certain definite series of phenomena to pass through, which we cannot arrest. There is no drug known to science capable of arresting the stages of a true malarial fever once it has entered on the first or cold stage. This doctrine, as Sir William Jenner has admirably stated it in his *Address on the Etiology of Acute Specific Diseases*, has been taught by the writer, as regards malarial fevers and cholera, in his lectures at Netley for the last twenty years. There is little, therefore, to be said as to the treatment of the stages of ague: they must take their course, the only interference being to supply the patient with the warm covering so much

desired during the cold stage; if this be protracted unduly, to give him draughts of warm tea; and should symptoms of collapse appear in any of the more 'pernicious' forms at the end of the hot stage, to administer such restoratives and stimulants as the case may demand. Excepting in cases where the stomach is oppressed by a recently taken meal, the time-honoured practice of administering an emetic may be safely omitted. The lamented traveller Livingstone, whose experience of malarial fevers was immense, began the treatment of nearly all cases with the following combination. Of resin of jalap and of rhubarb, from 6 to 8 grains, with 4 grains of calomel and a like quantity of quinine. According to the great traveller and his hardly less experienced companion, the Rev. Horace Waller, this combination was found very efficacious as the commencement of treatment, both in intermittent and remittent fever. In Livingstone's camp his pills were known as 'rousers,' and as such were at once administered to men who, 'from premonitory symptoms, became idle and lethargic.' In about five hours copious dark-coloured motions followed; if these were delayed, recourse was had to a brisk purgative enema. Quinine was then given in 4-grain doses every four hours, until twelve grains were administered in the twelve hours succeeding the purgative medicine. Livingstone and his followers deemed any other mode of dealing with the fevers of Africa to be 'mere trifling.' Common sense points to the necessity of caution in the use of such active purging in men debilitated by disease or climate, or both, or when the patients are delicate women, or Asiatics, often calling for as delicate handling. It is very necessary to add that this sharp treatment is not applicable to those who are labouring, or have previously laboured, under dysentery, or any other form of bowel-complaint.

In the 'interval,' energetic efforts must be made to bring the patient under the influence of quinine. At once the most effective and the most economical plan is to administer quinine, in solution, in a 10-grain dose at the end of the sweating stage, and to repeat it in from four to six hours. At least a scruple of the remedy should be given during the interval. If obstinate vomiting interferes with the retention of the quinine, which will rarely happen if the bowels have been well relieved, the quinine must be administered either by enema or by the hypodermic method. The first plan is very efficacious, and is safe; the latter is more effective, but is not without risk of inconvenience from troublesome ulceration round the site of injection, if a mineral acid has been used to dissolve the quinine; this in urgent circumstances, such as in pernicious agues dangerous to life, or in remittents (as will be explained under the head of that type of fever),

might be disregarded were it not that tetanus followed the operation in five cases in one year in the Bengal Presidency, all of them proving fatal. In the face of such a fact, this operation, trivial as it seems, should not be performed on light grounds. The neutral sulphate of quinine, which dissolves freely in water at a temperature of 99° F., does not, so far as the writer's experience goes, cause ulceration. The syringe used for this purpose should be a little larger than that for injections of morphine, and should have a platinum hollow needle, which should be dipped in an antiseptic before insertion. In many cases, more particularly where there is much restlessness and disturbance of the nervous system, marked benefit follows the use of full doses of bromide of potassium with the quinine.

Quinine, in one or other of the methods advised, should be used until the paroxysms are broken. The remedy should be continued daily, so long as the clinical thermometer indicates a rise in temperature at the time of expected attack, even if there be no sign of a regular cold stage; and within a lunar month from the time of first attack the patient should be again brought under the influence of quinine for some days. In pernicious agues, or in cases where complications arise, it is in a high degree dangerous to pause in the administration of quinine, to use remedies of a depressing kind for this or that set of symptoms. Those who so act will have little success in practice until taught by bitter experience the danger of departing from the golden rule of trusting to quinine. In this way, epilepsy, pneumonia, asthma, bronchitis may have to be met, aided by stimulation of the skin, support from proper food, and stimulants when called for. For many years the writer has urged this doctrine on the attention of young practitioners, and he is glad to see that it is even more strongly insisted on by Trousseau in his well-known lectures.

It is in such cases, and in grave remittents, that the *tinctura Warburgi* has been found so useful as to warrant a strong recommendation. The active ingredient in this febrifuge is quinine, in combination with a variety of aromatic drugs, which either are now or were formerly official. It is a powerful sudorific, and has been found in the writer's hands, and in the practice of many medical officers in Southern India, a remedy of great power in all malarial fevers. After opening the bowels, half an ounce of tincture is administered, undiluted, all drink being withheld: a second dose is given in three hours. It soon produces free action of the skin, the perspiration often having an aromatic odour. It is rare to see another paroxysm follow the use of the tincture. In adynamic cases it should be used in smaller doses, and with some caution, lest its exces-

sive sudorific action should be too depressing.<sup>1</sup>

*Substitutes for quinine.*—The officers in charge of the Government cinchona plantations in India now prepare from the red cinchona bark, by a very simple and economical process, a preparation known as 'cinchona alkaloid,' which, in somewhat larger doses than quinine, is found to be effectual in checking malarial fever; but this preparation, although very cheap, has fallen into disrepute from the distressing nausea, and even vomiting, it often causes.

Salicylic acid is now largely used as an antipyretic in cases of ague.

Arsenic has been used for ages, particularly in the East, in the treatment of agues. There is no doubt that it possesses considerable power as a so-called antiperiodic. The French military surgeons, who are obliged to study economy, use it largely, and in doses much larger than are commonly given by British practitioners. They seek, as Boudin expresses it, to oppose an arsenical to a malarial diathesis. In the brow-aches and other neuralgic sequels of malarial fever it is an effective remedy, either alone or in combination with quinine. The power of arsenic as a prophylactic is much insisted on by Professor Crudeli. The alkaline sulphites, so much lauded by Professor Polli, have hitherto disappointed the expectations of British medical officers who have tried them. In the great epidemic outbreak of malarial fever in the Mauritius they were found useless. The sulphate of cinchonine in scruple doses is much praised by Dr. Paul Turner. Biberine was largely tried by the writer in India, and found to be inert. Of late, various preparations of *eucalyptus globulus*, the blue-gum tree of Australia, have been much praised in the cure of ague, and more particularly in the treatment of its sequelæ. The writer is inclined to fear that this remedy, like many others, has been unduly vaunted. It certainly is often useful in the malarial cachexia, with occasional attacks of ague, but in the acute forms of the disease it is, in the experience of the writer, of no value. The best form is the tincture. Both in France and Germany it is much used for the reduction of enlarged spleens.

*Diet.*—This should be nutritious and easy

<sup>1</sup> Dr. Warburg has communicated to the writer the formula for the preparation of this tincture, which at Dr. Warburg's desire was published in the *Lancet*, and *Medical Times and Gazette*. (See *Medical Times and Gazette*, 1875, vol. ii. p. 540.) As stated in the text, quinine proved to be the active ingredient, in combination with a number of aromatic drugs common to ancient and modern pharmacy. It is consistent with the writer's knowledge that this tincture has maintained its high reputation in the treatment of malarial fevers of the most dangerous type, in the hands of the late General Gordon, in the pestilential regions traversed by him and his officers while carrying out the policy of the Khedive of Egypt in the Soudan.

of digestion. Dr. Cornish has pointed out how much the mortality from malarial fevers is increased amongst the natives of India by 'starvation treatment.'

*Treatment of malarial cachexia.*—On the first signs of this condition appearing, the sufferer should be sent to a non-malarial climate. If by sea-voyage, care must be taken so to regulate the diet as to avoid the risk of ingrafting the scorbutic on the malarial cachexia. Remembering also the danger of exposure to cold already insisted on, scrupulous attention to clothing is a point of cardinal importance. According to the writer's experience, one of the most effective means of reviving the action of the skin, improving the condition of the blood, and restoring the spleen and liver to a more healthy condition, is to send those whose circumstances admit of it to Carlsbad or Homburg, where, under proper local medical advice, they may drink the waters and use the baths. The good effects of this treatment are often very marked and lasting. It should be supplemented by a course of the syrup of the triple phosphates of iron, quinine, and strychnine, in half-drachm doses three times a day; which after a time should give way to iron in some more direct form. The Carlsbad water, in combination with that of Friedrichshall, if continued for a sufficient length of time, is often most useful in improving the condition of the abdominal organs, even when the patient can only use them in this country; and the action of the skin may be stimulated with profit by the occasional use of a Turkish bath, or by a wet sheet packing. The writer has long used the ointment of the iodide of mercury in reducing enlarged spleens, and often with great success. The strength of the ointment is 13 grains to an ounce of lard. Of this a piece as large as a walnut is to be well rubbed in before a good fire. The process is repeated on the afternoon of the same day, and again, if need be, in a fortnight. If ordinary care be taken to watch the effects, and not to use the remedy too often, no ill consequences need be feared. The writer has in this way again and again reduced spleens, extending even into the pelvis, to almost normal dimensions, without producing any of the inconveniences either of the mercurial or iodine ingredients of the ointment.

It is a point of great importance that patients should be placed under the most favourable hygienic conditions, and breathe the purest air available.

W. C. MACLEAN.

**INTERNAL EAR, Diseases of.**—See EAR, Diseases of.

**INTERSTITIAL** (*inter*, between; and *sto*, I stand).—Relating to the interstices of an organ. The term is applied in physiology to the tissue which exists between the proper

elements of any structure, namely, some form of connective tissue. In pathology the word is used in connexion with *absorption*, when a part is gradually removed without any obvious breaking off; and also to indicate the implication of the interstitial tissues in morbid processes, or their infiltration with morbid products, as *interstitial pneumonia*, *interstitial hepatitis*, &c.

**INTERTRIGO** (*inter*, between; and *tero*, I rub).—DEFINITION.—A slight inflammation of the skin occurring in the hollows of folds of the integument or joints, where two surfaces lie in contact with each other.

ÆTIOLOGY.—The cause of intertrigo is not, as might be implied by its name, friction alone; but rather moisture and heat associated with contact and pressure, acting on a sensitive skin. In certain situations the amount of inflammation is liable to be aggravated by the addition of irritant discharges, such as excessive perspiration, urine, and faecal matter.

Intertrigo is common in infants, in whom it is favoured by abundance of integument, and sensitiveness of skin. For a similar reason it is met with in corpulent persons; but it is not wanting in the emaciated, when there exists a tendency to eczema, or an eczematous diathesis. In infants it is seen in the perineum, extending from the anal fossa behind to the groins in front, and likewise in any other of the deep folds of the integument. Among adults, in addition to these situations it occurs in the axillæ, in the groove beneath the mammæ, and in the flexures of joints.

DESCRIPTION.—The term 'intertrigo' points to a rubbing together or chafing, fretting or galling of the skin by friction, and no doubt friction may have some share in producing the inflammation; but it is also certain that intertrigo results most frequently from irritation caused by the heat and moisture of the part. Intertrigo has been adopted as an erythema under the name of *erythema intertrigo*, but it very rarely remains at the erythematous stage, having a natural tendency to run on to exudation with the discharge of a muco-purulent fluid, and to be further complicated with excoriations and chaps. In this condition it becomes an eczema, and is very properly treated as such. Indeed, it is more consistent with the genius of modern dermatology to consider it, even from the beginning, as an eczema, under the name of *eczema erythematosum*.

PROGNOSIS.—The prognosis of this affection is favourable as to cure, but uncertain as to time, and in adults it is very apt to degenerate into chronic eczema.

TREATMENT.—The removal of the cause is the first indication to be attended to in the treatment of intertrigo. This may be effected by keeping the folds of skin apart by

medicated cotton wool or old linen rag. The parts should be kept as dry and cool as possible, and dusted with fuller's earth, or any unirritating desiccative powder. Where powder is unsuitable, a lotion of lime-water inspissated with oxide of zinc will be found useful; and if this should prove irritating, zinc ointment, with the addition of a drachm of spirit of wine to the ounce, should be kept constantly applied. Where there is much exudation, it is desirable, as in eczema, to avoid ablution, and confine the treatment to wiping with a soft cloth previously to each repetition of the zinc ointment. Constitutional symptoms are rarely present in intertrigo, but, under any circumstances, the indications to be considered are regulation of the digestive organs and associated functions, a suitable diet, and tonic and antacid regimen.

ERASMUS WILSON.

### INTESTINAL OBSTRUCTION.—

SYNON.: *Ileus*; Fr. *Occlusion Intestinale*; Ger. *Darmverschliessung*.

DEFINITION.—Under this term are included all those cases in which the contents of the intestinal canal are obstructed in their onward passage, by causes or conditions occurring within the abdomen or pelvis. Cases in which obstruction is due to causes or conditions affecting protruding or protruded bowel are included under the head of HERNIA.

The subject of intestinal obstruction will be best treated by first discussing the condition in general, and afterwards its different varieties in detail.

ÆTIOLGY AND PATHOLOGY.—The causes of intestinal obstruction may be enumerated as follows, arranged as nearly as possible corresponding to the acuteness and urgency of the symptoms, and the imminence of danger to life to which they give rise. The relative frequency with which they occur may be approximately estimated by the numbers appended, representing the results of an analysis of 1,839 fatal cases.

1. **Strangulation by Bands or in Apertures** (440).—(a) By peritoneal bands, the results of previous inflammatory mischief, either under such bands, or by loops or knots, or in buttonhole-like slits, or by kinking caused by traction, or by the margins of slits and rings produced by the adhesion of organs to one another, or to some part of the parietes (219). (b) By the omentum or mesentery forming bands or slits. (c) By diverticula or diverticular cords, due to the persistence of the vitelline duct (Meckel's) or of the omphalo-mesenteric vessels, free or attached (75). (d) By the appendix vermiformis (42). (e) By the margins of peritoneal pouches (retro-peritoneal hernia, hernia through the foramen of Winslow, and other forms of internal hernia) (39). (f) By bands resulting from inflammation about the necks of old herniæ.

2. **Volvulus**.—In some cases the intestine is obstructed by being twisted on its mesenteric axis or even on its own axis. In others, the lumen of the intestine is closed by its being intertwined or knotted up together with another portion of the gut (106).

3. **Intussusception or Invagination** (537).

4. **Impaction of Gall-stones** (51).

5. **Contractions**.—Under this term are included those cases in which the bowel is gradually obstructed by matting together of its coils. This may be due to knitting together of one or more coils, by peritonitis, originating, for example, in inflammation of the mesenteric glands.

6. **Stricture**.—(a) Cicatricial, from injury or ulceration; (b) new-growths (373).

7. **Compression**.—By new-growths, hydratis, &c., outside the bowel (66).

8. **Impaction of Foreign Bodies, or Intestinal Concretions** (78).

9. **Impaction of Fæces. Confirmed Constipation** (78).

10. **Congenital Malformations**.

SEATS.—Some parts of the intestinal tract are very much more frequently affected by certain particular causes. Thus, acute obstruction by bands most frequently affects the small intestine; volvulus, the sigmoid flexure. Intussusception most frequently involves the cæcum and colon, and, next, the ileum. Constrictions due to new-growths are most common in the large intestine, especially in its lower portion. Impaction of gall-stones, with very few exceptions, occurs in the jejunum or some part of the ileum. Contractions and traction affect the small intestine and the large about equally.

SYMPTOMS AND SIGNS.—The symptoms and physical signs of intestinal obstruction are, as a rule, sufficiently constant and characteristic to establish the general diagnosis. But it is often very difficult, and sometimes impossible, to determine without exploration the differential diagnosis.

The symptoms and signs common to intestinal obstruction will be first given, and then, more briefly, those specially characteristic of each form.

(1) *Pain*.—Cases of acute obstruction by a band, volvulus, many intussusceptions, or impaction of a gall-stone, are usually signalled by acute pain, often 'doubling the patient up,' and sometimes producing faintness and collapse. Where there has been a period of incarceration, as in some cases of obstruction by bands or in slits, pain supervenes somewhat more gradually, though later it becomes equally severe. In stricture of the large intestine there is, in the earlier stages, comparatively little pain. Again, in obstruction of the large intestine by impaction of fæces the patient complains merely of fulness, weight, and discomfort.

At the onset the pain is doubtless due

to the injury immediately inflicted on the serous and mucous coats, and is very speedily increased and maintained by the effects of the congestion of the blood-vessels on the nerves. This pain will obviously be more severe when the small intestine, with its more intimate association with the nerve-centres and its larger vascular supply, is affected. But it must always be remembered that the severity of the pain depends on the abruptness and completeness of the obstruction, and the extent to which the intestine is involved, rather than upon the variety and cause of the obstruction. Somewhat later comes the pain associated with distension of the bowel itself; this is increased at intervals by paroxysmal exacerbations of acute suffering, which accompany the futile peristaltic efforts of the intestine. Instances of this pain are only too well seen in patients with thin abdomens, in whom attacks of peristalsis, especially when the muscular coat has become hypertrophied, recur after the taking of food, examination of the abdomen, the administration of an enema, &c. Other causes of pain are traction upon inflamed parts, especially when ulceration is beginning to set in; and, at a later stage, enteritis and peritonitis.

With regard to the constant or spasmodic nature of the pain,—when the obstruction is complete the pain is constant, with periodic exacerbations; but with an incomplete obstruction it is likely to be intermittent. An important point to consider is how far the pain affords any guide as to the situation of the obstruction. In the great majority of cases of acute intestinal obstruction, wherever may be the seat of the mischief, the pain is referred to the neighbourhood of the umbilicus, probably because near this spot lies the solar plexus. Thus, in a large number of cases the site of the pain is of little value as a guide. But there are certainly some cases, especially those of acute obstruction by bands, or sudden strangulation in a slit, in which the pain is referred to a region other than the umbilical. Where the pain is accompanied by the occasional uprising of a coil of intestine here, this spot will very likely be a guide to the seat of the mischief. The readiness with which pain may be masked by preparations of opium, and the dangerously fallacious appearance of improvement which may follow; the way in which the distinct localised suffering of obstruction may be merged and lost in the more general and diffused pain of peritonitis; and, finally, the cessation of pain which may occur at the close, when the nervous system is dulled by impending death—need only be alluded to here.

(2) *Tenderness*.—Both in acute and chronic obstruction, tenderness is usually absent until peritonitis has supervened. Patients in the early stages of acute, and still more in chronic obstruction, sometimes state that

they find relief from firm compression of the abdomen.

(3) *Vomiting*.—This is a very constant symptom of intestinal obstruction. It may commence almost simultaneously with the occurrence of the obstruction, in which case it is due to the shock of the injury inflicted. Both the vomiting which occurs a little later in acute obstruction, and that in chronic cases much more tardily, recur with greater or less severity, although with sometimes comparatively long intermissions. This vomiting, and its characteristic feature of being liable to become fæulent, have been differently explained. It is undecided how far the obstruction causes the usual peristalsis to be reversed (anti-peristalsis), or how far, aided by the distension of the bowel above and the glandular secretion poured out, it converts the usual passage of intestinal contents into two currents—one, peripheral, in the natural direction up to the point of obstruction, and the other central or axial, which pours the intestinal contents into the stomach. As a general rule, the higher the seat of obstruction, and the more acute the cause, the more severe is the sickness, and the more frequently does it recur. When the obstruction is high up in the small intestine, the vomit is usually bilious and offensively sour, but without the fætor of fæces. When vomit has been retained for any time, as when the patient is under the influence of opiates, it may have had time to decompose and to acquire a fæulent odour (Treves). Again, true stercoraceous vomit may come from the lower ileum as well as from the colon, for the contents of the lower ileum may, in the natural state, have all the characters of fæces. Moreover, great distension of the intestine may render the ileo-cæcal valve inefficient to prevent regurgitation from the large into the small intestine.

(4) *Constipation*.—This may be absolute from the first, even to the passage of flatus, in acute complete intestinal obstruction, as in that due to bands, apertures, volvulus, or gall-stone.

In other cases, where the strangulation is less abrupt and severe, or where the intestine by some peristaltic movements frees itself for a time, fæcal evacuations may be passed for a while; and a careful inspection of these, especially as to presence of bile, consistency, fæulent odour, &c., may give valuable information as to the degree and seat of the obstruction. In intussusceptions, especially in the more chronic cases, the bowel is rarely altogether impermeable at first, though later it becomes so from the effect of inflammatory swelling. In such cases there is a period in which small quantities of fæcal matter mixed with blood and mucus are passed, frequently with much tenesmus; later on, blood and mucus only are passed. In strictures of the bowel, constipation comes

about comparatively slowly, being here replaced by irregular actions, a tendency to diarrhoea alternating with one towards constipation, the motions being altered in shape and size, with passage of blood, &c. Later on, constipation may be rendered absolute by impaction of some undigested article of diet, or by a twist or kink of the bowel above the obstruction.

First among the causes of constipation comes occlusion of the bowel. Another very important one is reflex nerve action. Thus, in cases of acute strangulation, the constipation is often absolute from the very commencement, although the obstruction may be in the small intestine, and much fecal matter may still be lodged below the point of occlusion. Again, constipation is very usual in those cases of partial obstruction of the bowel where a segment is suddenly and severely nipped. This is well observed, as a rule, in Littre's hernia, where only a part of the circumference of the bowel is involved in the strangulation. Another cause of constipation which must be remembered is paralysis and exhaustion of the intestinal wall.

The following fallacies must be borne in mind. Even after complete occlusion has occurred there may remain in the bowel, below the seat of obstruction, some portion of its contents; and the secretions of the mucous membrane being added, the evacuation of these by natural effort, aided by enemata, may give rise to the false idea that the occlusion is not complete, or that relief has been obtained. Again, the bubbling away of an enema must not be mistaken for the passage of flatus. Finally, false hopes are sometimes raised by the passage of a stool shortly before death. If this has come from above the obstruction, it may be due to some sloughing which has taken place or relaxation of spasm.

(5) *Aspect and condition of the abdomen: Distension, swelling, meteorism.*—The degree of *distension* and its rate of onset vary with the cause and site of the obstruction, the amount of vomiting, the food taken, &c. Thus, in acute obstruction of the lower part of the ileum, distension comes on rapidly and severely; but an even more rapid and a severer distension is seen to follow on one form of obstruction of the large intestine, namely, volvulus. In obstruction high up in the ileum or jejunum, the epigastric and upper regions appear distended, and the lower ones sunk. But in trying to diagnose the site of the obstruction from the distension the greatest caution must be maintained. 'There is a difference in the shape of the abdomen when distended, according as the arch of the colon is below or above the seat of obstruction. In the former case the belly is rounded, projecting well forwards, but with comparatively little fullness of the lateral and lumbar region. In the latter case it is more broad, and, if the hand be placed on the patient's

loins as he lies in bed, a feeling of resistance is experienced which is wanting when the small intestine is alone distended' (Fagge). But the same writer gives us the following cautions in interpreting signs. Absence of fullness in the course of the colon is no absolute proof that the disease is high up in the intestine. Again, the presence of fullness in the course of the colon is no proof that the seat of the obstruction is in the large intestine. For in more than one instance a prominence of the epigastrium and the appearance of a large horizontal coil have been due, not to the transverse colon, but to the ileum, dilated until it rivalled the colon itself. The presence of meteorism is, when rapid and severe, most dangerous and distressing.

Apart from the above distension from accumulation of intestinal contents, certain localised swellings in the abdomen may be occasionally recognised. Thus, intussusception commonly gives rise to an elongated sausage-shaped swelling lying in the course of the large intestine. Stricture of the sigmoid may sometimes be felt in a patient with a thin, relaxed abdominal wall, as an indistinct localised thickening. Other instances of tumour are very rarely met with. The fat-loaded abdominal walls usually met with in a sufferer from impacted gall-stone prevents this cause of obstruction being felt by palpation. A localised swelling will be of more value as a guide if pain has also been referred definitely to this same particular spot. A number of coils of small intestine, matted together by adhesion, may form a kind of tumour, and localised dullness on percussion has been caused by collapsed coils of small intestine grouped together below an obstruction (Treves).

(6) *Peristalsis.*—The fact that the movements of the intestine can be clearly recognised in some cases may be of much help in the diagnosis. One coil may be seen rising up and becoming prominent, and then sinking down and giving place to another, and sometimes waves of action, as it were, seem to pass along a considerable length of bowel. Such movements recur at irregular intervals, and are accompanied by gurgling noises (borborygmi) and sensations, and by exacerbation of suffering. This peristalsis, as Dr. Fagge pointed out, is characteristic of the chronic varieties of obstruction, in which the coats of the bowel have undergone hypertrophy. It also indicates an absence of peritonitis. While peristaltic movements are much more often seen in the small than in the large intestine, they may sometimes be observed in the latter. Dr. Fagge also pointed out, as worthy of especial note, that the large intestine, when distended, does not continue to lie horizontally across the upper part of the abdomen, but bends downwards, and may form a broad loop, lying vertically and (with

the dilated ascending and descending colon) filling the whole front of the abdomen. On the other hand, the coils of the ileum are, under similar circumstances, generally arranged transversely; and, as these coils are often quite as broad as the transverse colon, the uppermost one, lying horizontally just below the ribs, may easily be mistaken for that part of the large intestine.

(7) *Diminution of urine.*—In acute obstruction the urine is diminished in quantity. This fact has been differently explained. The late Dr. Barlow considered that it pointed to a site of obstruction high up in the jejunum, the area for the absorption of fluid being thus much diminished. Later on, Dr. Habershon attributed it to the urgency of the vomiting which occurs in acute obstruction high up. Dr. Fagge, following Dr. Sedgwick, argued that this symptom is merely one of the phenomena of collapse, and will occur in all forms of intestinal obstruction, whatever their seat, in which collapse is present. But as it is obstruction of the small intestine in which collapse is often present, while in that of the large bowel it seldom occurs, suppression or diminution of urine is common in the former, rare in the latter, condition. When the obstruction is low down there is no marked diminution in the quantity—there may even be an excess—of limpid urine, with sometimes a difficulty in voiding it.

(8) *General aspect and symptoms.*—In cases of acute obstruction, often from the first, and in the later stages of both acute and chronic obstruction, the general aspect of the patient is more or less characteristic. A condition of collapse frequently occurs; and when this appears *early* it is due to the shock of the injury, and is usually proportionate to the nerves and the amount of intestine involved, being more marked in the case of the small than of the large intestine; and, above all, to the abruptness and severity of the obstruction. The countenance is expressive of anxiety and distress; the eyes are sunk, often with dark circles round them, the nose pinched, the cheeks hollow, the lips pale or purplish, and the complexion faintly livid; the general surface is cool or cold (the ears, forehead, nose, and feet first becoming cold), and often covered with a clammy perspiration. Although the mental faculties are as a rule undisturbed, there is a disposition to torpor, from which the sufferer is from time to time aroused by exacerbations of pain, or recurrence of vomiting. The pulse is frequent and small, and towards the end becomes thready. The temperature is rarely above normal unless peritonitis sets in; usually it is normal or subnormal, sometimes falling to a marked extent. Sometimes there is more or less dyspnoea, which may be explained in part by pressure upwards of the diaphragm by the distended bowel below, and the latter cause often gives rise to distressing

hiccough. The voice varies, being feeble or whispering, or strong and full quite up to the end. The tongue becomes red, and later dry and brown. Towards the close there is often a combined odour of offensive sweetness and decomposition in the breath. The patient suffers from severe and constant thirst, which often he fears to assuage lest vomiting should be provoked.

In most cases the course is one of progressive emaciation, weakness, and exhaustion, the end being often preceded by peritonitis. In some exceptional cases death has been preceded by mild delirium and coma; and in others, still more exceptional, by violent delirium and convulsions, the latter being especially likely to be met with in little children suffering from intussusception.

*COURSE, COMPLICATIONS, AND TERMINATIONS.*—In the acute forms of intestinal obstruction the prospect of recovery is extremely slight; perhaps, in the large majority of cases, it would be just to say that practically there is none at all. And here a word must be said as to those extremely rare instances of spontaneous recovery which have occurred from time to time under every form of acute obstruction, volvulus only excepted. Very much of the value of these cases has been lost by their after-histories not being published, chances thus being lost of our knowing in what cases of certainly mechanical obstruction spontaneous recovery is really possible. In the second place there is strong reason to believe that in several of these cases the diagnosis has been at fault, the real cause of mischief being peritonitis, *e.g.* starting from the cæcum or its appendix, or one originating in tubercular mischief, an enteritis, and not one of mechanical obstruction.

In the more chronic forms of obstruction, with the exception of those depending upon impaction of fæces, and some others, perhaps (as chronic intussusception), death, though considerably longer delayed, ensues sooner or later, in spite of temporary relief afforded by operative measures or in other ways.

In cases of acute strangulation, in which the occlusion has been sudden and complete from the first, and in which timely relief has not been obtained, the average duration has been found to be from three or four to five or six days. In the cases reported as fatal within a few hours, it is possible that the case has been one of perforation of the intestine or stomach, rather than one of obstruction of the bowels (Fagge). Where the case is one of incarceration at first, followed by actual strangulation, death may not occur until after the lapse of eight or ten days. In cases of constriction or compression in which complete occlusion has come about gradually, or even has not been finally established, the duration of life varies greatly according to the complications which arise, and it may

be prolonged for weeks or even months. So, too, in cases of stricture of the large intestine, where the patients are of average intelligence, when they avail themselves regularly of medical supervision, and are amenable to the directions given, especially in the matter of diet, the end may be deferred for one and a-half or two years or more.

The *complications* and *accidents* that are liable to occur are: Peritonitis starting from the seat of the lesion, and more or less rapidly spreading and becoming general; enteritis; ulceration and perforation of the bowel; sloughing of the strangulated or intussuscepted portions; hæmorrhage into the peritoneal cavity or into the bowel; sudden asphyxia or broncho-pneumonia, due to the entrance of vomit into the air-passages; pyæmia or septicæmia from the absorption of decomposing material; gradual asphyxia from the interference with respiration by abdominal distension; anæmia; coma; and syncope from cardiac depression and cerebral anæmia.

**DIAGNOSIS.**—Nowhere does successful treatment depend more intimately on correctness of diagnosis; nowhere is a correct diagnosis more invariably difficult; nowhere is it so often impossible. We can only hope to arrive at it by careful attention to the following points, and by striving to keep before us a mental picture of the different hidden possibilities which may lie at the root of the mischief.

The following questions must be considered: I. *Is the obstruction acute or chronic?* II. *Where is the obstruction situated?* III. *What are the best methods of investigation?* IV. *What are the diseases most likely to be mistaken for intestinal obstruction?* V. *Is the case one favourable or unfavourable for operation?* This last question will be considered later under **TREATMENT**.

I. *Is the obstruction acute or chronic?* The symptoms given as distinctive of the two groups are often misleading when applied to individual cases.

(1) Under *acute obstruction* would be included the following: (a) Strangulation by bands or through apertures; (b) volvulus of the colon; (c) acute intussusception; (d) impaction of a gall-stone in the small intestine.

**SYMPTOMS.**—Pain sets in suddenly at a definite time, being generally referred to the neighbourhood of the umbilicus, whether agonising or colicky; it is usually paroxysmal at first, but tends to become continuous. It may have followed an unwise meal, going to work, some straining effort, active purgation, or a blow. Vomiting is early and severe—first the contents of the stomach—then bilious, excited by any effort to take food—and ultimately brownish, foul-smelling, or more completely stercoraceous. Constipation is absolute, even to the passage of

flatus, though small scybalous matter may come away from a point below the obstruction after enemata or straining efforts of the patient. The abdomen soon becomes distended, but its form presents little that is really distinctive of one form of obstruction rather than another; this being largely due to the facility with which one or two distended coils may be displaced into areas where they conceal the proper contents, and simulate their distension. The urine is diminished or suppressed. The collapse, the state of the tongue, pulse, and temperature are as already stated.

(2) Under *chronic obstruction* would come: (a) Stricture of the large intestine; (b) contractions or adhesions obstructing the intestine (usually small) by bending, dragging on, narrowing, or matting up one or more coils; (c) stricture of the small intestine after ulceration—traumatic, tubercular, or malignant; (d) chronic intussusception; (e) obstruction by compression of growths from outside; (f) faecal impaction.

**SYMPTOMS.**—In chronic obstruction these are characterised by their slow, irregular development. The pain comes on at first at long intervals, corresponding to troublesome attacks of constipation, and is put down to 'attacks of wind,' colic, or indigestion. Even late in the case fixed pain is often absent; but pain occurs in paroxysms which correspond to attacks of peristalsis, showing the writhing movements of intestine, with muscular fibre hypertrophied, and as yet not stilled by peritonitis. Constipation is for some time the chief trouble: the bowels never act satisfactorily without aperients. Constipation alternates with diarrhœa; or broken up or narrowed bits, unsatisfactory alike in form and bulk, are passed with difficulty, accompanied by much flatus and some loose motion. After one or more attacks of obstruction more or less complete, the contracted lumen of the bowel may be absolutely blocked. Vomiting is often long deferred, and the patient may keep comparatively well (with clean tongue, sleeping well, and taking food), even when his condition has become one of great danger.

While the above description applies to well-marked acute and chronic obstruction respectively, the two forms do not always fall into such distinct groups. Further, the above terms do not always correspond to obstruction of the small and obstruction of the large intestine. All the symptoms of chronic obstruction, lasting over six months, may be presented by a case of obstruction of the ileum from cancerous puckering of its mesentery; while, on the other hand, every symptom of the acutest obstruction of the small intestine, ending fatally in four days, may be shown by a volvulus of the sigmoid (Fagge). Again, the greatest difficulty in deciding between acute and chronic obstruc-

tion may be caused by a patient, whose age suggests malignant stricture of the large intestine, and in whom absolute constipation, a greatly distended belly, and evident peristalsis call loudly for relief, definitely declaring that the constipation is merely a matter of a few days, and denying absolutely any previous attacks of constipation or obstruction. Finally, there is a third class in which an attack of acute obstruction supervenes upon symptoms indicating a chronic obstruction. Such condition most frequently occurs in patients with malignant stricture, or with coils matted together by adhesions, with chronic intussusception, or gradual occlusion of the lumen of the bowel by a growth within or outside its walls, the actual obstruction being brought about by some sudden blocking, as by a faecal mass, or by kinking or bending of the bowel above. The chief help in the diagnosis is given by examination of the patient's history. There will usually be an account of symptoms pointing to chronic obstruction, with previous less severe attacks.

II. *Where is the obstruction situated?*—It may be said at once that a case of chronic obstruction is usually one of the following: (a) Stricture in rectum, sigmoid flexure, splenic or hepatic flexure; (b) stricture of the small intestine from mischief within its lumen, or from matting, kinking, or dragging on it by adhesions, &c., outside; (c) chronic intussusception; (d) faecal impaction. Of the above, obstruction of some point in the left side of the arch of the large intestine is by far the most frequent.

With regard to acute obstruction, usually in the small intestine, it must be confessed that it is very frequently quite impossible to go beyond localising it in the small intestine. It has been shown that the urgency of the chief symptoms is not related to the seat of the obstruction, but to its severity. No doubt obstruction high up in the small intestine will in many cases be accompanied by earlier vomiting, but less likely to become faeculent. Distension of the abdomen will be less in a case like the above than in one where the obstruction is low down in the ileum. The numerous fallacies which may beset us, depending on the amount of food taken, the influence of drugs, the erratic manner in which one or two coils may cause distension of an area at some distance from their proper habitat, must be borne in mind. It will be wiser to be content in acute obstruction with determining whether it is in the small intestine, as by a band; in the large, as in volvulus; or in both, as an acute intussusception—and to remember that all beyond may be blind guesswork.

III. *What are the best methods of investigation?*—A correct diagnosis can often only be arrived at by most careful attention to the following points. Trivial as some may

seem, all or any may be of the utmost importance; and as cases of acute obstruction may vary every hour, carefully repeated examinations may be needful to throw light on a condition which was quite obscure a few hours before.

(a) *The History.*—The value of this is often nil, but the smallest point may sometimes be of the greatest importance. Thus (i) *previous peritonitis* may lead to a suspicion of strangulation by a band, or by multiple adhesions. (ii) *Injury*, such as a kick from a horse, may have led to a rupture and subsequent hole in the omentum, thus causing later on a seat of strangulation; or to obstruction from cicatrisation of some bruised loop of intestine. (iii) *An imprudent meal* of heavy and indigestible food may determine the occurrence of obstruction, by the sudden formation of gas and turbulent peristalsis in cases of peritoneal adhesions, or stricture, by causing twisting or kinking. (iv) *Old-standing hernia.* Adhesions formed about the neck of an inguinal hernia may strangle a loop of small intestine quite independent of the hernia.

(b) *The Symptoms.*—These are next to be investigated. The symptoms characteristic of acute and chronic obstruction respectively, and their fallacies, have been already given.

(c) *Physical Examination.*—The most careful and thorough examination of the abdomen by palpation and percussion, and by exploration of the rings, the rectum, and, if need be, the vagina, should be instituted at the earliest practicable period, before general abdominal distension or the supervention of complications has obscured the indications first presented. The form of the abdomen is to be first studied. Then, any fulness which remains localised, especially if accompanied by abiding pain in the same place, any limited dulness, any repeated upspringing of one or more coils at the same place (especially if it is noticed that borborygmi are arrested here), may all be of value in helping to localise the seat of obstruction. In some cases it may be desirable to administer an anæsthetic, to facilitate the examination by relaxing the abdominal muscles, and to save additional suffering. But this step may be dangerous if vomiting is easily excited. With regard to the rings, the regions of umbilical, inguinal, femoral, and even obturator hernia should be carefully scrutinised. Not only may the modesty of a patient have led to the concealment of an ordinary hernia, but possibly a small knuckle may be tightly strangled in the neighbourhood of the internal ring, or in the canal, or the lumen of a piece of intestine may be partially closed in a tiny femoral hernia. As to the need of a rectal examination, it is only too certain that even nowadays this method of examination is still too much neglected, and thus intussusceptions are sometimes overlooked until

it is too late to reduce them by insufflation; and cases of malignant stricture are allowed to go on, and treated as indigestion, flatulence, or constipation. The introduction of the whole hand into the rectum has been rightly given up of late years; the information it gives is very limited, and only obtained at grave risk. Exploration *per vaginam* is often no less needful than that *per rectum*.

(d) *Use of Enemata and the Long Tube.*—The employment of these methods is often as useless in diagnosis as in treatment. Further, their use is often accompanied by danger, and may be misleading also. Finally, they frequently weaken and distress the already sufficiently handicapped patient. The following dangers and fallacies accompany their use: (i) An obstruction may be pervious to fluid from below and not from above (Fagge). In such a case the fluid pumped through may easily add to the accumulation above this point. (ii) When the abdomen is distended, the administration of large enemata must add to the misery of the patient. (iii) Further, the passage of an enema tube and the amount that can be injected may be misleading also. Thus, it is well known that the tube may be stopped by a fold of mucous membrane; or it may double on itself, thus leading to the belief that no obstruction exists within reach. Dr. Brinton thought that with a maximum injection of a pint the obstruction might be referred to a point not lower than the upper end of the rectum; one and a-half, two, and three pints belonging to corresponding segments of the sigmoid flexure. But it is obvious that the sources of fallacy—leakage, admission of air, the varying capacity of the rectum in different patients, &c.—are numerous. The use of enemata will again be alluded to under TREATMENT.

IV. *What are the diseases most likely to be mistaken for intestinal obstruction?*—Of these, the most important in frequency and gravity is peritonitis—especially that form due to mischief in the cæcum or the appendix, or a peritonitis of tubercular origin. Pain, vomiting, constipation, and tenderness may all be present and lead to the greatest difficulty in diagnosis. The points most likely to help are the following: (1) *The previous history.* Thus, though the onset may appear to be sudden, there will very likely have been attacks of 'stomach-ache,' irregular action of the bowels, and tenderness in the right iliac region. (2) *The temperature.* This is usually high in acute peritonitis; but, unless the case is seen early, it may be, as in intestinal obstruction, inclined to subnormal if prostration or collapse have set in. (3) *The vomiting* is usually less urgent in peritonitis, and more rarely feculent. (4) *The constipation* is less absolute. (5) There is usually diffused *tenderness and distension* in peritonitis. (6) There is no *peristalsis*. (7) *The abdomen* in peritonitis is often 'smooth, firm,

and barrel-like' (Hutchinson). Other conditions occasionally simulating intestinal obstruction are lead colic, hepatic or renal colic, enteritis: conditions bringing about an arrest of action but not mechanical obstruction, and to be distinguished by the history and progress of the case.

TREATMENT.—Methods of treatment will be first described, as tentative measures for relief are often called for before any definite conclusion as to the precise cause of the trouble can be arrived at.

A. NON-OPERATIVE TREATMENT.—1. *Purgatives.*—As a rule almost without exception, in acute or subacute cases, the use of all kinds of purgatives is to be emphatically condemned. They aggravate symptoms; while they are liable to light up quiescent into acute trouble, and convert chronic mischief into acute. Threatening sloughing or perforation may also be rendered imminent. It is only where all urgent symptoms have subsided, or, in more chronic cases, where there is no evidence of peristalsis or of peritonitis, that mild laxatives, especially some salines, often prove of great value, but great caution is necessary in their administration. These points, together with the use of stronger aperients in obstruction from fecal impaction, will be alluded to later.

2. *Sedatives.*—Opium and belladonna, conjointly or separately, are the most useful and reliable medicines we possess, as in the usual combination of opium gr.  $\frac{1}{2}$ —gr. 1, and extract of belladonna gr.  $\frac{1}{8}$ —gr.  $\frac{1}{4}$ , every three or four hours. But it must be remembered (1) that our knowledge of the action of these drugs on the inhibitory and accelerating nerves of the intestine is by no means complete. (2) That we are here especially in the dark. Thus, if we could tell the nature of the obstruction, how the coils lay, and which of them were capable of usefully directed peristaltic action, we might usefully employ belladonna in large doses. In other cases, where ulceration might be threatening, opium alone would be employed. (3) That while opium undoubtedly allays the pain, checks violent and hurtful peristaltic movements of the intestines, dulls the acuteness of the patient's mind constantly alive to his anxieties and distress, while it promotes sleep and thus husband his strength, it is an entire mistake to speak of opium by the often-quoted phrase 'as our sheet-anchor' in intestinal obstruction. It may dangerously modify symptoms and signs; thus, a patient drugged with opium may be quite unaware that the tenderness of his abdomen is increasing. It may apparently so improve the condition that an operation, later on proved beyond a shadow of a doubt to have been the patient's only hope, is put off till it is too late. In other words, while symptoms are lessened, the condition of the intestine may be hourly getting worse. (4) Adult

patients often show remarkable susceptibility to belladonna; and the excitement which may follow an over-use of this drug in these cases may be most harmful to the patient.

(5) In administering opium and belladonna we must be content with the smallest possible doses, especially of the former. In other words, the patient must be judiciously prescribed for, and not drugged. Later on, when operation is declined or set aside, it may be needful to give opium in larger doses, especially if peritonitis be present. (6) There is a certain class of adult cases of intestinal obstruction in which belladonna, in much larger doses than are usually tried, has been most successful. Thus, in more chronic cases, one or two grains of extract given every hour till from five to fourteen grains have been taken, have been followed by relief (Kerr, *Brit. Med. Journ.*, ii. 1878, p. 307).

Opium and belladonna should be administered by the mouth in the absence of contra-indication; but, if vomiting is severe and frequent, by subcutaneous injection of morphine and atropine. Their administration by suppository is the least satisfactory of all methods in cases of acute obstruction. In a few very acute and painful cases the inhalation of an anæsthetic may be asked for; it must always be remembered that this course, while temporarily relieving suffering, and perhaps enabling the case to be further cleared up, has certain inherent dangers of its own.

3. *Diet.*—Sufficient care is still but seldom taken in this simple matter, and the patient is allowed to add needlessly to his misery and danger. In acute obstruction, whenever the diagnosis is suspected or established, it is much best to cease feeding altogether by the mouth, and to trust to nutrient enemata. The small intestine and the stomach itself are liable to be gradually filled up by an ascending column of fluid, and vomiting is thus constantly impending; and when it has already set in, it is only made worse by giving food by the mouth. As a rule, nothing whatever should be given by the mouth save a little ice in small cracked pieces. Nutrient enemata may be made on some such plan as—milk ʒij, brandy or port wine ʒss., yolk of one egg, with liquor pancreaticus (Benger) ʒj, or a few drops of diluted hydrochloric acid and a little pepsin—the whole being gently warmed, but not cooked. Simple enemata of strong beef-tea, only just liquid, may be given every two, three, or four hours. After, or in place of either, a nutrient ymised suppository may be inserted. Before an enema is given, it must be ascertained that the rectum is empty. Thirst is best met by the use of ice and the frequent moistening of the patient's lips. Large liquid enemata sometimes relieve thirst markedly, but the objections to these have already been given. When it is clear from the failing pulse, the falling temperature, the cold ex-

trimities, the dry tongue, and most fetid breath that trusting to nutrient enemata is not sufficient, limited quantities of champagne, brandy and soda-water, or some kind of meat juice must be given by the mouth.

4. *Enemata.*—In intussusception and fæcal impaction, and in cases of stricture with a fæcal block above, large enemata are invaluable; but with these exceptions, and especially in all acute cases of obstruction, they are very rarely useful, and are only to be employed with the very greatest caution.

5. *Local applications.*—Material relief is often given by the assiduous use of quite hot applications, regularly renewed, on lint or flannel, wrung out of very hot water, to which preparations of opium, belladonna, and conium or hyoscyamus have been added. Poultrices are too heavy, and quickly get cold. Turpentine is sometimes applied on hot flannels. It has the objections of frequently slightly blistering the skin, thus rendering it tender for examination, and perhaps interfering later with the incision of an operation. In a few cases the patient experiences greater relief from local cold, as by cracked ice applied at frequent intervals between two layers of flannel tacked together with a few stitches.

6. *Electricity.*—This is occasionally of service in fæcal impaction.

7. *Abdominal taxis.*—This method has been strongly advocated by Mr. Hutchinson. He believes it to be a fact that the conditions in the early stages are such as to admit of the very easiest replacement. 'The first point is the full use of an anæsthetic so as to obliterate all muscular resistance. Next (the bowels and bladder being supposed to be empty) the surgeon will forcibly and repeatedly knead the abdomen, pressing its contents vigorously upward, downward, and from side to side. The patient is now to be turned on to his abdomen, and in this position to be held up by four strong men, and shaken backwards and forwards. This done, the trunk is to be held feet uppermost, and shaking again practised directly upwards and downwards. Whilst in this inverted position, copious enemata are to be given. The whole proceeding is to be carried out in a *bonâ fide* and energetic manner.'

Abdominal taxis to the extent just described has not hitherto recommended itself to the majority of English surgeons. To many minds conditions will occur in which this procedure, practised, as must be the case, with a certain amount of violence, though carefully and skilfully, will do harm and not good. Thus, if it does not release a loop, it may quite possibly drive one farther on, or send more than one beneath the obstruction. Those who, like the writers, believe that in many of the acuter cases softening and commencing sloughing have very early set in at the seat of strangulation, may well hesitate to recommend this method. Finally, it must be most

strongly insisted on that it is only in the very early stages of acute obstruction that this method of abdominal taxis can generally be safely used; and that evidence of peritonitis, a condition notoriously difficult of diagnosis, absolutely contra-indicates it.

#### B. OPERATIVE TREATMENT.

**Surgical Operations.**—These include—(1) *Abdominal section*; (2) *Puncture*; (3) *Colotomy*. Of these, colotomy will be referred to under the treatment of chronic obstruction.

1. *Abdominal Section.*—The advisability of this procedure is a matter which concerns physicians as well as surgeons, and especially the practitioner who is called upon to meet one of these most grave cases single-handed.

A few most important points, many of them fallacies still unrecognised, first call for attention: (1) Acute and chronic obstructions do not always fall into distinct groups. (2) Chronic obstruction often puts on an acute appearance. (3) The demand frequently put forward at the present day that cases of acute intestinal obstruction are surgical only, is a mistake. Not only are there difficulties in distinguishing between acute and chronic obstruction, but the existence of obstruction at all may be questioned. The possibility of typhlitis, peritonitis, or enteritis being at the root of the mischief has been already alluded to. But it is right that a surgeon should be called in as early as possible, not because he will desire to operate early in every case, but because it is only fair that he should see the case early and throughout. If the time should come when an operation is decided upon, the surgeon will be in a far better position than when called in, as too often happens, late in the case. (4) It is probably a mistake to expect a large proportion of successes, even after early operations for intestinal obstruction. Many leading surgeons have held strongly that the chief cause of the great mortality after operations for intestinal obstruction has been in their having been put off too long. The mistake here consists in grouping all cases of acute intestinal obstruction together as all suitable for operation. Now *post-mortem* evidence shows that a large proportion are from the first hopeless of relief by operation, others becoming hopeless so early in the case as to render any operation futile. Thus, with regard to operative treatment, cases of acute intestinal obstruction may be divided into these three groups: (a) *Cases probably hopeless from the first*—e.g. some cases of severe volvulus of sigmoid and colon; some cases of acute intussusception in infants; and cases of complicated snaring or matting, as in peritonitis started about the female pelvic organs, or in inflammation around mesenteric glands. (b) *Cases in which there is hope of doing good by operation*—e.g. cases of band, especially the single band in young subjects; some cases of intussusception, and of internal

hernia. The question of benefit from operation in acute obstruction is intimately bound up with these two points: (1) the impossibility of making an accurate diagnosis of the nature or the site of the obstruction in many cases; and (2) the peculiar structure of the parts; thus, the intimate association of the intestine with the nervous system, the weakness and easy paralysis of its propelling power, and the readiness with which it is strangled, &c. Finally, amongst the conditions which handicap the explorer are (a) the tightness with which his hand is gripped by the abdominal wall here, not stretched as by a large ovarian tumour; (b) the way in which distended coils of intestine crowd up and get in the way; (c) the risk of fatally disturbing parts already known to be damaged; (d) the condition of the patient. If an operation is to be performed, the pulse should be fair, the temperature not falling, the abdomen not much distended, and with sufficient peristalsis to make it probable that there is little or no peritonitis. On the other hand, the operation should not be performed, as it too often is, on the merest chance of relief, when the abdomen is enormously tympanitic, when the temperature and pulse are falling, and the patient passing into irrecoverable collapse.

*The Operation.*—Only the chief points can be noted here. The bladder being emptied, and the abdominal wall shaved and cleansed, the patient, with his extremities warmly clad, is placed on a water-bed filled with hot water. Everything that can possibly come within the field of operation must be scrupulously cleansed before the operation, and kept aseptic. An anæsthetic being given, the abdomen is quickly opened along the linea alba below the umbilicus; and all hæmorrhage being arrested, two or three fingers are introduced, and the following possible sites of strangulation first looked to: (1) The cæcum, its condition of distension or emptiness telling whether the obstruction is below or above it. (2) The inner aspects of the femoral, inguinal, and obturator apertures, to make sure that no tiny hernia exists, imperceptible from the outside. (3) The brim of the pelvis, as bands of omentum are often fixed hereabouts, and also because, in women, local peritonitis, originating in the uterus or its appendages, is not infrequently the source of the obstruction. If the above search with two or three fingers or the whole hand fails, and it often will when distension is present, embarrassing the fingers in their movements and obscuring the relation of parts, one or two loops which lie nearest the wound should be scrutinised and followed in the direction of increasing congestion and distension, thus leading to the obstruction. If this prove fruitless, the most distended portion of the intestine must be drawn out (under cover of a new towel, wrung out of hot carbolic acid solution 1 in 60, or of boric acid 2 per cent.) bit by bit from the

upper part of the wound and passed in again, after examination, into the lower angle, in such a way that at no time are more than five or six inches of intestine exposed. After drawing out and replacing some feet of intestine in this way, it is probable that, owing to the increasing congestion or resistance, the surgeon will reach the obstruction. An assistant should hold the coil from which the surgeon starts under a hot sponge, to prevent the same ground being traversed a second time. If after five to ten minutes' search the mischief is not found, the surgeon must decide between making an artificial anus and closing the wound, or allowing all the coils to prolapse under hot aseptic towels, frequently renewed. The decision must turn on the condition of the patient, and the amount of distension, it being exceedingly difficult to get distended coils back into their home, and the needful manipulations necessarily producing much shock, and perhaps inflicting serious damage. When the obstruction is found, it must be dealt with according to its nature; thus an opening (as in the mesentery) must be stretched with the finger-nail and the bowel withdrawn; omental bands, if not torn through, divided between two chromic gut ligatures; any diverticular band brought up into view, and divided with similar precautions, or, if the patient's condition admits of it, resected and the cut ends cleansed and closed. Intestinal anastomosis, by Senn's plates, or Paul's modification, will be resorted to where the choice lies between resection and making an artificial anus. The best mode of dealing with volvulus, intussusception, and gall-stone, are given under their respective headings. The peritoneal cavity must next be cleansed, if needful, either by sponges or by flushing with a hot solution of boric acid or salicylic acid ( $\frac{1}{2}$  per cent.), or boiled water, introduced by an irrigating tube. In these cases a Keith's tube should be introduced.

**1a. Formation of an Artificial Anus; Enterotomy.**—This is done under two chief conditions: (a) When the operator cannot detect the site of obstruction, or finds that he can neither deal with this nor perform resection, he then makes an artificial anus in the middle line. (b) When it is decided, owing to the patient's condition, not to perform an ordinary abdominal section, but to relieve the distension, &c., by opening the bowel above the obstruction, Nélaton's operation is usually adopted. Neither is more than palliative; and neither should be resorted to, save when other steps fail or are impracticable, and when it is certain that the obstruction is low down in the small intestine. The chief drawback to this method lies in the extreme annoyance the patient suffers afterwards from the continual escape of liquid fæces. If when proposing to make an artificial anus it is absolutely needful to open the bowel at once,

this may be effected by a small sharp trocar, the cannula being left in and plugged, or by making a small incision in the gut, turning the patient on one side, and washing away the escaping fæces with boiled water.

**2. Puncture of a Distended Coil by Trocar and Cannula.**—This method is still more merely palliative; it is done still more in the dark; and, though it has given marked relief in some cases, it is not without serious risks. While in a very few cases puncture has apparently proved curative, there is no doubt whatever that the number of cases in which this method has been fruitlessly employed, and which have never been published, is infinitely greater. It should only be employed where the distension and pain are very great, or where abdominal section is refused or unadvisable; and the practitioner and friends should be made well aware of the risks. The chief amongst these are: (a) Not hitting the right coil; thus one (though distended) may be hit so far from the obstruction as to give no real relief. In not a few the bowel below the obstruction may be punctured (Treves). (b) The puncture may give rise to no real relief, but to increased distress. (c) The puncture may be followed by fatal leakage into the peritoneal cavity.

**VARIETIES. — I. Strangulation by Bands or in Apertures.**—As some at least of these depend upon the results of previous inflammation, it is important to inquire minutely into the early history.

**ANATOMICAL CHARACTERS.**—The bands causing strangulation may be adhesions resulting from peritonitis, omental, a persistent Meckel's diverticulum, or some normal structure abnormally attached. In any case it is the small intestine, and usually the end of the ileum, which is especially liable to become thus strangulated. A band or bands, sometimes rounded, sometimes flattened, may stretch from one part of the mesentery or omentum to another, or, oftener, may be attached by one end to the omentum or mesentery, and by the other to the bowel (only exceptionally to the large intestine), or to the abdominal wall or pelvis. Much more rarely are they found passing from one part of the bowel to another. The broader adhesions sometimes present slits in which portions of bowel may be caught. Similarly, ruptures or slits may occur in the omentum or mesentery, or even in very rare instances in the suspensory ligament of the liver, or broad ligament of the uterus. The lower attachments of the omental bands are usually to the parietes or viscera, to the brim of the pelvis, or to near the inner orifice of one of the rings. Of the diverticular bands, the most important is the one called 'true,' which is due to a persistent or partially obliterated vitelline duct. This band is single, and is attached at one end to the ileum from one to three feet from its termination; its

other end may be free or attached in the vicinity of the umbilicus. Its structure may be like the intestine, or cord-like. The intestine may be strangled beneath it, when the band is adherent at both extremities, or snared and knotted up by it, in a much more complicated way, when its distal end is free. Other congenital bands may arise from the omphalo-mesenteric vessels. Strangulation may, much more rarely, take place in various forms of internal hernia (hernia into the foramen of Winslow, or into peritoneal pouches, mesocolic, duodeno-jejunal, or retro-peritoneal).

**ÆTIOLOGY.**—Strangulation of the small intestine by the above-mentioned bands or apertures is most commonly met with in early adult life, the average age being from twenty to forty. One sex does not appear more liable than the other, except in the case of strangulation by diverticula, which has been observed about twice as often in the male as in the female subject. The more youthful the patient, the more will a true diverticulum suggest itself. But strangulation is not unknown in much older patients, as shown by the subjects of herniæ and omental bands.

**SYMPTOMS.**—These are similar to the symptoms already described under Acute Obstruction.

**TREATMENT.**—If the ordinary measures fail, the surgeon may resort to abdominal section with much hope—especially if the age or anything in the history pointing to previous peritonitis make it probable that a single band is present. As to the date of interference, the earlier the better, before the patient's strength is lowered by vomiting, and the operation rendered additionally difficult by distension.

**II. Volvulus.**—By this is meant a folded or twisted condition of the intestine, which closes its canal. In one form, the only common one, the sigmoid is twisted upon its mesenteric axis. In other forms the intestine, again usually the sigmoid, is intermixed with some adjacent coil of small intestine. Very rarely the twist takes place not on the axis of the mesentery, but on that of the intestine itself. A few cases are on record in which the small intestine—*e.g.* the lower part of the ileum—has been the seat of the volvulus.

**SYMPTOMS.**—In addition to the usual symptoms of Acute Obstruction, there are certain special points which may aid in the diagnosis. The patients are usually adults; and constipation has most likely been of long standing and habitual. The symptoms are urgent (with the exception, perhaps, of vomiting, which may be less marked than in other forms of acute obstruction), and may end rapidly in death. But the point which is likely to be distinctive of volvulus is the urgent distension of the abdomen, often due to one or more huge coils of large intestine.

The diagnosis may perhaps be facilitated by the use of enemata. Mr. Treves has suggested that the passage of the hand under an anæsthetic into the rectum might clear up the case; but the attachment of the meso-rectum is often so short as to prevent the hand being safely passed sufficiently high; and with regard to the same surgeon's suggestion that auscultation be practised along the colon while enemata are given, however carefully this be attempted, and however well borne by the patient, the sound of the entrance of fluid is so diffused that it is not always easy to make sure of its presence or absence in the colon.

**TREATMENT.**—While the usual palliative treatment should be enforced at once, it must be remembered that all treatment is, in acute cases of this kind, most unsatisfactory, owing to the rapid onset, the huge size of the coils, and the early stage at which inflammatory changes set in. All attempts to reduce the volvulus by abdominal section, except in the very earliest stages, are likely to be futile, and perhaps disastrous. The distended sigmoid should be exposed and its contents evacuated, if gaseous by a trocar, if fæculent by washing them away as they escape by a stream of boiled water. If the volvulus can now be thoroughly reduced the opening may be closed; if not, it should be converted into an artificial anus. And if the practitioner is not sure that the distension is sufficiently relieved, he had better perform a lumbar colotomy, the right side being chosen if the cæcum needs relief. This has been done as a primary operation with complete success in three cases.

**III. Intussusception or Invagination.**—This form of obstruction is most important, owing to its frequency in early life, its rapid fatality in many cases, and the number of brilliant successes which have followed insufflation and abdominal section.

**ÆTIOLOGY.**—Intussusception is the most common variety of obstruction to which children are liable, about one-fourth of the cases on record having occurred during the first year, and more than one-half during the first seven years of life. In early life and childhood it is more frequent in males. Intussusception would appear to be brought about by some irregular peristaltic action of one part of the intestine, perhaps conjoined with inaction of another part. Sometimes it follows severe purging, diarrhœa, or violent straining, sometimes the presence of worms, or a polypoid growth, especially in the lower ileum. In a few cases it has followed movements or strain, especially during the dandling and dancing of an infant upwards and downwards in the arms.

**ANATOMICAL CHARACTERS.**—In ordinary cases a transverse section will show three rings of bowel, and a longitudinal one three layers of bowel on each side. The external

of these three layers is called the *intussusciptens* or sheath, the middle the returning layer, and the innermost the entering layer, these last two, taken together, forming the *intussusceptum*. Of these layers, the outer and middle have mucous surfaces in mutual contact, and the middle and inner have serous ones thus opposed. The increase of the intussusception takes place by the entering and middle layers moving in together, and dragging in the outer one after them. Thus, while the inner bend between the entering and the middle layers remains the foremost part of the intussusception, the outer bend between the middle and outer layers is constantly changing. Between the middle and inner layers the mesentery or mesocolon or both, as the case may be, are drawn in. This fact determines, from the first, peculiarities in the shape of the intussusception; and, later on, the compression of their vessels brings about congestion, ecchymosis, swelling, and sloughing. Thus the traction on the mesentery, often considerable as the invagination increases, causes a curving of the cylinder of the intussusceptum, and to a less degree that of the sheath, towards the root of the mesentery. It also may pull the axis of the intussusceptum and its terminal orifice out of that of its sheath, and nearer the mesenteric border of the intestine, the orifice of the end of the intussusception becoming also, by the traction, slit-like. Intussusceptions occur much more frequently in some parts than in others. Thus, when the small intestine, especially the ileum and lower jejunum, is involved, the *enteric* variety is spoken of (30 per cent.) In the ileo-cæcal region two forms are found: (a) the *ileo-cæcal proper*, in which the ileum and cæcum, preceded by the valve which forms the most advanced point of the intussusceptum, pass into the colon. This variety forms 44 per cent.; it may attain great size, the valve reaching as far as the anus, and being still liable to be mistaken by careless observers for a prolapsus recti. (b) In the other, *ileo-colic* (8 per cent.), the lower part of the ileum is prolapsed through the ileo-cæcal valve. The *colic* variety (18 per cent.) may occur anywhere in the large intestine, but is most frequent in the descending colon and sigmoid flexure.

**SYMPTOMS.**—In *acute* intussusception the onset is sudden; pain is an early symptom, and of a colicky, gripping character, but not as a rule so agonising as that which may be produced by a band. Like tenderness, it may here be a guide to the site of the intussusception. Vomiting is not so urgent as in strangulation by bands or other conditions. And the same is true of constipation: this is rarely absolute; usually it is replaced by a diarrhoea, at first fecal, then fecal mingled with blood-stained mucus, the admixture of fecal matter becoming less or ultimately absent altogether. Severe tenesmus and strain-

ing recur at frequent intervals; and these, with the absence of sleep, and the inability to take food, soon exhaust the strength of little patients. On examination of the abdomen, a firm, cylindrical, sausage-like swelling can be distinctly felt as the intussusception-mass in all cases of ileo-cæcal intussusception. In these cases it may traverse the right flank, the upper part of the abdomen, the left flank, and even be felt *per anum*, or seen projecting. With the above symptoms are present, to a varying degree, those of shock and peritonitis.

Under the name of *chronic intussusception* are included those cases of intussusception which have a duration of one or more months. Their extreme importance is shown in the words of Mr. Treves—that no form of intestinal obstruction offers so many difficulties in the way of its recognition, and no form has been the subject of more error in diagnosis. This form appears most frequently in adult males. Its course is most irregular. The onset is sudden only in about one-third of the cases—a point, when present, of much value in the diagnosis of this from other forms of chronic obstruction. Pain, seldom severe, may be entirely absent for long intervals, these becoming shorter as the disease advances. Vomiting is only marked in about half the cases; it is feculent only in about 7 per cent. The action of the bowels varies greatly. As a rule they are irregular, more often with a tendency to diarrhoea than towards constipation. In about 50 per cent. a bloody discharge from the anus may be expected. Distension of the abdomen is slight, and may be absent save during attacks of temporary obstruction. A tumour is to be felt in about half the cases. The general condition is one of wasting and anæmia, the patient not infrequently dying of marasmus.

**COURSE AND PROGRESS.**—(1) *Acute*. Unless relief is obtained, the case will end fatally, within three to five days in children, from shock and exhaustion; in infants it may be in a much shorter time, or within a week or ten days in older children; in adults between the second and third week. These cases lead up to the second class of *subacute* cases, in which life may be prolonged for three or four weeks. (2) The case may be more or less *chronic*, and terminate in death only after a period of several weeks or months, from wasting and exhaustion or peritonitis and enteritis, with or without perforation. Lastly, recovery may take place after sloughing, separation, and evacuation of the invaginated bowel. This last event, of great interest, but by no means necessarily curative, is extremely rare in children under two. It has occurred proportionately in 20 per cent. of ileo-cæcal, in 28 per cent. of colic, and in 6 per cent. of enteric cases. Its most frequent period of occurrence is from the eleventh day to the period

between the third and fourth week (Leichtenstein). Briefly stated, it is brought about by adhesions forming between the invaginated and receiving portions of the bowel, and death of the intussusceptum from strangulation of its blood-supply. In most of the recorded cases it has been some portion of the small intestine that has come away; in some the cæcum and its appendix with portions of the colon. Sometimes the sloughed intestine is evacuated in shreds, at other times in its entirety. While this result is usually met with in acute cases, it may occur in chronic ones that end acutely. The signs are great fœtor of the stools, and gangrenous shreds of intestine found in them. It has been already said that this process can by no means be relied on to save life. The giving way of the adhesions, and fæcal extravasation, chronic diarrhœa, hæmorrhage, pyæmia, and, later on, a stricture, may prove fatal.

**TREATMENT.**—Acute intussusception, especially in the child or infant, demands prompt and active measures. Opium and belladonna should be given at once and sufficiently to check undue peristaltic action and relieve pain. The abdomen should be kept covered with hot fomentations; and the amount of nourishment given by the mouth should be most strictly limited. In little children especially, owing to the rapidity with which their strength runs down, and in all cases, so as to ensure a condition which still admits of replacement, reduction should be attempted as early as possible. Inflation should be preferred to injection as somewhat less risky. The child being placed fully under an anæsthetic, and its pelvis and lower limbs somewhat raised, the nozzle of a Lund's inflator, a rectal enema-tube, or a full-sized catheter well smeared with vaseline, attached by tubing to a bellows, is carefully passed into the bowel. The nates being securely pressed around the tube, air is steadily pumped into the colon, while the surgeon keeps one hand on the abdomen not only to prevent over-distension, but also to watch for any receding of the tumour towards the cæcal region. With regard to the amount of force used, replacement of the bowel can usually be effected only by considerable distension of the whole colon, and this requires a good deal of rather forcible pumping to complete it. This is especially the case with regard to the last few amounts of air sent in (Goodhart). By carefully kneading the abdomen so as to facilitate the passage of air upwards, and thus preventing the sudden over-distension of short lengths of the colon, the above risk will be still further reduced (Taylor). Inflation failing, if the condition of the patient admit of it, more powerful means may be made use of by connecting the rectal tube with an improvised water-cistern placed high above the bed, a more equable

and forcible distension being thus obtained (Goodhart). This authority points out that the last method entails a greater risk of rupture of the bowel, but that the end justifies the means, considering the great danger of these cases. If the above methods fail, abdominal section should at once be resorted to in suitable cases. The following points are here important: (a) *The age.* In infants under a year, unless reduction is early tried and quickly successful, the prognosis is very desperate, whether an operation be performed or no. (b) *The condition of the patient as to collapse, &c.* (c) *The duration of the case.* In the majority, especially in children, the tendency of the condition is to strangulation and not simply incarceration, and while the rapidity of the strangulation varies a good deal, the chances of inflation or injection are small, unless in recent cases. (d) It is thus of the utmost importance to decide *whether the bowel is strangulated or incarcerated.* Mr. Hutchinson points out that the severity of the symptoms will be helpful here, namely, the urgency of the vomiting, the degree of the constipation, the character of any stools passed, and, above all, as utterly incompatible with gangrene, advance of the tumour onwards.

Only the chief points in *the operation* can be given here. The peritoneal cavity being opened by a median incision with the umbilicus for its centre, the intussusception is found. For a few minutes careful pressure should be made on this to diminish the œdema and inflammatory swelling, before any attempts at reduction are made (Senn). The wound being sufficiently enlarged so as to admit of two fingers of each hand, and the small intestine packed away, if the mass cannot be hooked up into the wound it must be reduced *in situ*. This can be done, if there are no adhesions, either by traction upwards on the upper part of the invagination; or, as thought better by Mr. Hutchinson, by finding the lower part, holding the ensheathing part so that it cannot be drawn into constricting folds, and backing out the contained bowel by gentle squeezing movements between the finger and thumb. These movements must be continued, and the finger and thumb gradually shifted upwards along the gut till every atom of the mass is reduced; this being often made known by the appearance of the vermiform appendix. Every care must be adopted to finish the operation as speedily as possible, and every precaution taken against shock. The chief points in the after-treatment are the application of warmth, and the administration of milk, brandy, and laudanum.

**IV. Impaction of Gall-stones.**—These may enter the intestine either after passing down the gradually dilated duct, or after a process of inflammatory adhesion and ulceration between the gall-bladder and duodenum

or colon. It is in this latter way that gall-stones large enough to block its canal get into the bowel; and hence in such cases, though there is usually a previous history of more or less suffering in the hypochondriac region (which may assist in the diagnosis), those paroxysms of pain and the jaundice which accompany the passage of gall-stones down the duct may not have been experienced. Gall-stones may either escape by the anus, or become firmly impacted and completely obstruct the intestine. Obstruction from this cause is, however, comparatively rare. It is met with far more frequently in the female than in the male, and with very few exceptions after late middle life. The patients are often very obese. As a rule the obstructing gall-stone is single. The most common seat of impaction is the lower ileum, and next to this the jejunum or duodenum.

**SYMPTOMS.**—These are usually sudden and acute; death from enteritis conjoined with those of acute obstruction occurring, as a rule, in about five days, perhaps earlier, as the result of shock. Recovery rarely takes place. In some few exceptional cases relief has come about even after periods of severest suffering, by the release and onward passage of the stone; in some the bowel has become stretched into a kind of diverticulum, in which the stone has remained lodged; in some, after adhesion and ulceration, an opening has been formed between the small intestine and the neighbouring part of the colon, or by similar processes the stone has escaped externally.

**DIAGNOSIS.**—This is aided by the sex, age, and previous history of the patient; the acuteness of the symptoms; and perhaps by the recognition of a more or less distinct hard lump corresponding to the obstructing gall-stone.

**TREATMENT.**—If a very short trial of palliative treatment is futile, abdominal section is here the patient's only chance; though, owing to the usual age, habits, and condition of the tissues, the step must always be a very anxious one. The stone having been found, it may be dealt with—(1) By trying to pass it on into the large bowel. (2) By intra-intestinal crushing, either between the fingers or by flat-bladed forceps guarded with indiarubber tube. (3) This may be facilitated by Mr. Tait's suggestion of puncturing the stone with a needle, passed obliquely. (5) The loop being drawn outside, the stone may be extracted and the opening most carefully closed.

**V. Contractions.**—In this class may be included cases due to adhesions, kinking or compressing the gut, or matting together several coils; and those where mischief, inflammatory or malignant, has closed the lumen by traction, or narrowed it—as when the mesentery puckers from old inflamma-

tory or malignant mischief. These cases are amongst the gravest and the most hopeless of intestinal obstructions; they affect the small more frequently than the large intestine; and are often preceded by old peritonitis.

**SYMPTOMS.**—Owing to the rarity of stricture of the small intestine, this group practically comprises all cases of chronic obstruction of that part of the bowel (Fagge). The attacks are often repeated, and the final one is in duration subacute or chronic rather than acute. Thus, sickness or constipation will not be severe, however high up the obstruction may be; severe collapse or suppression of urine will probably not appear. On the other hand, visible peristalsis, and attacks of pain with loud rumblings, will probably be marked features.

**TREATMENT.**—This must be mainly palliative, on the lines already laid down. Abdominal section is here especially likely to be disappointing, owing to the complicated nature of the obstructing agency, the fact that it is often not very localised, and the intimacy with which it is blended with one or more coils of intestine, or with these and some other viscus as well. Formation of an artificial anus, and still more colotomy where the large intestine is affected, may give relief.

## VI. Obstruction from Stricture.—

(a) *Simple cicatricial stenoses.* These may result from the effects of dysenteric, tubercular, or syphilitic ulceration of considerable extent. The dysenteric are most frequent in the lower part of the large intestine, the tubercular in the lower ileum and about the ileo-cæcal valve, and the syphilitic in the rectum. Rarer instances of stenoses occur after injury, the strangling of an intussusception, the strangling of hernia, or the ulceration produced by temporarily impacted gall-stones, fæcal masses, or foreign bodies.

(b) *New-growths.*—A more exact pathology has practically narrowed down these to one kind, namely, cylindrical epithelioma. Very rarely innocent growths—*e.g.* fibromata or adenomata—may cause more or less obstruction, usually by becoming polypoid. The locality attacked is in the very great majority of cases on the left side. Most cases occur in about equal proportions in the rectum and sigmoid flexure; then about half as frequently in the left colon and splenic flexure; the remainder being met with in the ascending colon and hepatic flexure.

**SYMPTOMS.**—In the earlier stages these are often by no means marked. Disordered, irregular, and unsatisfactory action of the bowels, and general discomfort, with intervals of comparative ease, constitute the earliest indications. At this time, owing to the forgetting or deferring of an examination, the cases are far too frequently treated as instances of flatulence, indigestion, and constipation. Later on, the symptoms of obstruction

become more pronounced. In most cases abdominal distension, accompanied by fetid eructations and pain, comes on, varying in degree from time to time. The pain is usually distinguished by its paroxysmal character, and its relation to unwise feeding. Vomiting usually appears late, until then being replaced by nausea, and usually only becomes feculent in the final attack of obstruction. The motions are always unsatisfactory, the bowels not acting without aperients, loose stools often alternating with scybala, or broken up bits with slime and mucus coming away. Troublesome, teasing tenesmus, causing the patient to repeatedly seek relief from the sensations of an imperfectly emptied bowel, is often present when the stricture is low down. Peristaltic movements of the intestines, accompanied by colicky pain and rumbling borborygmi, become increasingly frequent and manifest as the muscular fibre hypertrophies. As the case progresses, the general condition of the patient, with the increasing emaciation and cachexia, and the failure of treatment, all point to the malignant aspect of the case.

**COURSE AND TERMINATIONS.**—The course of all such cases, though very variable in duration, is progressively unfavourable, and sooner or later death supervenes, usually from peritonitis, with or without perforation, or from exhaustion by prolonged suffering and possibly suppuration. Absolute occlusion, as a rule, comes about slowly. In some cases it is never completely established; in others, it occurs suddenly, from impaction of hardened feces or undigested food. It is worthy of note that, even in cases in which the seat of constriction is in the sigmoid flexure or rectum, the greatest evidence of stress of the fecal accumulation, distension, ulceration, and perforation, is often in the cæcum.

**TREATMENT.**—From the very first, as soon as there is reason to suspect the presence of a stricture, the most careful attention to diet, and the regular administration of such medicines as will favour soft, semi-solid motions, should be insisted on. If these points are attended to, the patients may be kept, for the year or two which the disease takes to run its course, in comparative ease; but it is extraordinary how frequently and at what cost these most obvious precautions are still neglected. Patients, the subjects of stricture, should be warned that they are living on the brink of a precipice. As a rule, carelessness in diet brings its own penalty, and most patients willingly acknowledge the relief which such a diet as the following gives. Soups; milk, bread and milk, milk and oatmeal, arrowroot; poached or whipped-up eggs, a few oysters, a little vegetable (especially those which leave a small residue) with plenty of gravy, or well puréed with milk; a little underdone meat, well pounded or shredded. In the early stages, and later on

in the intervals of relief from threatening obstruction, laxatives should be given regularly. Thus cascara sagrada tablets, compound liquorice powder, a pill containing aloes, compound extract of colocynth and oil of juniper—one of these may be administered overnight, while Hunyadi János, or a similar water, is given in the morning. In other cases castor oil is preferable, or strychnine, belladonna, and aloes may be given in the form of pill twice or three times a day. Where obstruction is actually threatening, a laxative such as castor oil or sulphate of magnesium may still give great relief if combined with a little tincture of opium, and aided by enemata of castor oil. But where obstruction is actually present, the case must be treated on the usual lines, a sedative here replacing laxative treatment.

When the above treatment has failed, lumbar colotomy (on the right or left side, according to the indications afforded) should not be too long deferred. The merits of this operation in relieving suffering and prolonging life in such cases can scarcely be esteemed too highly.

**VII. Obstruction from Compression from without.**—Various viscera, enlarged and displaced, especially the uterus and ovaries, and in rarer instances the spleen, the kidney, or even a distended bladder; tuberculous or cancerous glands, tumours of the omentum, growths from one part or other of the abdominal or pelvic parietes, hydatid cysts, &c., may so compress a neighbouring portion of bowel as to lead to obstruction.

**DIAGNOSIS.**—Careful examination, including that *per vaginam* and *per anum* (sometimes the introduction of the hand into the rectum may be useful), together with the history of the case, consideration of the collateral signs and symptoms, and in some cases, as where the swelling is cystic, an exploratory tapping or aspiration, will generally suffice to establish, approximately at any rate, the existence of this form of obstruction.

**TREATMENT.**—This consists, first, in the removal of the cause, if practicable; secondly, where this is impossible, in relieving the obstruction. By altering the position of the body, the displaced viscera may sometimes be moved so as no longer to compress the bowel; tumours—ovarian, uterine, renal, or hydatid—may be dealt with by operation. Where this is impossible, manipulation or copious enemata may be employed, or sedatives may be tried, in the hope that the bowel may, under their influence, release itself. But if none of these measures should be applicable or successful, and if the symptoms of obstruction be severe, resort to colotomy, or the making of an artificial anus by abdominal section, may become needful. In some cases of this class more especially, relief has been afforded by puncture with a fine trocar and cannula.

### VIII. Obstruction from Impaction of Foreign Bodies, Intestinal Concretions (Enteroliths), &c.

(a) Foreign bodies in bulk, such as bones, coins, &c., occasionally find their way into the intestines. In a considerable proportion of cases they are evacuated *per anum* without much inconvenience; in some cases they give rise to enteritis and various other intestinal troubles; in some rare cases they lead to more or less complete occlusion, with acute or subacute symptoms.

(b) Foreign bodies, as hair, &c.; skins, seeds, and stones of fruit (the husks of cereals, and oats especially), the curds of milk in young children, and some medicinal substances, as magnesia, chalk, iron oxide, &c., taken over a long interval, may accumulate and give rise to more or less complete obstruction. Such masses constitute the large proportion of the so-called 'intestinal concretions' in the human subject.

(c) Hard concretions (enteroliths), consisting for the most part of phosphates of lime and magnesium, with organic material, and having usually as a nucleus some foreign body or hardened fæces, have rarely been met with.

Intestinal concretions are most frequently found in the cæcum or rectum; much more rarely in the ileum. They are slowly formed, and rarely bring about intestinal obstruction.

TREATMENT.—As a rule, purgatives can only do mischief. Soothing remedies give temporary relief, and favour the gradual onward passage of the foreign body. If absolute impaction has clearly taken place, and the symptoms are urgent, abdominal section and extraction may be imperative.

**IX. Obstruction from Impaction of Fæces.**—Prolonged constipation may lead to definite obstruction by impaction of faecal masses, conjoined with paralysis and inaction of the bowel from distension, and contraction of the empty portion below. Sometimes the occlusion is rendered more absolute and irremediable by the doubling or dragging down of the bowel by the weight of its contents. The seat of the obstructing faecal mass is usually the sigmoid flexure or the rectum; but great accumulation and its effects—distension, ulceration, perforation—are often most manifested in the cæcum. This cause of obstruction is most frequently met with in women of sedentary habits, after middle life, and especially amongst hypochondriacs or lunatics.

SYMPTOMS.—These are characterised by their chronicity; complete occlusion, as a rule, coming about slowly. There is little or no actual pain during the earlier stages; and even during the later stages, in the absence of complications, it rarely becomes acute. Vomiting is altogether absent at first; in the later stages there may be much nausea, accompanied with foul evacuations, and, towards the last, fæculent vomiting. Absolute con-

stipation is slowly established, and then may last for two or three months. In a few cases the extraordinary periods of seven, eight, or even nine months have been reached without relief (Treves). Before the constipation has thus become absolute, it has very likely been interrupted by attacks of diarrhoea, due to catarrh of the intestine above the impaction, and giving very imperfect relief. The faecal masses can sometimes be felt on examination of the abdomen, or they may be concealed by the presence of fat, flatus, and distension.

COURSE.—In a considerable proportion of these cases relief may be afforded by appropriate treatment. In some, death ensues from gradual exhaustion, from peritonitis after ulceration and perforation, or from acute obstruction owing to the bowel becoming suddenly blocked or acutely kinked.

TREATMENT.—Very copious enemata, administered through a long soft tube, are especially useful, the rectum being first cleared out with a finger or spoon. Such enemata, consisting of thin gruel, soap and water, with turpentine, castor oil, or ox-gall added, should be repeated from time to time, and carefully insinuated onwards as far as practicable. Or a stream of warm water from a vessel raised to a height through a long tube, may be advantageously made to play upon and wash away, portion by portion, the faecal mass (Gay). In some cases galvanism and massage, aided by the sudden application of heat and cold, may be useful.

In the earlier stages, laxatives or purgatives may be given with safety and advantage. A teaspoonful of Rochelle salt in a cupful of mutton-broth is an old but often efficacious remedy. In the later stages, and when purgatives alone are contra-indicated, they may be combined with sedatives, castor oil or sulphate of magnesium being given with small doses of laudanum, or calomel being similarly combined with a little Dover's powder. The use of belladonna in large doses is often peculiarly beneficial.

In the extremest cases, while colotomy is to be looked on as the very last resort, the practitioner should avail himself of this rather than allow his patient to die of over-distension and ulceration of the intestine. When relief has been obtained, the greatest care as to diet and after-management is necessary, in order to prevent that recurrence of trouble to which the patient remains liable. See CONSTIPATION.

**X. Obstruction from Congenital Malformation.**—Constriction or occlusion of this kind is very rarely met with in the duodenum at or about the entrance of the common bile-duct, or about the junction of the duodenum with the jejunum; and in some cases has appeared to depend upon valve-like folds of mucous membrane, resembling enlarged or confluent valvulæ conniventes. The lower portion of the ileum, near

the ileo-cæcal valve, or about the junction with the omphalo-mesenteric duct, appears most likely to be so affected. The colon (and almost exclusively the sigmoid flexure) is the part of the bowel most frequently constricted by the effects of fœtal peritonitis, but instances are very rare.

All such cases are of pathological interest rather than of practical importance. Vomiting of meconium, absence of proper evacuation, straining, convulsions, and evidence of more or less severe suffering, are followed by speedy death, though in some rare instances life has been prolonged for weeks or even months. No treatment can avail, and surgical operations can only hasten death, or at best succeed in prolonging misery.

Very much more common, and somewhat more hopeful, are those cases in which there is congenital defect of the lower part of the rectum or anus, or both. They may be divided into—(a) imperforate anus; (b) anus in natural position, but the rectum deficient.

**TREATMENT.**—Immediate relief may often be afforded by surgical operations, and in some instances more or less permanent good results have been obtained, and by persevering management maintained; but survival to adolescence or adult age has seldom ensued. See RECTUM, Diseases of.

ARTHUR E. DURHAM.  
W. H. A. JACOBSON.

**INTESTINAL WORMS.**—This combined term was formerly much employed in medical literature, as an equivalent for the simpler expression *entozoa*, which latter title is far better, more comprehensive, and now in general use. To be sure, nearly all the internal parasites of man, at some time or other during the course of their development, play the part of *intestinal* worms, within either the human or animal host; but since this particular residence frequently constitutes neither the only locality they occupy, nor the principal feature of their life-record, it is well that the misleading words in question should be altogether abandoned from medical science. See ENTOZOA; and PARASITES.

T. S. COBBOLD.

**INTESTINES, Diseases of.**—SYNON.: Fr. *Maladies de l'Intestine*; Ger. *Krankheiten des Darmes*.

**GENERAL REMARKS.**—Morbid affections of the intestinal tract are of very frequent occurrence at all ages.

The direct exposure which this canal offers to external influences, in the form of ingesta, will account for a large proportion of cases; so many irritants can exert their immediate influence, and produce what may be termed *primary* affections of the canal. On the other hand, since much of the physiological work of the tract depends for its performance on a healthy condition of other functions, especially those of the blood-circulation and

nervous system, any disturbances of these processes will tend to influence injuriously intestinal digestion, and thus give rise to *secondary* diseases of the bowel. And it is evident that an improper preparation of the food in the intestine must in its turn affect the nutrition of the tissues generally, and among others those of the canal itself.

Due to these varied causes are the most diverse forms of structural disease, implicating especially the mucosa and submucosa, and less frequently the muscular and peritoneal coats. Perversions of vascularity, such as hyperæmia and congestion with their results, hæmorrhage and œdema, are often met with; and all degrees of inflammation, from a simple catarrh to suppurative enteritis with abscess, or diphtheritic and other specific forms of disease, such as typhoid, dysenteric, and choleraic. Among the forms of degeneration the lardaceous is the most important; of new-growths, carcinoma is most common, with tubercular next, whilst the connective tissue, fatty, mucous, and vascular neoplasms are rarer; malformations and malpositions complete the list of structural affections. See DIGESTIVE ORGANS, Diseases of.

Although structurally continuous with the stomach, and closely associated with it in its working, the intestine nevertheless is exceedingly prone to be diseased independently of that organ, while at other times both suffer together. Certain regions of the tract favour the development of disease, and it is rare to find the entire length of the canal involved; whilst one portion of the tube, the jejunum, is probably less liable to disease than any other organ of the body.

Thus it is that the symptoms due to a disease of the intestines may be masked by more prominent signs of mischief elsewhere, though secondary to the intestinal affection; whilst in other cases the disease we have to treat is but an expression on the part of the bowels of a morbid state, primarily connected with some other organ.

It must be remembered that many symptoms referred to the intestines are really due to improper digestion in the canal induced by causes remote from the bowels, which may be practically normal. Such conditions are to be distinguished from structural affections of the tissues of the intestines, which are mainly the subject-matter of this article, the functional perversions being treated of in the article, DIGESTION, Disorders of.

The indications of actual intestinal disease are frequently vague and uncertain. The subjective symptoms, such as pain, may be completely wanting in some of the most serious forms of disease, or out of all proportion to the severity of the case. An ulcer may proceed to perforation with but a minimum of discomfort, whilst an attack of simple colic may be agonising. Nor is physical examination so fruitful in its results in the

case of intestinal disease as it is in the affections of many other organs, though perhaps in no other region is the *tactus eruditus* so valuable. Many of the states to be considered here undoubtedly pass through their whole course without giving the slightest indication that can be recognised by physical examination. An investigation of the evacuations at present furnishes information within the narrowest limits. From all these circumstances, a diagnosis of many diseases of the intestines must be almost a matter of inference and conjecture.

In respect to treatment, very much may be done with the means at our command. The removal of causes is in a large proportion of cases easy, and a complete cure may be effected. And whilst some of the remaining cases admit of little or no relief, a greater number can be partially alleviated by suitable remedies.

The several diseased conditions of the intestines will be discussed in the following alphabetical order:—(1) Abscess; (2) Albuminoid disease; (3) Atrophy; (4) Concretions in; (5) Contraction; (6) Dilatation; (7) Gangrene; (8) Hæmorrhage; (9) Hyperæmia and Congestion; (10) Hypertrophy; (11) Inflammation, Acute and Chronic; (12) Malformations; (13) Malignant disease; (14) Malpositions; (15) Micro-organisms; (16) Morbid growths, Non-Malignant; (17) Paralysis; (18) Parasites; (19) Perforation and Rupture; (20) Spasm; (21) Syphilitic disease; (22) Tubercular disease; and (23) Ulceration.

**1. Intestines, Abscess in Walls of.**—In the course of severe cases of enteritis—phlegmonous—where the inflammatory process affects all the coats of the bowel, and the products infiltrate the different tissues, collections of pus may be found, but with no well-defined limit, such as constitute an abscess. Such collections may open into the intestine, leaving small ulcers; or through the peritoneal coat, and so cause perforation.

In the chronic enteritis met with in scrofulous subjects, the solitary and agminated glands may undergo slow suppuration, and form abscesses which end by opening into the bowel.

Such morbid products are rather of *post-mortem* interest, giving rise to no special symptoms during life which will permit of their being diagnosed, apart from the existing enteritis; and they are incapable of special treatment.

**2. Intestines, Albuminoid Disease of.** The intestines appear to be affected with lardaceous disease next in frequency to the spleen, liver, kidneys, and lymphatic glands, and it is rare for the alimentary canal to show signs of this degeneration until it has become far advanced in the above-named organs. It is stated that the intestines are affected in 42 per cent. of all cases (Haber-shon).

**ANATOMICAL CHARACTERS.**—As in other organs, the inner coat of the arteries, particularly of those surrounding the solitary and agminated glands, appears to be the starting point of the albuminoid change, from which it gradually extends to adjacent tissues, until the whole thickness of the bowel may be replaced by this material. In milder cases it is limited to the mucous and submucous coats, which in all cases are the first to suffer, the villi being specially affected. Considering the exceeding proneness of the Malpighian corpuscles of the spleen to undergo this change, it is noticeable that the solitary and agminated glands of the intestine, which are of similar structure, should long resist the degeneration, and in many cases may be quite unaffected. Sooner or later, however, the albuminoid granules appear in these structures, until the whole gland is involved. The mesenteric glands are usually implicated; and in severe cases the mesenteric and peritoneal vessels, and even the appendices epiploicæ (Hayem).

To the naked eye the mucous membrane appears smooth, pale, and thickened, often of a 'peculiar glistening aspect' (Friedreich), resembling wet wash-leather (Moxon). The pallor is very striking. When the degeneration is extreme, the surface ulcerates, especially over the follicles, from fatty degeneration and breaking down of the new material, the diminished blood-supply by the constricted vessels leading to this result.

The small intestine, and particularly the lower part of the ileum, is the usual seat of the disease, which sometimes extends upwards to the duodenum and stomach; the colon is occasionally affected.

**SYMPTOMS.**—The most prominent symptoms which this condition gives rise to, so far as the alimentary canal is concerned, are diarrhœa and hæmorrhage. Since the other important viscera are always simultaneously affected, other symptoms coexist. The diarrhœa is rather characterised by fluidity than undue frequency of the stools, though the latter does occur; the evacuations are often greenish from altered blood. It is rare to find either pain or tenderness; and the diarrhœa when once established rarely ceases.

Hæmorrhage from the surface of the mucous membrane, independently of any ulceration, may occur from rupture of the diseased vessels.

**TREATMENT.**—Enemata of starch and opium are useful, though their effect is but temporary. Recovery, even from an advanced state, may follow if the cause, such as a suppurating joint, can be completely removed.

**3. Intestines, Atrophy of.**—A general atrophy of the intestines accompanies a wasting of the entire body from any serious cause of malnutrition, such as starvation, where the organs are estimated in fatal cases to lose

42 per cent. of their weight, becoming extremely thin and transparent.

Intestinal catarrh may lead to atrophy of the bowels, even to an extreme degree. Nothnagel, who has specially studied this condition, found evidence of it in 80 per cent. of deaths from all causes, and at all ages, even six weeks after birth. It may follow on a catarrh which is acute—this being frequent in children—or on the sub-acute or chronic forms. Certain parts of the canal are more liable to be affected than others, the cæcum, ascending colon, and lower end of ileum being the regions most commonly attacked. The change is almost restricted to the mucosa, the villi of the small intestine especially suffering, the thickness of the coat being reduced to one-fifth the normal in extreme cases, together with complete disappearance of Lieberkuhn's glands. The entire layer is replaced by connective tissue containing a few round cells, and presents a smooth appearance, with irregular thickenings at places; there is none of the pigmentation so constantly seen in the fibroid change of chronic catarrh; the muscular tissue, even of the mucosa, and the lymphoid follicles are unaffected. Nothnagel has also described areas of the muscular coat congenitally atrophied.

The symptoms referable to this condition, when the small intestine is involved, are those of general malnutrition from deficient absorption of the digested foods, and this may be so severe as to prove fatal, especially in children and infants. When the change is limited to the colon, there may be nothing beyond a slight diarrhoea, with bulky pul-taceous offensive stools free from mucus. No treatment is specially available for this condition.

4. Intestines, Casts of.—See CASTS.

5. Intestines, Catarrh of.—See 13. Inflammation.

6. Intestines, Concretions in.—SYNON.: *Enteroliths*.—These are commonly met with in the colon, cæcum, and appendix, which offer greater facilities of lodgment, and are liable to cause enteritis, ulceration, and perforation, or varying degrees of obstruction. See CALCULI; and INTESTINAL OBSTRUCTION, p. 1010.

7. Intestines, Contraction of.—The calibre of the intestinal canal may be diminished by the pressure of tumours; by structural changes in the walls, such as cicatrices; or by displacements of portions of the bowel in invagination, &c. Such causes of stricture are more properly described under intestinal obstructions. See INTESTINAL OBSTRUCTION.

The term 'contraction' may be applied to that state of shrinking which the gut is liable to present below the seat of any permanent stricture.

Congenital malformations, producing con-

traction of the canal, even to complete occlusion, may be met with.

8. Intestines, Dilatation of.—The normal diameter of the small intestine may be taken as  $1\frac{1}{2}$  inch throughout; and that of the large intestine as gradually diminishing from  $2\frac{1}{2}$  inches at the cæcum, to  $1\frac{1}{2}$  inch at the upper part of the rectum. But the canal is evidently capable of distension much beyond these limits, as may be recognised when large accumulations of flatus or fæces occur. Such conditions, however, may disappear after death, the bowel returning to its proper capacity. These dilatations, therefore, may be regarded as temporary.

Other forms of distension of a more permanent nature are frequently observed.

Cases have been recorded where extreme distension of the bowel, with considerable hypertrophy of the muscular coat, occurred without any obvious cause, but associated with marked constipation. These have been provisionally termed *idiopathic*, and compared to similar affections of the œsophagus and stomach. The condition has been met with in children even at birth, as well as in adults, and usually runs an acute and fatal course. In one case the colon was uniformly distended to a diameter of 6 to 8 inches; and in another the average diameter of the small intestine was twice the normal, the stomach also sharing in the distension; the person had been a large eater, and was extremely fat. The sigmoid flexure appears to be very prone to this distension.

In the greater number of cases the dilatation is attributable to the existence of some stricture in the course of the canal, and inasmuch as a persistent obstruction is usually located somewhere in the large intestine, it is the colon which most frequently suffers, and this may be so excessive as practically to obliterate the ileo-cæcal valve. The mere accumulation and retention of the contents above the obstruction is doubtless one factor in causing the distension; but a diminished resisting power on the part of the gut probably co-exists, brought about by malnutrition of its textures. The muscular coat of the dilated portions is usually hypertrophied, while the mucous membrane is thinned and peculiarly liable to ulceration, the decomposing contents furnishing an exciting cause for this result.

*Localised* bulgings or sacculations of the bowel may be met with, sometimes involving all the coats, and occasionally as diverticula formed by protrusions of the mucous membrane between the muscular fibres covered only by peritoneum. These 'false or distension diverticula' are commonly found in old people suffering from chronic constipation or some condition causing general distension of the intestines; they are single or multiple, occasionally occurring in great numbers and globular in shape; are most frequent in the

colon, where they extend into the appendices epiploicae, and occur less often in the small intestine along the mesenteric border. Inflammation from the irritation of faecal or foreign matter is liable to be set up in them, leading to perforation into the peritoneum, or very rarely to communication with the bladder.

The existence of any extreme dilatation may be recognised by inspection or manipulation of the abdomen, especially if the parietes be thin and wasted, as they frequently are in such cases. Tympanites is present to a variable degree, uniformly distending the abdomen or causing asymmetrical swelling. Faecal vomiting may occur in connexion with the existence of a dilated intestine, but this is rather to be attributed to the primary obstructing cause, in the symptoms of which the few indications peculiar to this condition are merged. In extreme cases, the movements of the diaphragm may be interfered with, and the heart's action impaired even fatally.

Paralysis or atony from wasting or degeneration of the muscular coats, by diminishing the resistance of the bowel, allows of its distension. This is well exemplified in the extreme dilatation from flatus which so frequently accompanies acute peritonitis; and is comparable to certain cases of gastro-ectasis of a temporary character.

### 9. Intestines, Gangrene of.

**ÆTIOLOGY.**—The immediate cause of the absolute death of a portion of the intestine is the complete arrest of the flow of blood through the part affected. This obstruction may be produced by:—

(i.) Embolus of the superior mesenteric artery. Several cases of this condition have been recorded.

(ii.) Thrombus of the mesenteric veins. The perfect stasis induced by this cause is of very rare occurrence, but it has been seen to follow invasion of the portal vein by malignant disease, and associated with thrombosis of the femoral vein in the puerperal state.

(iii.) Detachment of the mesentery close to the intestine will be followed by gangrene of the part which is thus deprived of its blood-supply.

(iv.) Local constrictions of the bowel. This is by far the commonest class of causes of gangrene, and is the probable sequence of an unredacted strangulated hernia, an invagination or ileus. In these states the vessels are pressed upon, owing to the altered position of the gut, which, with the continuously increasing pressure of the œdema that follows the venous obstruction, leads to complete stasis.

(v.) The more gradual obstruction to the blood-flow, from constriction of the vessels by diseases of their walls, leads to sloughing, and tends to occur in lardaceous disease of the intestines.

(vi.) Sloughing also occurs as a sequence of the long-continued pressure of hard faeces, or of the inflammatory state, when the process is of such intensity that complete cessation of the circulation takes place in localised spots, usually affecting the mucous membrane only, though occasionally penetrating deeper, ulcers remaining after separation of the sloughs.

**ANATOMICAL CHARACTERS.**—From the nature of the constructive tissues of the intestine, the gangrene which is met with is of the moist variety. The portion of bowel which is affected is at first of an intense red colour, gradually increasing to purple, and even to black. The extreme congestion of the vessels leads to effusion of blood into the tissues, which are uniformly coloured; decomposition rapidly takes place in the stagnant blood, and the products acted on by the sulphuretted hydrogen of the intestines become black, all traces of red colour being soon lost. Meanwhile the mucous membrane and muscular coats are swollen and sodden by the serum and blood with which they are infiltrated, and a dark, black to ash-grey, soft, pulpy mass is finally thrown off from the healthy tissue. The extent of substance which may undergo this necrosis and be separated is extremely variable, from a mere slough of half an inch in diameter or smaller, to portions of bowel several feet in length. The late Dr. Peacock records a case where twelve feet were passed in eight portions during a period of three years.

**SYMPTOMS.**—The occurrence of symptoms whereby mortification of the bowels can be diagnosed is scarcely to be expected. The signs for the most part resolve themselves into those of the cause, whether that be a plugging of the mesenteric vessels, or a localised enteritis. Extreme factor of the stools may suggest its presence, but can afford no indication of the extent or depth of bowel involved, which are all important data for prognosis, and to some degree for treatment. It is not until the sphacelus has been passed *per anum*, or that signs of ulceration are manifest, that the positive existence of gangrene can be ascertained. A very few hours suffice to produce this condition when once the cause is established; and since it cannot be either arrested or cured, the separation of the slough is to be desired, although fatal hæmorrhage or perforation may be associated with this process. Intestinal gangrene is always to be regarded as grave, though recovery not infrequently follows sloughing or considerable tracts when confined to the mucous membrane.

**TREATMENT.**—The circumstances associated with this morbid process as a rule preclude any treatment being specially directed towards it. If there be reason to believe that the entire thickness of the intestine is gangrenous, operative proceedings should be at once

resorted to. Considerable success—even 50 per cent. of recoveries according to some—has followed resection of the dead part of the bowel, and union of the segments by sutures (*enterorrhaphy*).

**10. Intestines, Hæmorrhage from.**—An escape of blood from the intestines is a sign of certain morbid conditions rather than an actual disease itself, hence the cause of the hæmorrhage must be sought for.

**ÆTIOLGY.**—The causes of intestinal hæmorrhage may be thus indicated:—

(*a*) *Increased blood-pressure*: Intense hyperæmia or extreme congestion from heart-disease, portal obstruction, embolism or thrombosis of the mesenteric vessels, intussusception, &c.

(*β*) *Affections of the intestinal walls*: Injuries of the bowels; ulceration; vascular growths, hæmorrhoids; lardaceous disease of the walls.

(*γ*) *Primarily altered blood-states or deterioration of vessels*: Purpura hæmorrhagica; scurvy; leucocythæmia; yellow fever and severe intermittent and remittent fevers; hæmophilia.

(*δ*) *Occasional causes*: Disease of the stomach; rupture of aneurysm into intestine; vicarious menstruation.

The mere enumeration of the causes must here suffice. It is obvious that the relative frequency of these conditions differs considerably, and in many cases the cause is at once apparent, whilst occasionally the source of the blood may be more obscure. It would seem from statistics that intestinal hæmorrhage is of more frequent occurrence in males, as gastric hæmorrhage is more common in women; the latter fact being explained by the greater liability of females to ulcer of the stomach, as the former appears to be by the preponderance of males suffering from the determining causes of hæmorrhage, such as liver-disease.

**SYMPTOMS.**—Associated with the symptoms special to the loss of blood, and which are in the main similar to bleeding from any other organ, there are the signs and symptoms of the causal disease. The extent of the hæmorrhage will necessarily largely determine the symptoms, many bleedings being so trivial as to give rise to no appreciable effects, and in extreme cases the loss being so great and sudden as to lead to rapid collapse and death. Between these extremes all degrees of anæmia, faintness, pallor, giddiness, and failing pulse may be observed. A sensation as of a warm fluid flowing into the abdomen is occasionally complained of, but otherwise hæmorrhage in this situation is seldom possessed of characteristic features. Abdominal pain may accompany intestinal hæmorrhage, and is specially severe in embolus of the mesenteric arteries, but this symptom is not to be attributed to the bleeding, both being associated manifestations of a common cause. The

occurrence of the above mentioned indications in the course of a disease liable to lead to this condition, would point to hæmorrhage, especially if there be a fall in temperature from a previous pyrexial state.

Occasionally the escape of blood is beneficial. This is particularly the case where the cause is a congestion of the intestinal tract, with or without hæmorrhoids. Thereby the fulness of the bowels is relieved, and a more equable circulation is established. In some cases of typhoid fever, contrary to what might be supposed, improvement has been noticed to follow a moderate loss of blood (Trousseau).

Except in such cases as when the effusion of blood is so excessive that death takes place before any escapes from the bowel, intestinal hæmorrhage reveals itself sooner or later in the character of the evacuations. If the cause be situated immediately within the anus, or the blood be sufficient in amount to escape alteration, then the red colour is retained. The hæmatin is readily affected by the sulphuretted hydrogen in the canal, and converted into a blackened material, sulphide of iron being formed, which stains the fæces; or a black tarry substance is evacuated, being the altered clotted blood (*see* MELÆNA). As a rule, when the blood has undergone this change, the source of it is in the stomach or small intestine; blood from the colon—where it is usually due to ulceration—being passed adherent to the fæces. The height of the source, the quantity, and the duration of its stay in the canal, largely determine the extent of alteration in the blood, and its degree of admixture with the fæces.

**DIAGNOSIS.**—The history of the case; the condition of the patient; and the character of the voided blood (*see* MELÆNA; and STOOLS), are the points upon which a diagnosis of the cause of intestinal hæmorrhage is to be based.

**PROGNOSIS.**—The amount of blood evacuated is not a sure guide to forming an opinion of the result. It is difficult to estimate the actual quantity lost, since much may be retained in the bowel. The general condition of the patient, especially the state of the pulse, is of far more importance; whilst allowance must be made for the nature of the cause, not forgetting the occasional favourable import of a flux.

**TREATMENT.**—In a certain number of cases bleeding from the bowel is uncontrollable; in others it is capable of cure; whilst in a third group it is rather to be encouraged. When arrest of the hæmorrhage is desired, rest, both general and local, is essential; the patient should be maintained in the recumbent position, as thereby the liability to syncope is averted; and the canal is to be kept quiet by abstinence from food, and the free use of opium, to prevent peristalsis.

The active treatment is to be directed to withdrawing the blood as much as possible from the affected region, by means of heat, sinapisms, dry-cupping, &c.; and to the application of styptics to the bleeding surface, or the administration of such remedies as arrest bleeding after their absorption into the blood. The most effective agents given by the mouth are turpentine in a full dose of 40 to 60 drops, followed by half-drachm doses every three hours; and the *pilula plumbi cum opio* (B.P.) gr. v., every four or six hours. Tannic acid and the vegetable astringents are usually too slow in their action to be of much avail. Bitartrate of potassium in two-drachm doses is of much benefit in arresting the bleeding of piles; for which purpose also, as well as for vicarious hæmorrhage, or the flux of passive congestion from the lower bowel, the writer has found frequently repeated doses (℥ v. to ℥ viii.) of tincture of hamamelis most efficacious. The liquid extract of coca in doses of one and a half drachms at intervals of a few hours, for three or four doses, has been recommended for the same purpose. Probably the most reliable remedy is ergotin in two-grain doses administered subcutaneously, and repeated if needful. Should the source of the hæmorrhage be within the range of local application by rectal injections, the most useful agents for this purpose are turpentine one to three drachms, with four to eight ounces of mucilage of starch; equal parts of tincture of perchloride of iron and water; or tincture of hamamelis, a drachm to three ounces, as an enema. Such astringents as tannic acid, opium, or hamamelin may be conveniently administered in the form of suppositories.

When the hæmorrhage is distinctly the result of engorged vessels, its occurrence should not be checked, provided it be not excessive. Sulphate of magnesium in full doses, with a few minims of diluted sulphuric acid, is then of great service.

The giving of stimulants is a procedure that involves careful judgment. Whilst undoubtedly the tendency of loss of blood is to produce death by syncope, it is also true that faintness itself favours the cessation of the bleeding, and, so far as a general direction can be given, stimulants should be avoided, unless there be reason to fear, from the condition of the patient, character of the pulse, &c., that the syncope is grave. Short of that, alcohol, by temporarily increasing the heart's power, increases the bleeding.

Transfusion of blood, when practicable, should be resorted to in extreme cases. See TRANSFUSION.

**11. Intestines, Hyperæmia and Congestion of.**—The former term is here applied to those conditions of vascular engorgement where the excess of blood is, primarily at least, on the arterial side of the capillaries

(active congestion, fluxion, determination of blood); whilst the latter term is restricted to cases where the fulness is caused by some obstruction to the venous flow (passive congestion). Doubtless either of these conditions may lead to the establishment of the other, but it is desirable to consider them separately, not so much for the difference in the causes producing them, as for the great difference in their results.

It should be remembered that even within the limits of health a considerable variation is met with in the degree of vascularity of the alimentary canal. The fluctuating periods of activity and rest undergone by the tube are associated of necessity with alternations of comparative hyperæmia and anæmia, as during the digestion of a meal or during fasting. It is impossible, therefore, to draw any line beyond which the vascular fulness can be said to be abnormal; as it is equally impossible to say exactly where hyperæmia and normal gland-change end, and catarrh begins. These states, on the border-line between health and disease, stop short of producing recognisable tissue-change, and are of transient duration. See DIGESTIVE ORGANS, Diseases of; I. Affections of the Vascular State.

**ANATOMICAL CHARACTERS.**—The appearances seen *post mortem* are far from being always indicative of what existed during life. For an extreme arterial fulness may completely disappear after death, from contraction of the vessels; whilst venous engorgement more or less completely remains.

**ÆTIOLOGY.**—The causes of intestinal hyperæmia are as follows: (a) Mechanical and chemical irritants, foreign bodies, and poisonous drugs. Spices and highly seasoned food, and alcohol; any substance, in fact, which may be swallowed, and at all exceeds the blandest nature, may bring about an abnormal degree of hyperæmia of the whole or part of the canal. These causes act locally and directly upon the vessels.

(β) Vaso-motor paralysis of the splanchnic area. If from any cause the normal tone of the mesenteric vessels is diminished, by inhibition or removal of the tonic influence excited by the sympathetic, the vessels dilate and hyperæmia ensues. It is in this way that diarrhœa following certain emotional states may be explained. The intimate relation which has been shown experimentally to exist between the splanchnic nerves and the vaso-motor system generally, but especially with the cardiac innervation by means of the 'depressor nerve,' whereby any considerable peripheral resistance in the systemic capillary area which impedes the action of the heart is compensated for by a dilatation of the mesenteric vessels, renders it probable that an undue hyperæmia of the intestines is of very frequent occurrence.

(γ) Collateral hyperæmia, or the fulness of

the vessels of one region caused by contraction of the vessels of another, as in the shrinking of the cutaneous vessels from cold, extensive burns, &c. In such cases the blood, remaining constant in amount, must distend other vessels; and those of the abdominal viscera, including the intestines, are peculiarly liable to become engorged, as explained by what may be called their compensating paralysis.

(δ) Among less frequent causes of intestinal plethora are, the suppression of habitual discharges—menstrual, &c.; the removal of pressure, as of ascitic fluid; and sudden chills in hot climates.

The causes of *passive congestion* are the following: (a) A general congestion of the entire intestinal tract will be produced by any of those causes which lead to universal congestion of the tissues, as dilatation of the right heart from lung-disease. Pressure by tumours or other conditions on the inferior vena cava above the liver, or on the portal vein, will bring about the same result. So also will any obstruction to the portal circulation in the liver. This is by far the commonest cause of intestinal congestion, since cirrhosis of the liver, however produced, directly tends to it.

(b) A congestion of a portion of the tube occurs when any obstruction exists to the venous flow of that part, as is marked in cases of invagination and strangulation of the bowel.

The rarer conditions of embolism of branches of the mesenteric arteries, or thrombosis of the veins, will induce intense congestion of the region supplied by the occluded vessels. The resulting infarction is somewhat smaller than the actual extent fed by the vessels, owing to the free anastomosis at the periphery of the area. Sloughing and ulceration of the mucous membrane, or extensive gangrene of all the coats, is very liable to follow on this condition.

Both active hyperæmia and congestion are essential features of the vascular changes comprised in inflammation.

**SYMPTOMS AND EFFECTS.**—These conditions may of themselves give no evidence of their existence, although an excessive and sudden hyperæmia of the splanchnic area will cause a fall in the general blood-pressure with symptoms of fainting. Overfulness, just as anæmia, of the intestinal vessels leads to increased peristalsis, probably due to the deficiency of oxygen and excess of carbonic acid which attend each of these conditions.

From hyperæmia, there is an increased secretion of mucus and other intestinal fluids, often more watery than normal, which with an increased peristalsis, induced by the same irritant that led to hyperæmia, produces a diarrhœa. Provided this over-functional activity be limited to the production of the normal secretions of the part, and increased

healthy action only, the condition of hyperæmia is not exceeded; but the passage into catarrh is easy, and unmarked by any abrupt lines.

The increased blood-pressure may be sufficient to induce diapedesis of the red corpuscles, or rupture of the capillaries, leading to capillary hæmorrhage and submucous petechiæ.

As regards congestion, the more complete the obstruction to the flow, the greater will be the pressure in the veins, whose thin walls favour the transudation of the serous part of the blood, and so produce first an œdema of the mucous membrane and entire thickness of the bowel, and later an escape of fluid into the canal itself and into the peritoneal cavity, the latter being more marked. The effusion in this case is dependent entirely on mechanical conditions, with possibly some deterioration of the vessel walls; whilst the flow in hyperæmia is mainly the result of increased secreting activity. Hæmorrhage from rupture of the smaller vessels is of frequent occurrence, and may be very considerable. Profuse bleeding, often accompanied by severe abdominal pain, usually follows plugging of the mesenteric vessels.

A prolonged state of congestion—and as a rule the cause is such as to determine a permanent state—leads to certain structural alterations in the tissues of the bowel, from the imperfect nutrition that a chronic venous fulness brings about. The nature of the change is chiefly the infiltration of the mucous and submucous coats with an imperfectly formed connective tissue, often pigmented, which causes a thickening and toughness of the bowels, almost identical with the results of chronic inflammation.

**TREATMENT.**—It is seldom that these conditions are such as call for treatment. The hyperæmia is usually of a transient nature; and the cause of congestion is generally irremovable. Aperients, such as jalap and gamboge, are sometimes beneficial, by inducing watery evacuations and so relieving the vessels, but they demand constant repetition.

The treatment of hæmorrhage has been considered; but this and diarrhœa, when due to congestion, are, unless excessive, often beneficial, and are not to be checked.

**12. Intestines, Hypertrophy of.**—This is always of local occurrence, a general hypertrophy, involving the entire length of the bowel, being practically unknown.

In chronic enteritis the mucous, submucous, and even muscular coats are apt to become much thickened, and though this is partly due to an excessive formation of connective tissue, there is also some actual hyperplasia of the normal textures.

In portions of the intestines above an obstruction, a true hypertrophy of the gut, particularly of the muscular layers, is to be found; and, as already said, this is usually

associated with dilatation of the tube. Marked hypertrophy, especially of the longitudinal muscular bands of the colon, has been noticed in those cases of so-called idiopathic dilatation where no obvious obstruction exists. The pathogeny of this condition is obscure, for it does not appear probable that it precedes the dilatation, as in the heart, but is rather developed coincidentally.

It is rare for this condition to be other than inferred during life; it gives rise to no symptoms and calls for no treatment; and when established is rather of the nature of a compensatory lesion.

**13. Intestines, Inflammation of.**—  
 SYNON.: Enteritis; Fr. *Entérite*; Ger. *Darm-entzündung*.

Under this term are included all those structural changes in the mucous membrane of the intestinal tract which primarily follow the application of an abnormal irritant, provided that the irritant be not of sufficient intensity to produce absolute destruction of tissue. Such changes will involve more or less all the tissue-elements of the mucous membrane, and may extend to the muscular, or even the peritoneal coat. They are essentially characterised by productive, coincident with destructive features, the former leading to the formation of new material, as pus or connective tissue, the latter to ulceration or gangrene. The inflammatory process may present considerable variety in type. The simplest form, to which the term 'catarrh' may be applied, passes, by almost insensible gradations, from the tissue-changes met with in the course of normal digestion, to a distinct condition of disease. Or there may be superadded certain specific characters due either to the nature of the cause, or to the predisposition of the tissue affected, or to both, which determine the conditions known as diphtheritic, phlegmonous, dysenteric, &c. See INFLAMMATION.

There are thus differences in the severity with which enteritis may occur. But in all cases the essential characters of inflammation are present, which may be regarded as the results of the irritant *plus* the efforts at repair on the part of the affected tissue.

The morbid process may affect the intestine throughout its greater length, either in common with or independently of the stomach—*general enteritis*; or it may be distinctly limited to certain parts of the canal—*local enteritis*, including duodenitis, ileitis, typhlitis, colitis, and proctitis. As a rule, the term *enteritis* is restricted to inflammation of the small intestines, and it is in this sense that the disease is treated in this article.

In respect to duration and intensity, enteritis may be *acute* or *chronic*.

(A) **Acute Enteritis.**—Acute enteritis is sometimes called *gastric remittent*, or *infantile remittent fever*—terms it is advisable to discard entirely, since they are

frequently applied to very different diseases. Acute enteritis is meant to include all those cases where the essential features of an inflammation are present, varying in severity from a simple catarrh or muco-enteritis, to those severer forms possessed of special features, such as phlegmonous or diphtheritic. The more severe cases, especially in children, are sometimes called simple or English cholera, or *cholera infantum*.

**ETIOLOGY.**—It is doubtful whether an idiopathic enteritis is ever met with; some cause is generally to be found.

*Predisposing causes.*—(a) The exposed situation of the intestinal tract to irritating substances swallowed, causes this disease to be one of frequent occurrence.

(β) The structure of the intestinal mucous membrane, with its delicate and susceptible epithelial cells, and slightly protected blood-capillaries, favours the occurrence of those changes which constitute inflammation.

(γ) Age especially predisposes to enteritis. For although it may occur at any period of life, infants and children during the period of dentition are peculiarly susceptible. A moderate intestinal catarrh may almost be regarded as a normal accompaniment of dentition, like to the increased activity of the salivary and other glands at that period. From the moderate it may easily pass into the serious or even fatal degree.

(δ) The season of the year appears to exercise an influence, for during the summer and early autumn this disease is certainly much more frequent; and particularly so when there is extreme difference between day and night temperatures, or when the heat is associated with much moisture.

(ε) Occasionally this malady would appear to be epidemic.

(ζ) Certain mental and emotional states undoubtedly confer a liability to the occurrence of symptoms which are practically indistinguishable from those of a catarrhal enteritis.

(η) Conditions of general ill-health and marasmus, especially in children, predispose to intestinal catarrh.

*Exciting causes.*—These, whatever their nature, would appear to act by first inducing a hyperæmia of the tissues, which thence pass into a state of inflammation.

1. Irritating ingesta of the most varied kind, such as abnormal, ill-cooked, or improperly digested food, toxic products of digestion, and irritant drugs or poisons, often cause enteritis, though not so frequently as in the corresponding affection of the stomach. Of these, improper food is by far the most common, especially during the first year of life. Cow's milk alone may be at that time sufficient to produce it, and it is rare for infants to escape an attack. An excessive flow of bile into the intestine is an occasional cause. Certain irritants, such as corrosive

sublimate, used as lotions or injections, have been known to produce enteritis—even fatal—without direct introduction into the alimentary canal.

Specific forms of intestinal inflammation are determined by various micro-organisms and their products, and to such causes may be referred cholera, dysentery, typhoid fever, and probably many cases of 'summer diarrhoea' or cholera infantum, all of which are primarily varieties of enteritis.

2. Exposure to cold may be followed by inflammation of the intestines, as it may be by inflammation of the lungs, kidneys, or pleura; what determines the particular organ involved is unknown. There would seem to be some other factor than the mere determination to the viscera of an excess of cooled blood from the contracted cutaneous capillaries, which probably affects the nutrition of the tissue-elements *viâ* the nervous system.

The occurrence of inflammation and ulceration of the duodenum which occasionally follows extensive superficial burns, cannot be altogether explained by the hyperæmia of the intestines, which is said to follow the superficial injury; but whether the cause be embolic, or the elimination by the bile of some irritant poison, is unknown.

3. Wounds, new-growths, volvulus, intussusception, herniæ, impaction of fæces, gall-stones, parasites, will lead to enteritis.

4. Inflammation of neighbouring parts may involve the intestines by extension, as from the stomach, peritoneum, or bile-ducts.

5. The specific poison of diphtheria not infrequently leads to enteritis of a characteristic nature. An inflammatory state of the intestinal mucous membrane is said to accompany or follow the exanthemata, and more especially scarlet fever; and it sometimes complicates septicæmia, particularly when this is of puerperal origin.

**ANATOMICAL CHARACTERS.**—It probably never occurs that the whole length of intestine is the seat of inflammation, and it is not often that even the entire small intestine is so affected. It is far more frequent to find certain tracts, of a few inches or a few feet, involved. Speaking generally, the colon, cæcum, rectum, duodenum, ileum, and jejunum are attacked, as regards frequency, in the order named. In some situations special features are present, but the essential characters of inflammation always exist, whatever be the site.

Owing to the physical properties of the intestinal tissues, and the rapid onset of softening and putrefactive changes, the appearances seen after death by no means necessarily correspond to what actually exists during life. Thus, the hyperæmic state of the mucous membrane, with the increased redness, varying from a more intense pink than normal up to a deep dark red, may

leave but a trace *post mortem*, the vessels having become considerably emptied from the constriction of the vessels in *rigor mortis*. An increased vascularity, however, is one of the important features of the state under consideration, and it may sometimes be so intense as to lead to capillary rupture and formation of petechiæ in the mucous membrane. The tissue-elements of the gut, as a result of the irritant causing the inflammation, and with the accompaniment of an increased vascular supply, undergo changes in their appearance and behaviour. Thus the epithelial cells are in a state of cloudy swelling and of increased activity in multiplication, each successive progeny approaching nearer and nearer to the embryonic type; the connective-tissue elements are similarly affected; and leucocytes transude into the tissues from the vessels. These new-formed cells constitute pus-corpuscles, which are thrown off from the surface of the mucous membrane, and crowd to a variable depth the tissues of the different coats. If the primary irritant to the inflammation be of a specific character, such as the poison of diphtheria, or of an extremely severe nature, such as an intussusception or hernia, then the new-formed cells become entangled in a fibrinous, coagulable exudation from the blood, and form with the necrosed tissue-elements patches of false-membrane adherent to the surface of the bowel. In all cases there is some œdema of the intestinal walls from serous effusion, and the free surface of the membrane is covered with a glairy mucus, containing pus-cells, and frequently crystals of triple phosphate.

The epithelium of the follicles of Lieberkühn becomes extremely granular, and proliferates extensively, with the frequent result of blocking up the lumen of the gland, which thus becomes very prominent; or the tubular glands may loosen and fall out, leaving well-defined empty pits, particularly if the examination of the mucous membrane be delayed long after death. The solitary and agminated glands are invariably much swollen, and very often the process of inflammation is most intense in their vicinity. Occasionally the mesenteric glands are similarly affected.

How this inflammation may terminate very much depends on the cause and extent: it may subside, and the bowel gradually assume its normal characters with no impairment of function; it may lapse into a chronic state; or it may pass on into ulceration, or even sloughing and gangrene. *See DIGESTIVE ORGANS, Diseases of.*

To these various degrees and varieties of the inflammatory state different terms have been applied.

*Catarrhal or muco-enteritis.*—The essential characters of this are an excessive mucous secretion, with serous exudation or slight

pus formation, in very variable amount, and desquamation of the epithelium. The submucosa may be infiltrated with leucocytes, but it usually remains normal, as also the muscular coat, unless the morbid condition be prolonged. Complete recovery generally takes place, but atrophy with considerable destruction of villi and Lieberkühn's glands, leaving the surface smooth and bare, not infrequently follows, especially in children. There is often some superficial erosion or simple ulceration. This form of inflammation involves great lengths of the canal, and is more common in the colon, or ileum, less so higher up.

*Phlegmonous.*—This term denotes a more severe form of inflammation, in which there is a more considerable formation of pus which permeates all the coats. This is a rare condition, and affects only a short distance of the bowel, which is deep red and distended, and frequently contains a sanious fluid.

*Membranous enteritis.*—When patches of false membrane are met with, of the nature above described, in association with diphtheria, we have a true *diphtheritic enteritis*; and the expression *pellicular* or *croupous* may be more fitly applied to those cases where a similar membrane is formed, though not in connexion with the diphtheria poison. Such a state is of not infrequent occurrence on the prominent edges of the *valvula conniventes*, and still oftener of the *sacculæ* of the colon, due to the irritation of hardened feces or impacted calculi. Both these forms are associated with ulceration, except, it is said, in children, and affect only limited areas of the intestine.

The term *dysenteric* is very indifferently applied to more than one form of enteritis or colitis. The writer thinks it had better be limited to that form of inflammation due to the specific poison of dysentery, although the morbid appearances of such cases are almost or quite identical with some of the varieties enumerated above, the differences being dependent on their causation and clinical history. See DYSENTERY.

It frequently happens that the inflammation is limited to the lymphoid follicles, solitary and agminated, or at least primarily affects them. There is a great tendency for this condition to proceed to ulceration, which rarely extends beyond the mucosa, and never perforates. The ulcers coalesce, forming extensive areas of destruction, with undermining of the remaining mucous membrane. The ileum is seldom thus affected alone, oftener the ileum and colon, and most commonly only the colon.

In fatal cases of diphtheria in children the agminated, and to a less extent the solitary glands throughout the small intestine have been found much swollen and hyperæmic, though not ulcerated, and without diphtheritic membrane, although there was con-

siderable round-celled infiltration of the mucosa and submucosa.

Various micro-organisms have been found in the tissues of the intestine in the different kinds of inflammation, both simple and specific.

**SYMPTOMS.**—That a considerable variation is met with in the kind and severity of the symptoms presented in cases of enteritis, is only to be expected when the great difference in degree and extent of morbid change that is met with is remembered; and in a very large number of cases the symptoms are quite out of proportion to the appearances found *post mortem*. Whilst the pathological changes that take place in a limited part of the canal are identical with those that may be found in another, to a very large extent the symptoms that arise in the two cases may be widely different. For whereas one patient may suffer from an attack of intestinal catarrh, with but a trifling array of symptoms, another may succumb within a few days. There is no one symptom or even group of symptoms that is absolutely characteristic of the disease; even the general condition of the patient is not constant. In the milder forms of intestinal catarrh there may be slight pyrexia, with thirst and quickened pulse, but these symptoms may be scarcely noticeable, and the actual existence of the condition is often assumed, without anything like proof; while, on the other hand, in the severe phlegmonous enteritis they are extreme, and the patient is in a state of considerable prostration.

The following symptoms, more or less marked, occur in different cases.

**Stools.**—Diarrhœa is in some respects the most constant symptom, though, when the affection is limited to the higher part of the canal, and space is given for the re-absorption of the excessive exudation, it may not only be wanting, but there may be actual constipation. The lower down the bowel is inflamed, the greater the liability to diarrhœa, which hence becomes a marked character of colitis and proctitis. In the severe forms of the affection complete constipation may be due to the paralysis of the inflamed bowel, arresting the peristalsis, and allowing of the accumulation of the intestinal contents above the lesion; this is obviously more complete when the enteritis is associated with any state producing mechanical obstruction, such as intussusception or ileus.

The character of the evacuations is very variable. As a rule they are semi-fluid when diarrhœa exists; or they may consist chiefly of an almost clear liquid with a few feculent flakes; but, when time has permitted a partial re-absorption of the fluid portion, the stools become more consistent, and in cases of enteritis which are chiefly due to fecal accumulation, solid, hard masses are passed.

Mucus, in greater or less quantity, is con-

stantly present—being especially abundant in affections of the large intestine and rectum, when it is often discharged as complete tubular casts of the bowel. In catarrhal affections of the small intestine, the mucus may only occur in microscopic particles, and when considerable in amount and intimately mixed with the fæces, generally comes from the first part of the colon, as motions consisting of almost pure mucus come from below the splenic flexure.

Blood is not usual except in proctitis, or unless there be ulceration or hæmorrhoids; and pus is seldom noticed unless the rectum be inflamed. Quantities of intestinal epithelium and micro-organisms may be detected by the microscope.

Owing to the imperfect performance of digestion or absorption, the motions are liable to contain many abnormal constituents—as fat, when the duodenum and upper part of the jejunum are involved, or even masses of food scarcely changed; and the altered characters of the intestinal contents, with the products of decomposition, are in themselves most effective in maintaining a diarrhœa. As a rule, the discharges are paler than normal, or may be even colourless; the greenish tint so often seen in the enteritis of children is due either to a pigment formed by a bacillus, or to biliverdin, and is then considered as indicating some abnormal alkaline change in the intestine, whereby the biliverdin has become altered, though the stool itself as passed is acid in reaction. Unaltered bile pigment in the fæces is abnormal, and its presence indicates catarrh high up in the small intestine, and is always associated with fluid evacuations from increased peristalsis of the ileum and colon. The odour is usually extremely offensive or even putrid, especially the white or greyish putty-like masses passed by children; though sometimes, when the evacuations are very liquid and colourless, smell may be altogether absent. *See STOOLS.*

Owing to a large production of gases, discharges of flatus are of very frequent occurrence, but unless there be actual obstruction, tympanitic distension of the abdomen is not usual.

*Vomiting*, except in the severe forms, is not a common symptom of enteritis, unless the stomach be involved. In phlegmonous enteritis, however, vomiting may be persistent, and even stercoraceous; and it is relatively more frequent in children than in adults. Short of actual vomiting, nausea is frequently complained of.

*Pain and tenderness.*—Pain in itself is a most uncertain symptom, perhaps being scarcely noticeable in the milder forms of catarrh; whilst in colitis, the colicky, griping pains, which may or may not be relieved by pressure, are characteristic. Still more is this the case when the rectum is affected,

when the straining and tenesmus constitute one of the most distressing symptoms of the malady. When the peritoneum is involved, the pain and tenderness are marked and characteristic. Both may be generally diffused over the abdomen, or may be local in character, as over the cæcum; in a large number of cases the pain is referred to the umbilical region.

*General symptoms.*—Among the more general symptoms, or those associated with inflammation of special regions, are the phenomena of the febrile state. The temperature may reach 104° F., or even higher; or in some cases it may be scarcely elevated. The appetite may be unaffected, especially if the upper part of the tract be free from the disease; whilst there may be complete anorexia when the reverse is the case. Thirst is of usual occurrence, and it becomes very marked when the evacuations are abundant and fluid. The tongue indicates rather the general state of the patient, and is a less reliable index of the actual state of the intestinal mucous membrane even than in corresponding affections of the stomach. It may be dry, red, irritable, and glazed; or coated with a thick fur, with the edges and papillæ bright and prominent; in milder cases it is often unaffected. The urine may contain a large amount of indican, indicative of albuminous putrefaction in the intestines. The character of the pulse varies with the general state. Provided that the pyrexia be extreme, there is the usual dry skin and concentrated urine, with a tendency towards the production of the typhoid state, which usually is reached in fatal cases. In many cases the prostration is excessive, though the mind is usually unaffected to the end, and very often there is a marked irritability of temper. Notwithstanding the intimate sympathy between the alimentary tract and the brain, headache is of rare occurrence in enteritis. A persistent hiccough is met with sometimes. In children the disease rapidly leads to a condition of collapse. The child lies in a languid, almost torpid, state; with the skin of the abdomen intensely hot and dry; whilst the extremities are cold and blue, the face is pinched, and the body generally appears shrunken. Frequently this state is interrupted by attacks of convulsions, especially if dentition be in progress. Or the child is extremely fretful, and maintains an almost constant, short, feeble cry, evidently accompanied with pain.

When the disease affects the duodenum, jaundice, due to closure of the bile-duct, very often occurs. The intimate nervous relation between the rectum and base of the bladder explains the frequency of micturition so commonly associated with proctitis.

*DIAGNOSIS.*—The variability and oftentimes vagueness of the symptoms frequently admit of a diagnosis of enteritis being made only by

a process of exclusion. The history of improper feeding, whether temporary or prolonged, often indicates the nature of the disease; though it cannot be denied that all rules of a rational dietary are frequently violated with apparent impunity both by children and adults.

Diarrhœa alone can by no means be taken necessarily to indicate the existence of intestinal inflammation, and the same may be said of constipation, pain, vomiting, and other single symptoms. It is rather to a group of symptoms, with the previous history, that the observer must look. The character of the stools, as already described, often indicates the region of gut affected; and the existence of extreme tenderness and pain, with a hard, quick pulse, and the abdominal decubitus, point to the involvement of the peritoneum, which an exacerbation of temperature tends to confirm. The distinctive features of typhlitis, colitis, and proctitis are elsewhere described sufficiently to form material for diagnosis in most cases. To distinguish between inflammation of the jejunum and ileum is usually impossible, nor, practically, is it a matter of importance. The history of the case, the course of the temperature, and the characteristic rash and headache, should serve to separate typhoid fever from acute enteritis, which is sometimes mistaken for it.

**PROGNOSIS.**—This will clearly depend on the degree of severity of the affection, no less than on its seat and extent. Rest, which is of prime necessity to an inflamed organ, is almost incompatible with maintaining the due nutrition of the patient, and this fact renders the prognosis very uncertain.

A simple intestinal catarrh occurring in a healthy subject certainly tends, after a few days, to complete recovery; but occurring, as it frequently does, in persons in ill-health, it is far more liable to pass into an obstinate chronic condition. In children, the opinion should be very guarded; for whilst in a large number of cases perfect recovery follows removal of cause and suitable treatment, others, for no very apparent reason, will, in spite of everything, progress to a fatal termination; and this is, of course, more likely to be the result where a strumous or tubercular diathesis exists. The mildest diarrhœa in children suffering from atrepsia quickly becomes serious, killing as it were from rapidly supervening shock; the onset of so-called summer diarrhœa during convalescence from such diseases as whooping-cough or pneumonia is also of grave import; and in true cholera infantum, hyperpyrexia, great prostration, and especially uncontrollable vomiting, are almost surely fatal indications. Enteritis in different degrees of severity constitutes one of the most important causes, if not the most important cause, of infantile mortality.

In the severer forms of enteritis, as they affect adults, opinion must be guided by the nature of the cause, and the general state of the patient. Recognising that the unfavourable tendencies of the disease are towards extreme prostration, to perforation with fatal collapse, or to chronic ulceration—according as these conditions are threatened, so may the prognosis be fairly made. The duration of extreme cases rarely extends beyond a few days, when, if death do not occur, the symptoms abate, and recovery, with oftentimes a tedious convalescence, follows, or a chronic condition of disease is established. A very insidious form of enteritis, with rapidly fatal termination from ulceration and perforation, has been noticed in young girls, the symptoms only lasting about twenty-four hours.

**TREATMENT.**—Although great variety exists in the degree of severity of the symptoms of acute enteritis, and a corresponding difference obtains in the treatment to be pursued, yet certain general principles may be first laid down, and the more special details adapted to certain conditions afterwards indicated. Inasmuch as the disease is one of a febrile nature, where, among other things, the tissue-waste is out of proportion to the repair, and at the same time the organs concerned with the preparation of the food are those mainly at fault, every effort should be made to minimise the bodily waste. This is best attained by keeping the patient in bed, which also offers the additional advantage of providing a uniform warmth.

As regards diet, in the greater number of cases of acute intestinal inflammation the appetite is much impaired, even to complete anorexia. Providing the person attacked have been previously in good health, no harm is done by complete abstinence from food for twenty-four or even forty-eight hours. This gives a much better chance of rest to the intestine, and a better opportunity for the removal of any irritant ingesta which may have been the cause of the inflammation. The thirst during this period may be relieved by ice-cold water, with or without a little lemon-juice. It must not be forgotten that, with the mucous membrane and its glands inflamed, the conditions of normal digestion and absorption are materially interfered with, and articles of diet that ordinarily are most nutritious and easily digested, may and do become, under these altered circumstances, positively harmful. The aim in feeding the patient should be to give those materials which require the least digestion, and, being most quickly absorbed, leave the smallest amount of indigestible residue. Provided that the stomach be implicated—and it is rare that it is not so—meat foods are badly borne. Instead of the proteid constituents being digested, they undergo putrefactive decomposition, and thus add fresh irritants to the canal lower down. If, how-

ever, the stomach be tolerably free, then meat essences, made thin and allowed to stand till cold, may be given. The nausea or vomiting which is usually present is more easily overcome by giving the nourishment cold; and a few drops of lemon-juice are of great service if added to the beef-tea. Milk is very uncertain in the way it is tolerated by such patients. Occasionally it is impossible to give it, the vomiting or diarrhoea being increased by it; but equal parts of milk and soda-water may constitute sufficient nourishment to last for several days in extreme cases, and may be well borne. Lime-water may be substituted for the soda-water, but, as a rule, effervescing fluids are more grateful, koumiss being often of great service. The milk should be sterilised and as free as possible from cream, for fats in all forms are to be avoided, since the products of their decomposition are extremely irritating. Beef-tea or chicken-broth, made with milk in place of water, may be tried with advantage. Farinaceous substances, if given at all, should be only in small quantities at a time; a remark which equally applies to all other food. Nutrient enemata are of much use in some cases.

A very great deal may be done for the patient with drugs, both in the relief of symptoms and in aiding the cure.

It is seldom advisable to check the diarrhoea in acute enteritis; and an aperient to begin with, except evidence exist of there being any peritonitis, is a rational treatment. Thereby the irritant, whatever it may be, is removed, and a better chance for recovery is given; improper food is so commonly the cause, that the majority of cases are benefited by a preliminary purgation. Probably the best aperients in this case are castor oil, and calomel in doses of one to four grains according to age, but it may be necessary to follow up the latter after a few hours with a quickly acting saline aperient. If the inflammation be confined to the colon, where, as already said, accumulations of fæces are the common cause, copious simple enemata, repeated every six or eight hours, are of great advantage; and this plan may be pursued in conjunction with the aperient given by the mouth. The object is to clear out the alimentary canal; and provided that this has been done, abstinence from food for twelve hours, and some bismuth in an effervescing form, are frequently sufficient in milder cases to put the attack on the road to cure. The writer places great reliance on bismuth, either in the form of solution with an effervescing citrate of potassium, and three or four minims of diluted hydrocyanic acid; or granular effervescing lime-juice and bismuth. The nausea and vomiting are best relieved by this treatment. For the pain, poultices or poppy-head fomentations, or the internal administration of opium, may be

very effective. Should there be peritonitis, the opium must be increased in amount, to the end of giving complete rest to the bowel. Several leeches applied to the anus, with the view of relieving the hyperæmia of the intestinal tract, are occasionally necessary in extreme cases. When the attack is distinctly attributable to cold, a profuse sweating induced by hot baths, and ten grains of Dover's powder, is often of great benefit. In those cases where, from the duration, character of stools, and previous treatment, there is reason to believe that the irritating causes are got rid of, the diarrhoea may then require special treatment, especially when the patient has been in ill-health, or is constitutionally debilitated. A powder, consisting of Dover's powder 5 grains, and carbonate of bismuth 10 grains, given every six hours, is very efficacious. Sulphate of copper, nitrate of silver, and vegetable astringents are frequently used for the same purpose.

So soon as the more acute symptoms have subsided, the bismuth may be still continued, and presently given with a vegetable bitter, such as calumba. But ten to fifteen minims of the diluted hydrochloric acid in an ounce of water much assist the recovery of the digestive power of the stomach, for which purpose also pepsin is valuable.

In consequence of the great liability to a second attack which this disease engenders, avoidance of well-known harmful articles of diet, and the use of warm clothing or flannel belts, are demanded as a prophylaxis.

In infants and young children the great liability to collapse, often rapidly fatal, must be borne in mind. Stimulants in some form are almost a necessity. The following prescription may be employed: Liguoris Bismuthi et Ammonii Citratis ℥j-ij, Spiritus Ammonia Aromatici ℥ij-v, Tinctura Cardamomi Composita ℥ij-v; Aqua ʒj-ʒij—according to age. Brandy in small quantities is often the means of saving life in these cases. When the collapse is not threatening, two or three drops of the official solution of corrosive sublimate, with half a fluid-drachm of syrup, and a fluid-drachm and a half of water, every two or three hours, may be of great service. It is a more convenient mode of giving mercury than in the form of grey powder. But in one form or another the writer believes mercury to be of prime necessity. Corrections of diet on the lines indicated above are of course essential. Hot baths and other means to keep the child warm must be employed.

In the severer cases, which are lapsing into the typhoid state, the general principles for that condition must be followed; but, except in such a state, alcoholic stimulants are rarely called for.

Inasmuch as there is good reason to believe that many cases of intestinal inflammation,

especially those of an epidemic character, are due to the activity of micro-organisms, the treatment of the disease on antiseptic principles has naturally been suggested. The problem has been to find a germicide which should pass to the ileum and colon without itself undergoing decomposition in the mouth, stomach, or upper part of the intestine, and should at the same time be harmless to the patient. Many are the agents which have been proposed, and with varying success. Few, if any, are superior to calomel or corrosive sublimate, or the solid preparations of bismuth. Thymol and salol are occasionally useful, but these latter, like resorcin, salicylic acid, salicylate and benzoate of sodium, salicylate of bismuth and cerium, the sulphocarbolates of sodium or zinc, creasote, naphthalin, iodoform, and peppermint, are far more efficacious in checking fermentation in the stomach and upper bowel. Whichever remedy be employed, it should be given in frequently repeated doses according to age. Irrigation of the colon by copious enemata of water, containing small proportions of one or other of the above-mentioned substances, is an effective method of intestinal antiseptics.

#### (B) Chronic Enteritis.

**ÆTIOLOGY.**—1. A certain proportion of the acute cases become chronic, the original cause persisting.

2. Those conditions which lead to a chronic state of congestion of the intestinal tract will thereby so affect the constitution of the tissues, with a consequent disturbance of function, as to constitute a chronic inflammation. The most important of these conditions is obstruction, either at the right side of the heart, or affecting the portal circulation in the liver.

3. Chronic enteritis is the occasional accompaniment of some general chronic disease, such as Bright's disease, when deteriorated blood may be regarded as leading to a chronic inflammation.

4. Residence in tropical climates is a not infrequent cause of lasting inflammatory disease of the bowels.

**ANATOMICAL CHARACTERS.**—The intestinal mucous membrane, when it has been the seat of chronic inflammation, is generally thickened, tough, and of a grey colour, from a deposition of pigment, due to the chronic congestion. The epithelial cells are cloudy and ill-defined, and there is a round-celled infiltration of the mucous and submucous layers passing into the stage of connective tissue; hence the thickness and toughness. This fibrous hyperplasia is often localised as polypoid elevations with intervening areas of atrophic mucous membrane. The lymphoid follicles are prominent and hard; the intestinal glands are frequently blocked with cells and secretion, and form minute solid, though perceptible masses, or are atrophied or even cystic; and the villi of the small

intestine are shrunken and stunted. The surface of the membrane is more or less covered with a viscid glairy mucus, containing pus and imperfectly formed epithelial cells; not infrequently such mucus may be voided in the form of membranous-looking shreds or even complete casts of the tube, and this is particularly the case in the pellicular form of colitis (*see* CASTS). Sometimes the muscular coat is thickened from connective-tissue formation. As a rule, therefore, the bowel is increased in thickness; but in children it not infrequently happens that a chronic enteritis is associated with an atrophy of all the coats and the contained glands, the tube being much thinned and parchment-like, and of a slaty tint. It is unusual for a chronic inflammation of the intestine to exist in adults without coincident ulceration, which is often most extensive; but in children the disease may proceed to a fatal termination, and show no such condition after death.

**SYMPTOMS.**—It is not always easy to say exactly when an acute case has lapsed into a chronic state, very much the same symptoms being continued. In such affections of the small intestine, the diarrhoea may be wholly wanting, and the bowels may be very confined. This is due to the diminished peristalsis, from oedema of the muscular coat and impaired irritability. When, however, ulceration is extreme, and especially if it be the colon or rectum that is mainly affected, chronic diarrhoea is an invariable symptom. The remarks made on the character of the stools in acute enteritis are equally applicable to the chronic state, with the addition, that solid and liquid evacuations frequently alternate. Lasting as the disease often does for many months or even years, a general impairment of nutrition results. The function concerned in the elaboration of the food, as well as that by which the digested products are absorbed, are necessarily perverted. The marasmus is speedily noticed in infants and children, whose growing tissues the less readily withstand malnutrition. Apart from the general ill-health produced, the mental qualities become affected, so that the intellect may become dulled and sluggish, the temper irritable, and the patient may fall into a condition of marked hypochondriasis; this is particularly liable to be the case when the colon is the seat of the disease. The emaciated appearance; the dirty, muddy complexion; complicated often with a short, dry cough, dependent on reflex causes from the stomach, frequently lead a superficial observer to suspect the existence of phthisis.

That form of enteritis which chiefly occurs in the colon, and is characterised by the passage of mucoid shreds and 'casts'—the 'membranous enteritis,' 'mucous colic,' or pellicular colitis of writers, is accompanied

by a fairly definite group of symptoms, and is, the writer believes, of far more common occurrence than is generally supposed. The patients are most frequently women, especially those of a nervous temperament, both single and married. The malady is very persistent, but is particularly characterised by manifesting itself in periodical attacks, with intervals of moderately good health. The attacks, lasting a few days to a few months, may follow an indiscretion in diet, exposure, or mental disturbance, but not unusually occurs without any obvious cause. The symptoms are abdominal pain and tenderness along the course of the colon, vomiting, which is often considerable, and a sense of severe illness, together with great irregularity of bowels, the motions containing much mucus and shreds of various size, with very often some blood.

**DIAGNOSIS.**—The grounds on which a diagnosis can be made are sufficiently obvious from a consideration of the foregoing remarks.

**PROGNOSIS.**—Chronic enteritis almost invariably tends towards a fatal termination, though this may be long delayed. As already said, it is, at least in adults, the colon that is chiefly the seat of this malady, where it is very often associated with ulceration, obstinately resisting all treatment, which at best can only be palliative; the general nutrition becomes more and more deranged; and death from inanition finally terminates an existence of prolonged suffering and discomfort.

**TREATMENT.**—Owing to the unfavourable tendency of this disease, the treatment can be rarely more than palliative. The debilitating and wearying character of the malady emphatically calls for good feeding. The diet should be abundant, no less than nutritious. During the exacerbations of membranous enteritis above described, the giving of food is often difficult owing to the vomiting and pain produced; in such cases koumiss is of great value. When the disease affects the large intestine, the ordinary digestive changes in the food have taken place, and the contents of the canal reach the colon in the normal semi-fluid condition; in this state they may be passed; but owing to the impaired movements of the affected bowel the fæces are apt to accumulate, and constipation results. This should be guarded against by simple enemata, and the soothing effect of injections of warm water only is often very marked. Distinct benefit has been known to follow hydrotherapeutic treatment, especially if accompanied by free rectal injection. In those cases where the enteritis is a sequence of a congestion of the intestine, the treatment must be directed to relieve if possible the cause of that congestion. Since this is usually some such intractable condition as Bright's disease, cardiac dilatation, or cir-

rhosis of the liver, attention should be directed to the relief of these affections.

Tonics—such as quinine, iron, bark, with sea air—are of undoubted benefit; and, so far as possible, causes of mental worry should be removed. Massage of the abdomen and gentle movements specially directed to the exercise of the abdominal muscles are most beneficial.

**14. Intestines, Malformations of.**—These may be (a) *congenital*; or (b) *acquired*.

(a) *Congenital.*—Though seldom of much clinical importance, congenital malformations of the intestines are often of great interest from a developmental point of view. The malformation may be of the nature of an *excess*. Thus certain parts of the canal—duodenum, colon, and appendix vermiformis—have very rarely been found double. The commonest of all these malformations is a diverticulum of the ileum, which may arise from the free margin of the ileum from one to three feet above the ileo-cæcal valve. The cæcal extremity of the process (which is occasionally hammer-shaped) may be connected with the umbilicus by a thin fibrous cord, showing it to be an unobliterated portion of the vitelline duct, and is commonly known as Meckel's diverticulum. It varies in length from half an inch to six inches, or even more; in structure it is exactly that of the ileum; and it has been found the seat of typhoid ulceration, or of perforation from the irritation of foreign bodies that have become lodged therein. In very exceptional cases diverticula of similar structure have been found protruding from the jejunum, and even multiple, but they are not connected with the umbilicus. The vermiform appendix may vary from half to twice the natural size.

*Deficiencies* of development may affect the whole alimentary canal, or only certain parts. Andral records a case where only a straight tube joined the rectum and œsophagus. The ileum may open upon an ectopic bladder. The rectum may end in a cloaca common to the urino-genital organs, or in either the bladder, urethra, or vagina. The bowel may terminate in a closed extremity anywhere between the brim of the pelvis (the rectum being generally represented by a fibrous cord, though even this may be wanting), and immediately beneath the skin; the anal pouch, which develops from without inwards, is in the latter case absolutely wanting; and all degrees between this and a pouch that has just failed to establish a junction with the rectum may be met with, producing the lesion known as *imperforate anus*. The valvulæ conniventes are sometimes wanting, or very imperfect, over varying areas of the small intestines. Congenital constrictions of different parts of the canal are occasionally met with—in the duodenum, either close to the opening of the common

bile-duct or at the junction with the jejunum; in the lower end of the ileum, where some abnormality in the closure of the vitelline duct appears to be the cause; or in the sigmoid flexure. Such constrictions may be multiple and of very short extent, the canal being much dilated above, and extremely narrowed and shrunken below. The ileo-cæcal orifice has been seen contracted to the diameter of a small cedar-pencil. The cause of these lesions is very obscure, though they may be occasionally accounted for by the existence of prominent valve-like folds of the mucous membrane.

Hernial protrusions of the mucous membrane through the other coats, often very numerous, and varying in size from a pin to a walnut, have been seen in the colon, and less often in the small intestine, sometimes extending into the appendices epiploicæ. They are liable to become developed in cases of long-standing constipation.

(b) *Acquired*.—The acquired malformations include the dilatations and contractions that are associated with stenosis; and the adhesions and abnormal communications established by ulceration and peritonitis, elsewhere referred to.

When any symptoms are produced by malformation of the intestines they are usually those of obstruction; and the only condition that may be amenable to treatment is imperforate anus. See **INTESTINAL OBSTRUCTION**; and **ANUS, Diseases of**.

**15. Intestines, Malignant Disease of.**—The new-growths which are met with in connexion with the intestine may, for the present purpose, most conveniently be divided into *malignant* and *non-malignant*—a clinical distinction irrespective of their minute structure.

**ÆTIOLOGY.**—Malignant growths of the intestine, as in other situations, whilst not wholly unknown in the earlier periods of life, are rarely met with before the age of forty and oftener after fifty. Statistics show a slight preponderance of occurrence in males. From an examination of 9,000 fatal cases of cancer, the relative frequency of intestinal cancer to that of all other organs was found to be as 1 to 25 (Tanchou).

Cancer of the intestine is nearly always primary, and very frequently runs its course without any secondary formations elsewhere. Occasionally the bowels are affected by extension from neighbouring parts, and this is especially liable to be the case in the rectum, when the uterus or vagina is the seat of the disease, and in the duodenum, which may become involved in an extension from the pancreas, liver, gall-bladder, or stomach. Very rarely small nodules are found in the solitary and agminated glands secondary to carcinoma existing elsewhere.

**ANATOMICAL CHARACTERS.**—Malignant disease may occur at any spot throughout

the entire length of both small and large intestine, but is infinitely more often to be met with at certain special parts, notably the rectum, sigmoid flexure, cæcum, colon generally, and duodenum near the opening of the bile-duct; the jejunum and ileum being rarely affected. It has been estimated that in 80 per cent. of the cases of intestinal cancer the rectum is affected; in 11·5 the colon; in 4·2 the cæcum, appendix, and ileo-cæcal valve; and in 4·3 the small intestine. There is undoubtedly a predilection for those spots where any delay may occur in the passage of the intestinal contents, such as the flexures of the large intestine, especially the sigmoid.

The greater number of intestinal growths included in the category of malignant are of the epitheliomatous type, especially the cylindrical and glandular varieties; somewhat less frequent is the colloid form; whilst the scirrhus and encephaloid are much rarer. As a rule, malignant growths commence in the mucosa and submucosa, and then gradually involve the other tissues. The microscopic characters of these new-growths present no special features. See **CANCER**.

The mesenteric glands are generally affected, though often but slightly. Secondary infection is much less common than in cancer of other organs; when present, the liver and peritoneum are the usual seats.

Following the general course of these neoplasms when found elsewhere, they may undergo degeneration and ulceration, thus suffering a diminution in bulk at one spot whilst they extend in other directions. In the course of their development they may set up adhesions between the bowel and other parts; and two or more coils of intestine may be thus involved, and fistulous communications established between them. The colloid form is the more liable to invade the peritoneum, where large gelatinous masses may be developed.

The new-formed tissue may constitute an irregular mass of very variable size and extent, of a nodulated or of a villous appearance, perhaps partially ulcerated, and extending into the passage of the canal, producing an obstruction. Or the new-growth may develop in an annular manner, involving the whole circumference of the bowel; the obstruction produced in such circumstances may be extreme, even to narrowing the lumen of the tube to barely the size of a probe. The extent of obstruction may be altered by partial destruction of the new-growth by sloughing, though the subsequent cicatrices that may result will again constrict the gut.

**SYMPTOMS.**—For a varying time before this disease definitely asserts itself, the patient may complain of vague dyspeptic symptoms; a sense of uneasiness in the abdomen, not amounting to pain, and usually increased after

meals; and marked irregularity in the action of the bowels, with or without flatulent distension, and an increasing tendency to constipation, for which the patient has often taken enormous quantities of aperients of a drastic and cathartic character, which one by one lose their effect. The persistence and gradual increase of these symptoms, especially if there be any loss of flesh, is very significant, and should excite suspicion. Sooner or later, according to the duration of the case, the usual cachexia is established; and in the greater number of cases the patient rapidly emaciates, especially towards the end, though in cases of very short duration the wasting may not be so excessive. The emaciation depends not only on the general perversion of nutrition caused by the development of the cancer, but also on the direct influence it exerts on organs concerned in the digestion and absorption of nutriment; hence the higher up in the bowel the growth is situated the sooner is the loss of flesh determined.

The local signs and symptoms referable to the new-growth itself are very variable in their occurrence, and often are singularly slight in comparison with the gravity of the cause. Thus pain may be completely wanting, and perhaps there is but little tenderness on pressure; when present the pain is usually of a dull character, and quite localised.

The indications of the tumour produced by the new-growth are very uncertain, being often little more than an ill-defined fulness in one region, scarcely even amounting to that in the annular form of cancerous stricture; at other times presenting a distinct hard irregular mass of variable size. This last quality, being partly dependent on fæces, is very characteristic. Should the growth happen to be situated over the aorta or iliac arteries, an indistinct pulsation may be communicated to it. The percussion-note over the tumour is usually imperfectly tympanic, from the existence of coils of intestines between it and the abdominal wall, the thickness of which will of necessity considerably modify the signs of the existence of the growth. The mass may present all degrees from free mobility to complete fixity, dependent on the nature of its seat, and also on the existence of adhesions to neighbouring parts.

Symptoms of intestinal obstruction are rarely wanting, and may be the first for which the patient seeks relief. Vomiting, constipation of increasing severity, with signs of intestinal distension above the lesion, are among the most constant. The character of the vomit will largely depend on the duration and situation of the lesion, and the actual degree of obstruction. When the jejunum or lower part of the duodenum is the seat of the growth, the vomiting is more copious and constantly bile-stained, the bowel and stomach above the stricture sharing in the distension.

When the obstruction is lower down, in the large intestine, the vomiting may be slight, and consist of little more than the gastric secretions, with such food as is taken, only becoming stercoraceous when the obstruction becomes complete. Occasional diarrhœa, determined by the chronic enteritis which exists, may alternate with the constipation; and rupture of the intestines, especially the cæcum, has been met with as a result of the atrophy and ulceration of the over-distended, chronically inflamed bowel. As in other forms of intestinal obstruction, the amount of urine voided is likely to be very much diminished when the stricture is high up, and the vomiting is excessive. See **INTESTINAL OBSTRUCTION**.

The stools are usually characteristic of the obstruction, consisting of small separate masses, frequently hard and round, or flattened or 'pipe-stem' like, and often mixed with sloughed-off portions of the new-growth, or with blood that has escaped from the ulcerated surface. The nearer to the anus the growth is situated, the less change will there be in the blood, which sometimes may be considerable in amount.

If the peritoneum be involved, peritonitis may arise; and ascites, often considerable, is usually developed with colloid cancer. Superadded to these symptoms will be those caused by the morbid condition of any other organ that may be affected, such as the liver, bladder, or uterus.

**COURSE AND TERMINATIONS.**—Malignant disease of the intestines in the majority of cases progresses continuously. It is difficult to state even an average duration, owing to the insidious onset and vagueness of the first symptoms; but the greater number of cases rarely go beyond twelve to eighteen months from the time when the disease is clearly established, whilst some may be fatal in a few weeks, and a few may last for years.

Death may result as the direct consequence of the cachexia; or from hæmorrhage, peritonitis, or other effects of the growth.

**DIAGNOSIS.**—An exact diagnosis is often not to be made, and the nature of the case remains throughout uncertain, if not actually as to the existence of a malignant growth, at least as to the seat of it. The insidious and ill-defined character of the earliest symptoms presents nothing diagnostic, though their progressive character and resistance to treatment would cause a suspicion, especially in a person over middle age, and in whom a gradual even though slight loss of weight is noticed. Even in the later stages, the symptoms are almost identical with those of chronic enteritis, which really co-exists with the new-growth to a greater or less extent; and in the not infrequent cases in which a tumour is not to be felt, or is uncertain in its indications, the diagnosis becomes extremely difficult. Some cases also in which the new-

growth is unrecognisable to palpation, and at the same time causes little or no obstruction, closely simulate in their course diseases of the suprarenal capsule; for the latter are not invariably accompanied by cutaneous pigmentation, and the rapid and progressive emaciation, with more or less persistent vomiting, may be common to both. It is true that the malignant cachexia is frequently productive of a characteristic *facies*, but this would equally occur in cancer of the capsules.

Supposing that the existence of an abdominal tumour be clearly ascertained, it is not always easy to determine its connexion with the intestine, since the variability in position, in mobility, and in size (due to the accumulation, or the reverse, of feces), precludes any diagnostic sign, although this very variability is regarded by some as almost indicative of intestinal cancer. In distinguishing between an intestinal tumour and one connected with the liver, pancreas, kidney, mesenteric glands, uterus, abdominal wall, or the inflammatory new-growth following a perityphlitis, an aneurysm, or a simple fecal accumulation, the history of the case, age, progressive nature of the condition, existence of tumour, signs of obstruction, and character of the stools, are the points to be considered in forming a diagnosis. Any one or even two of these points might equally indicate other lesions, but, taken collectively, they will usually justify the formation of an opinion. A rectal cancer, which is accessible to the touch or even to inspection, need offer no difficulty, but it is otherwise where it comes to distinguishing a duodenal growth from one strictly limited to the pylorus. The vomiting in the latter case is more persistent than in the former, and there is a greater liability to hæmatemesis; but these are most uncertain signs, as also is the existence of jaundice, which oftener complicates the duodenal affection, from the greater chance of involving or pressing on the bile-duct; but jaundice is not a necessary accompaniment. A firm epigastric tumour, felt close to the margin of the thorax, and associated with distinct dyspeptic symptoms, may also be due to primary cancer of the head of the pancreas; in such case the symptoms of obstruction are less marked at first, but the growth will probably involve the bowel in its progress, and cause stenosis; owing to the destruction of the gland-tissue, the pancreatic juice is not secreted, and undigested fat may be found in the stools.

Similar difficulties may surround the investigation of a tumour situated in the right iliac fossa. Emaciation, constipation, and melæna might equally indicate a malignant tumour of the cæcum, or the remains of a chronic perityphlitis.

**TREATMENT.**—The extremely rare cases of reputed natural cure of malignant disease of

the intestines, brought about by sloughing of the growth and subsequent cicatrization of its site, afford no hope of our being able to artificially imitate the process, and the treatment remains at the best symptomatic and palliative.

The diet should be so arranged as to contain the minimum of indigestible residue, and permit the chief digestion and absorption in the stomach, if it be the upper part of the tube that is affected. But in the majority of such cases the utmost disinclination from food exists, even apart from any vomiting or pain its ingestion may produce, and hence, whatever the directions, the patient in the later stages practically takes nothing. The anorexia is frequently as marked even when the mischief is seated in the colon, and the area for digestion and absorption is uninterfered with.

In the earlier stages it may be advisable to insist upon as much nutritious food as possible being taken, either by the mouth or in the form of enema or suppository, so as to offer the most prolonged resistance to the inevitable end; but at the same time there is no slight reason for thinking that the same course favours the speedier development of the new-growth. Preparations of iron may be given with the same view.

The symptoms dependent upon the obstruction we can do very little to relieve short of operation. Only the mildest aperients are permissible, to combat the constipation, and these should be salines with the object of rendering the feces as fluid as possible; while the vomiting is as a rule uncontrollable, and, indeed, is often a relief.

Hæmorrhage may require special attention, the tincture of hamamelis being among the most effective styptics; and the pain may be so severe as to necessitate free administration of morphine subcutaneously; in other cases belladonna is of value in alleviating the local discomfort, and acts favourably by allaying any spasm.

The operative treatment of malignant disease of the intestine is described in the article **INTESTINAL OBSTRUCTION**.

**16. Intestines, Malpositions of.**—Displacements of the intestines, like malformations, are both *congenital* and *acquired*.

Among the former may be mentioned complete transposition of the viscera, the cæcum and ascending colon being on the left side, and the sigmoid flexure and descending colon on the right, the liver, spleen, stomach, &c., sharing in the change. Certain parts only of the intestinal canal may occupy an abnormal situation, as seen in the various congenital herniæ; or the displacement may be due to unusual length of the mesenteries, the cæcum and sigmoid flexure being the parts that present the most usual alteration from this cause. Thus the cæcum may occupy the left hypochondrium or left iliac fossa, or

be found in the pelvic cavity, and, of course, other parts of the canal must correspond to these malpositions. The sigmoid flexure has been seen lying to the right side of the left kidney, which was situated immediately below the bifurcation of the aorta. Similar displacements are referable to adhesions, determined by intra-uterine peritonitis, which is frequently associated with syphilis.

The malpositions which the intestines may come to present from changes set up after birth are so variable as scarcely to admit of classification. Hernia, both external and internal, volvulus, and intussusception are among the well-recognised displacements; but there is scarcely any limit to the changes in position which the traction and pressure of tumours and the effects of peritonitis may produce. See **INTESTINAL OBSTRUCTION**; and **HERNIA**.

**17. Intestines, Micro-organisms of.** The intestinal canal from within a very few hours after birth, to judge from the fæces, swarms with numerous forms of microbes, which, having gained entrance with the food and air, find in the canal suitable conditions for their development. Our present knowledge would lead to the belief that many of the species have no effect upon the human body, whilst others take an active share in intestinal digestion, as effective though wholly superfluous aids to the pancreatic secretion. How far any of these, which may be regarded as normal inhabitants of the canal, may under certain conditions of the contents or excessive development, &c., become responsible for symptoms of disease, is not known with certainty, though it appears probable such may be the case. But it is now well recognised that certain specific diseases, such as tuberculosis, cholera, and typhoid fever, are connected with specific organisms which, gaining admission with the ingesta to the alimentary canal, produce the characteristic symptoms of these several diseases.

Besides various micrococci, the most constant members of the schizomycetes found in the intestines are the bacillus lactis aërogenes, chiefly in the upper part of the canal, and the bacillus coli commune, most abundant in the colon. Certain other species, such as Brieger's bacillus or bacillus cavida, Bienstock's putrefactive or 'drumstick' bacillus (?), a putrefying green fluorescing bacillus, and various spirilla, are met with in the evacuations of patients suffering from different forms of enteritis; and it is probable that the poisonous substances which these organisms form from the intestinal contents, especially the albuminous, take a large share in the causation of the disease, or at least that many of the symptoms are to be referred to the ptomaines which are absorbed. But our present knowledge does not enable us to associate with each of the multiform diarrheal conditions which occur its own specific

causal organism, as we can do in the case of cholera or typhoid fever.

Actinomycosis has been met with in the intestine, where it forms raised whitish patches, setting up inflammation, and proceeding to abscess, which may burst into the peritoneum or present at the integuments. As the disease spreads, some healing may take place. See **ACTINOMYCOSIS**; and **MICRO-ORGANISMS**.

The bacillus anthracis also occurs in the intestine, causing swollen œdematous patches of the mucous membrane, which are intensely congested and rapidly slough. The intestinal lesion may be primary, or it may be part of a general infection. See **ANTHRAX**.

A few species of the yeast fungi or saccharomycetes, such as torula, and of the hyphomycetes or moulds, such as the oidium albicans or 'thrush,' occasionally develop in the intestines.

**18. Intestines, Morbid Growths of.** Owing to the variety of tissues that enter into the formation of the intestine, no less than to the origin of these tissues from two of the three primary layers of the blastoderm, the new-growths that may develop in connexion with it are exceedingly numerous.

It will be convenient here to consider them from their clinical rather than from their genetic or histological point of view, and to divide them into *malignant* and *non-malignant* growths. The former have already been treated of, and the special neoplasms of tubercular and syphilitic origin are more conveniently referred to separately.

**VARIETIES.**—1. *Fibromata.*—These growths, which are developed from the connective tissue of the submucous coat, are usually of small size, frequently pedunculated, though sometimes appearing as sessile, flattened nodules, of half the size of a pea, projecting into the canal. The smaller ones may be scattered throughout the length of the bowel, whilst the larger ones (up to the size of a walnut) are fewer in number or single, and are usually found in the rectum. They present the ordinary microscopic characters of fibrous tissue.

2. *Lipomata.*—Pedunculated growths of adipose tissue, springing from the submucous coat, are not of uncommon occurrence in any part of the intestines.

3. *Myomata.*—Very rarely small growths are met with, chiefly composed of contractile fibre-cells, with a variable amount of connective tissue.

4. *Vascular tumours.*—*Angiomata.*—Besides hæmorrhoids, other vascular growths are sometimes found, of an erectile character, similar to nævi of the skin.

5. *Mucous growths.*—It is to these growths that the term 'polypi' is generally applied. They essentially consist of the tissues of the mucous membrane, though differing in their vascularity, and also in their glandular ele-

ments. When these latter are excessive in amount, they are liable to present characters which connect them with malignant forms of new-growth, especially if their surface assume a villous character. Polypi are not limited to any one part of the canal, though undoubtedly they are most common in the rectum, and are sometimes pigmented. They are occasionally multiple, 170 have been seen in the lower half of the colon in a single case; and have been met with at all ages.

6. *Lymphoid growths.*—*Lymphadenomata.*—Neoplasms whose structures correspond to that of the solitary or agminated glands, or of the lymphoid layer of the submucosa, are met with in association with these normal constituents of the intestinal wall, and quite independently of leucocythæmia. Nodular formations and diffuse infiltrations of similar structure have been met with throughout the stomach and intestines in Hodgkins' disease (lymphadenoma), but though giving rise to large masses, they produced no obstruction.

7. *Sarcomata.*—Small nodules of this type of neoplasm have been described as of very rare occurrence in the intestine.

8. *Cysts.*—These have been rarely met with, and the contrast to their comparative frequency in the uterine mucous membrane is remarkable. Several instances are on record of congenital dermoid cysts which have been found attached to the rectum or sigmoid flexure, and probably originating from the neurenteric canal.

Cystic enlargement of the vermiform appendix, or of diverticula of the intestine, may follow on closure of their communication with the lumen of the bowels, and accumulation of fluid in their interior—*enterocystomata.*

EFFECTS AND SYMPTOMS.—As a rule, the growths mentioned above present very little interest, unless they be situated just within the rectum, and accessible to digital examination, for it is seldom that they are of sufficient size to form tumours which can be felt in the abdomen. They cannot be diagnosed, though they may give rise to certain symptoms, as, for instance, hæmorrhage from the vascular polyp and erectile tumours, or partial obstruction if they attain any size; but such symptoms are not diagnostic. One of their most striking effects appears to be the liability that the polypoid forms occurring in the small intestine have of inducing serious intussusception, from interfering with the due progress of peristalsis. Prolapse of the rectum is similarly found to be occasionally due to polypi.

TREATMENT.—No treatment can be attempted, beyond that of the symptoms which may arise; or the removal of growths within reach of the anus, or very exceptionally by laparotomy.

19. *Intestines, Paralysis of.*—The peristalsis of the intestinal tube is normally dependent on automatic nerve-impulses, originating in the intrinsic ganglia of the canal, controlled by both reflex and direct impressions from the cerebro-spinal and sympathetic systems, the former acting *viâ* the vagus in an accelerating manner, the latter *viâ* the splanchnics in an inhibitory direction. The integrity of the involuntary muscular fibre is assumed. The quantity and the quality of the blood in the vessels of the intestines also appear to modify peristalsis, since the movements are increased both when there is anæmia and hyperæmia of the canal, probably owing to the diminution of oxygen and excess of carbonic acid, which these conditions equally determine.

ÆTIOLOGY AND PATHOLOGY.—A paresis of the intestinal movements may be brought about by causes acting (1) through the *nervous system*; or (2) through imperfection of the *muscular tissue.*

(1) *Nervous.*—Cases are recorded in which the intrinsic ganglia and nerves of the intestinal muscular coat have been the seat of degeneration, but the symptoms dependent thereon are doubtful, beyond atrophy of the muscular coat. Certain lesions of the brain are accompanied by symptoms of intestinal paralysis, but with no hitherto recognised regularity, and it is assumed that such lesions act by interfering with the function of the vagi. It is doubtful how far disease of the spinal cord produces actual paralysis of the intestines, though constipation may result, a circumstance that may be explained by assuming an interference with the centre that controls defæcation. See FÆCES, Retention of.

(2) *Muscular.*—The irritability of the muscular tissue may be much weakened by degeneration (cloudy, fatty, or lardaceous). Inflammation of the mucous or serous coat, especially the latter, is liable to determine granular change in the muscular fibres, which, aided by a coexistent œdema, largely impairs the contractile power of the tissue. Chronic congestion of the bowel leads to the same result. The irritability is also liable to suffer from the over-stimulation of too powerful and too frequent purgative medicines; and the muscular fibres of a much dilated portion of the bowel are apt to become paralysed from distension and stretching. The general want of tone that the muscular and nervous systems manifest subsequent to debilitating diseases, or from want of food, hysteria, and other conditions, also finds expression in the alimentary canal, in diminished peristaltic action.

Certain astringent drugs produce their effect possibly by diminishing the excitability of the nervous system, as appears to be the case with opium. Lead, which would seem to cause both paralysis and spasm of the

muscular coat, may act on the nerve terminals or on the muscular fibres.

**SYMPTOMS.**—The prominent symptom of intestinal paralysis is constipation, though other signs of obstruction, such as vomiting and meteorism, may be superadded. See CONSTIPATION; and INTESTINAL OBSTRUCTION.

**TREATMENT.**—As a rule, this is directed to the primary cause, but great benefit has followed the application of electricity to the abdominal parietes. Friction applied on systematic principles is of undoubted service.

**20. Intestines, Parasites of.**—Besides the micro-organisms referred to in a previous article, there are frequently to be found in the intestines various species of animal parasites which mostly obtain their nourishment from the contents of the canal. The majority of these organisms, with the symptoms they give rise to and their treatment, are described under ENTOZOA, and are merely enumerated here collectively.

**Class RHIZOPODA.**—Under the name *amœba coli* are possibly included several distinct species which extended knowledge may differentiate. These animalcules have been occasionally found in healthy fæces, and more often in dysenteric stools, in dysenteric ulcers, as well as in the hepatic abscesses which so frequently complicate this malady (see DYSENTERY; and ENTOZOA). Various monads have been found in the stools, both in health and disease.

**Class INFUSORIA.**—The best known of these are *cercomonas intestinalis*, *trichomonas intestinalis*, and *paramœcium coli*. They commonly occur in diarrhoeal states, which they may help to intensify, though not actually cause; and their presence may be generally taken to indicate an unhealthy state of the intestinal mucous membrane.

**Class CESTODA.**—*Tœnia mediocanellata* v. *saginata*. *T. solium*. *T. nana*. *T. cucumerina*. *T. flavopunctata* (?). *Bothrioccephalus latus*. *B. cordatus*.

**Class TREMATODA.**—*Distoma hepaticum*. *D. lanceolatum*. The spinous ova of bilharzia hæmatobia have been found in the mucosa and submucosa of the rectum, causing much irritation, even to the formation of polypoid overgrowths.

**Class NEMATODA.**—*Ascaris lumbricoides*. *A. mystax*. *Oxyuris vermicularis*. *Ankylostoma duodenale*. *Tricocephalus dispar*. *Trichina spiralis* (mature stage). *Anguillula intestinalis*.

**Class INSECTA.**—Very rarely the ova and larvæ of certain insects, chiefly of the family Muscidae, being swallowed, are found in the canal.

**21. Intestines, Perforation and Rupture of.**—**ÆTIOLGY.**—The causes of perforation or rupture of the intestines may be arranged thus: (1) *External injuries*, such

as blows, being run over, &c., which, though more liable to rupture the solid abdominal viscera, frequently cause laceration of the intestines, especially the ileum or jejunum.

(2) *Corrosive poisons*, when swallowed in any considerable amount, may destroy not only the walls of the stomach, but also those of the upper part of the intestines.

(3) *Extreme distension by flatus or fæces* above the site of a constriction may cause the bowel to burst. It is the cæcum which usually gives way in chronic obstruction, a clinical fact which has received experimental confirmation.

(4) *Ulcerations*, pre-eminently the so-called peptic ulceration, and less commonly typhoid and catarrhal ulceration, may lead to intestinal perforation.

(5) *Perforations* may be produced *ab extrâ*, by the bursting of abscesses or aneurysms into the canal.

**SYMPTOMS.**—The most striking symptoms which perforations of the bowel present, are a sudden attack of severe abdominal pain, followed by vomiting and collapse; and it is a noticeable fact that rupture of the hollow abdominal viscera is more liable to induce this condition than a similar lesion of such organs as the liver or spleen. Rigors are of occasional occurrence at the onset, and the pulse is quickened, with sometimes a rise in temperature.

Should the patient live twenty-four hours after the establishment of a perforation, signs of peritonitis will assert themselves—severe abdominal pain and tenderness, pyrexia, vomiting, and other symptoms. Supposing that the perforation follow an ulceration in the course of a previously high temperature, such as enteric fever, there is sometimes, though not always, a sudden and considerable fall in the body-heat; this, with the acute pain, may be the first indication that perforation has taken place.

Perforation or rupture of the intestines usually proves fatal within forty-eight hours of its occurrence, although cases are recorded which have lasted for weeks; very rarely recovery has taken place.

There are no reliable signs whereby rupture of the stomach may be distinguished from that of the intestines, nor is it of any practical importance.

**TREATMENT.**—Rest is of primary importance, in regard both to the whole body and the bowels themselves. This object is best attained by the free use of opium, commencing with a grain, and repeating it in a few hours until its influence is fully established. It is also desirable to stop all food, except an occasional teaspoonful of meat-essence; to give ice to suck; to administer nutrient enemata, and brandy and ether subcutaneously if the collapse be profound; and to apply warmth to the extremities. Resection of the ruptured portion of the bowel, with

suturing of the divided ends, and washing out of the peritoneum, has been performed with success, and should be attempted in suitable cases.

**22. Intestines, Spasm of.**—The irregular and forcible movements of the bowels, usually accompanied with pain, are known as *colic*. Under ordinary circumstances we are unconscious of the peristaltic action, but when the contractions of the muscular coats become violent, more or less pain is likely to occur. See **ENTERALGIA**.

**ÆTIOLOGY.**—The determining causes of intestinal spasm are:—

(1) The direct irritation of indigestible ingesta. (2) Exposure to cold. (3) Certain poisons, such as lead and strychnine, which probably affect the muscular fibres through the nervous system. Associated with lead colic, even if it be not actually responsible for it, is arterial spasm and high tension in the splanchnic area.

It is difficult to ascertain the exact condition of the contracted bowel, since the appearances seen after death are not an index of what existed during life; but it would seem that the spasm may start from several points in the course of the canal, and, after persisting for a variable time, either yield or travel on as a wave of spasmodic contraction. For how long a portion of bowel may remain contracted is quite conjectural.

**SYMPTOMS.**—The existence of an absolutely painless spasm of the intestines is very doubtful; as a rule, it is the pain which indicates this condition, and except the contraction be maintained, no further symptoms may occur. There are, however, conditions in which the tonic intestinal spasm is considerable, and yet the pain is but slight. Such is the case in basilar meningitis, where the bowels are firmly contracted and empty, producing the characteristic boat-shaped appearance of the abdomen. In many cases of chronic enteritis and colitis marked spasm may occur, causing great irregularities of the abdomen from distended and contracted lengths of bowel, with constantly varying differences in position and degree of dullness or resonance. Constipation, vomiting, meteorism, may all be present in varying degrees, dependent upon the extent of obstruction which is produced.

The specially painful spasm of the anal sphincters and lower portion of the rectum, termed *tenesmus*, is usually associated with ulceration and other lesions in that locality.

**TREATMENT.**—The external application of moist heat in the form of poultices or fomentations, preferably of poppy-head or other opiates, is of great value, for relaxing the spasm or removing the pain. Since an irritant is so frequently the cause, an aperient, such as castor oil or calomel, combined with opium, is essential. Turpentine enemata are sometimes of great service. The collapse

may be so severe as to call for energetic stimulation by brandy, ammonia, or ether.

**23. Intestines, Syphilitic Disease of.** The intestinal canal is rarely the seat of the specific lesions of syphilis, except at the lower end of the rectum, and margin of the anus. Small gummata have been found in the submucous tissue of various parts of the bowel, and sometimes the ulcers to which these growths give rise by their degeneration and breaking down; radiating fibrous thickenings and cicatrices of the mucous membrane have also been seen in syphilitic subjects, sufficient even to cause obstruction. It is doubtful whether there be any specific ulceration of the intestine which is not preceded by gummata, although small ulcers do occur in new-born children, the subjects of congenital syphilis.

Syphilitic ulceration and stricture of the rectum are not of infrequent occurrence, the former especially in women, where the infection has spread from the vagina and perinæum. See **RECTUM, Diseases of**.

**24. Intestines, Tubercular Disease of.**—The specific lesions of the tubercular diathesis, namely, grey granulations or miliary tubercles, are of frequent occurrence throughout the intestinal canal; the extensive distribution of lymphoid tissue throughout the submucous coat, and the special aggregations of it which form the solitary and agminated glands, offering the most favourable opportunity for their formation. The abundant supply of lymphatic vessels in the thickness of the walls of the canal, and the close connexion of the serous surface with the lymphatic system, all predispose to the development and spread of a tubercular growth which may have become established in a subject of the diathesis.

**ÆTIOLOGY.**—As a primary growth, tubercle very rarely attacks the intestines in adults, though it is of very frequent occurrence in children as part of a general tuberculosis, or possibly from the consumption of milk from tuberculous cows. In adults, on the contrary, tubercular disease of the intestines is very commonly developed secondary to a similar affection of the lungs, due probably to the swallowing of the sputum.

**ANATOMICAL CHARACTERS.**—The submucous layer and the peritoneal coat are the structures in which the tubercle originates; in the former situation it especially favours the ileum and cæcum, although it may develop throughout the entire length of the tube, whilst the peritoneal tubercle is about equally distributed. The mesenteric glands are always considerably involved. The rarity with which the stomach is affected by tubercle is in marked contrast to the frequency of the intestinal lesion.

In extreme cases of tuberculosis in children, death may take place before any changes in the tuberculous formation have

taken place, and countless grey granulations, from the size of a pin's head to bodies quite microscopic, are to be found in the sub-mucosa, and in the solitary and agminated glands. Later on, however, these non-vascular new-growths coalesce, and form distinct masses, which from lack of nutrition undergo caseous degeneration and break down, thus forming the tubercular ulcers. The ulcers tend to spread, and rarely to heal, and whilst they may be at first limited to the glands, they invade the adjacent mucous membrane, especially in a direction round the bowel, their extension being preceded by the development of fresh tubercles, chiefly along the course of the main blood-vessels, to the progressive formation and destruction of which the spread of the ulcer is really due. Large masses of the mucous surface may be thus destroyed, leaving a ragged, flocculent surface, formed of the muscular fibres, or even of deeper structures, which lesions rarely proceed to perforation into the peritoneal cavity, adhesive peritonitis having established attachments to adjacent parts. The thickened, congested, irregular edges of the ulcers, with miliary tubercles close to the margin, are very distinctive.

**SYMPTOMS.**—Until ulceration be established, there will be no symptoms of tubercular disease referable to the intestinal canal; and even when this stage is reached, there are no special symptoms.

**TREATMENT.**—Little can be done for intestinal tuberculosis. The course of the disease is almost invariably to a fatal end, and it is very rare for healing and cicatrization to take place. The necessity for feeding the patient is almost contra-indicated by the existence of a destroyed digesting and absorbent surface, whereby the food becomes a positive irritant. Such nourishment as is taken should therefore be in the most digestible and concentrated form, that as much as possible may be taken up from the stomach. Starch and opium enemata may do a little to check the diarrhoea, but their efficacy is soon lost. Hæmorrhage, should it set in, is scarcely amenable to treatment, though astringent enemata may be of some use, combined with the internal administration of acetate of lead and opium. No treatment has as yet been effectual in arresting the spread of tubercle.

**25. Intestines, Ulceration of.**—Ulceration of the intestinal wall, from one cause or another, is of extremely common occurrence. The morbid processes involved in the production of the ulcers are in all cases essentially the same, namely, a molecular death and disintegration of the tissue, leaving a solution in continuity, of varying extent.

**VARIETIES :** (1) *Primary inflammatory ulcers.*—Any enteritis, whether of the mildest catarrhal character, or of a specific type such

as diphtheritic, dysenteric, or typhoid, may lead to ulceration of the bowels. As a rule, the more severe the cause of the inflammation, the greater the liability to this complication; and the same holds in respect of any intestinal catarrh, developed in the course of any serious state, such as typhus fever or Bright's disease. The ulcer may appear either as a small abrasion of the epithelial layer, which gradually extends and deepens until the whole mucosa is involved; or the first indication may be a thin glairy pellicle, adherent to the mucous membrane which in time is thrown off, leaving a breach in the subjacent tissue. In other cases the destructive process commences in the thickness of the bowel, either from the rupture of small collections of inflammatory products, resulting from an enteritis, or from inflammation of the lymphoid follicles. The escape of these products into the tube leaves behind an ulcer.

These lesions may be found anywhere throughout the bowels, although they are much more frequent in the large than in the small intestine; and one form of follicular ulceration, associated with the specific poison of dysentery, is practically limited to the former situation. The colon is also—especially in elderly persons—the seat of a very extensive simple ulcerative colitis, in which the mucosa is destroyed over large areas, but which does not commence in the follicles. At those places where any delay is likely to arise in the passage of the fæces—the cæcum, sigmoid flexure, and rectum—and at those spots which are most prominent, such as the edges of the valvæ conniventes, and the sacculæ of the colon, where an enteritis is most likely to be produced, there will be the probable site of inflammatory ulcers. The specific ulcers of dysentery and typhoid fever are described elsewhere. In cholera, ulceration of the lower end of the ileum and colon is sometimes met with.

(2) *Ulcers resulting from the separation of necrosed tissue.*—The process of molecular disintegration which takes place along the line between living and dead tissue, resulting in the separation of a slough and the leaving of an ulcer, takes place in the intestines as elsewhere. The causes leading to the death of circumscribed areas of tissue are various. Sometimes the vitality of a portion of the mucous membrane is destroyed by degeneration, such as the lardaceous, and an ulcer marks the spot of the removed patch, which usually occurs in the ileum or colon. More frequently the local death is induced by capillary hæmorrhage or a cessation of blood-flow through a limited area; the cause of this stasis is not very apparent, though believed to be due to emboli. Under such circumstances the solvent power of the digestive juices may be exerted on the non-living tissues, which are thus removed, and

an ulcer is left. To such ulcers the term *peptic* has been applied, and identical lesions are met with in the stomach. They almost invariably occur in the first part of the duodenum, above the point of entrance of the alkaline bile and pancreatic juices, although very rarely they have been seen in the jejunum. Ulcers of this character appear to be occasionally connected with large superficial burns, but how the relationship is established is not known. It is a singular fact in regard to them, that they are ten times more common in men than in women, which is quite the reverse of what obtains in the stomach, although the relative frequency of gastric and duodenal ulcers is estimated as thirty to one.

(3) *Ulceration of new-growths.*—Almost any neoplasm of the intestinal wall may ulcerate, though as a rule the more rapidly developed forms are the more liable. Tubercular growths primarily connected with the solitary and agminated glands invariably end in this manner. Syphilitic gummata and malignant growths are especially prone to ulceration.

**CHARACTERS.**—The appearances presented by the various ulcers differ with the cause and the duration.

They may be single, as is generally the case with the duodenal ulcers; or innumerable, as the follicular ulcers of the colon. Typhoid and tubercular ulcers are as a rule multiple, and are most numerous at the lower end of the ileum, where the agminated glands are most abundant. Occasionally large surfaces of the mucous membrane are destroyed, with here and there small isolated spots of the membrane left, due to the spread and coalescence of many separately arising ulcers. In dysentery and chronic tubercular ulceration this is especially liable to happen. Many of the catarrhal and follicular ulcers are extremely small, not more than a line in diameter.

The peptic ulcers are distinguished by their very definite, 'clean-punched' appearance; the edges are slightly sloping, and but very little, if at all, thickened; whilst the mucous membrane immediately adjacent has a perfectly healthy appearance. In most of the other varieties the edges are thickened, irregular, and shaggy, frequently excavated and undermined; the ulcerative process extending beneath the mucous membrane, which gradually dies and sloughs away as its nutrition is cut off. Dependent upon the depth and course of the ulcer will be the nature of its base, which may be formed of the muscular coat, of the peritoneum much thickened, or of adjacent structures with which adhesion has been established, such as the liver or abdominal wall. The floor of the ulcerated tubercular and malignant new-growths usually presents small nodules of the neoplasm, which are being developed coin-

cidently with the ulceration. The buff or ash-grey pigmented sloughs, partially separated, give a characteristic appearance to the old-standing ulcers of dysentery and tubercle. The tubercular and typhoid ulcers of Peyer's patches present a certain difference in the direction in which they extend: whilst at first both are limited to the patch, the former tend to spread in an annular manner, whilst the latter have usually their long axis corresponding to the length of the bowel. This difference depends rather on the duration of the ulcer than on any specific distinction due to the two diseases; for the more acute enteric lesion rarely spreads much beyond the area of the patch, which is in the long axis of the bowel, whilst the chronic tubercular ulcer follows the distribution of the lymphoid tissue outside the patches, and particularly along the course of the blood-vessels and lymphatics.

**COURSE.**—The course of an intestinal ulcer may be acute or chronic, lasting a few days or for years. Some of the simple ulcers of an acute intestinal catarrh belong to the former group; whilst the ulceration that accompanies chronic enteritis may be of indefinite duration.

The acute forms may either heal or go on to perforation; in the former case their existence can only be inferred, and catarrhal, follicular, and enteric ulcers belong to this category. The peptic and typhoid ulcers are those most liable to perforate the bowel. Occasionally the perforation may lead to a communication between one coil of intestine and another, without any rupture into the peritoneal cavity, which is the commonest end of a perforating typhoid ulcer. In chronic ulcers, where no adhesion or communication takes place, the base is thickened by a new-formed connective tissue, which is developed as fast as or even faster than the destructive process proceeds, and hence the intestinal wall adjacent to and involved in such ulcers is usually much thickened and indurated. Short of actual perforation or adhesion to other parts, the site of the ulceration most frequently is marked on the external surface of the bowel by a sub-acute peritonitis, which may produce a partial matting together of the intestines.

In the course of the healing of the larger ulcers, by the formation of a contracting cicatricial tissue, the gut may be considerably constricted, and a formidable obstruction established. But this does not necessarily follow even large ulcers, such as those of typhoid fever; and the depth of the destruction would seem to influence this result. When the superficial portion of the mucous membrane only is destroyed very little contraction follows, but when the deeper parts of the wall are involved the subsequently developed cicatrix tends to shrink considerably.

**SYMPTOMS.**—The greatest diversity is met

with in the symptoms of intestinal ulceration, and few, if any, can be regarded as characteristic. Inasmuch as the lesion may occur without producing any symptoms; or those that do exist may be determined by the course of, or by the conditions associated with, the ulceration; or, lastly, the results of this condition, such as perforation, may entirely obscure the actual ulceration itself: it frequently happens therefore that the existence of an ulcer is not recognised. Nor may the severity of the symptoms be taken as a measure of the extent of the ulceration, for the most marked pain, tenderness, diarrhœa, and other symptoms may be produced by an area of typhoid ulceration that heals; whilst a perforating duodenal ulcer may give scarcely any indication of its existence, until within a few hours of a fatal ending. This course appears to be very characteristic of duodenal ulcers. The writer has recorded a case of a young man who was suddenly attacked with all the symptoms of apparently intestinal colic, after constipation of a week's duration, no vomiting and no tenderness, the pain being relieved by pressure on the abdomen. Collapse set in, and death resulted in less than twenty-four hours from the commencement of the attack. The necropsy showed a perforating duodenal ulcer. Such a case is not singular, and may be preceded, as this case was, by nothing beyond an occasional feeling of discomfort at the epigastrium, not serious enough to call for advice or treatment.

Such symptoms as diarrhœa, vomiting, pain, tenderness, and pyrexia are as much dependent on a co-existent enteritis or new-growth, as they are upon the ulcer. Doubtless the exposed surface of an ulcer offers the opportunity for increased peristalsis being induced, but this is not of necessity, for constipation may be present. Where the area of ulceration is extensive, the absorbing surface is by so much diminished, and thus while the general nutrition suffers, the unabsorbed products of digestion are liable to decompose and induce diarrhœa. Vomiting may arise from a duodenal ulcer, but not always, and when present it may be due to peritonitis; icterus may also complicate an ulcer in this situation, by involving the opening of the bile-duct, or by extension of the duodenal catarrh. Pain may be quite absent or quite insignificant, unless the rectum be the seat of the disease, when the pain and tenesmus are excruciating.

The passage of blood in the stools, especially if bright, is probably a most characteristic indication of ulceration, but it does not always occur, and it may be due to hæmorrhoids or general venous congestion from portal obstruction. Pus is of occasional occurrence in the stools, especially when the ulceration has followed on phlegmonous enteritis or extensive follicular inflammation. Mucus is more commonly present, as discrete masses

of the size of a hemp-seed or in larger quantities. In the simple ulcerative colitis already mentioned there is a singular absence of blood, pus, and mucus, thus being in marked contrast to true dysentery. See *SROOLS*; and *MELÆNA*.

The symptoms due to perforation have been already described. See *Perforation and Rupture*, p. 1032.

**DIAGNOSIS.**—From what has been said, the formation of a diagnosis of intestinal ulceration is frequently impossible, and an ulcer is assumed rather than proved to exist. In enteric fever, ulceration is taken for granted as existing, though no special symptoms may indicate its presence. But if a severe and persistent diarrhœa, with liquid stools and shreds of mucus, and much pain and tenderness over the abdomen, supervene in a case of tubercular phthisis, it is a fair inference to assume ulceration of the intestines. In dysentery, like enteric fever, the ulceration is a specific part of the disease, and the diarrhœa, pain, and characteristic stools are in this case directly dependent upon the ulceration. Since there are no constant distinctive symptoms of ulceration, the ground for a diagnosis must remain uncertain. In distinguishing between gastric and duodenal ulcer the deferred occurrence of pain after taking food, even to two or three hours, is characteristic of the latter lesion, as well as the site of the pain being rather in the right hypochondrium, and less marked at the xiphoid cartilage and in the dorsal region, which are such frequent situations of gastric ulcer; the digestion is also much less disturbed in the duodenal affection, and hæmatemesis is much less common, being more constantly replaced by melæna. The greater frequency among males has been mentioned.

**PROGNOSIS.**—This largely depends upon the cause. Except through perforation or fatal hæmorrhage by erosion of vessels, death does not take place from the ulceration itself. But a tubercular ulceration is not to be expected to heal, and it may by its development hasten the end of a phthisical patient. The prognosis of typhoid ulceration will almost entirely be founded on the general state of the patient, since the indications of the ulceration itself may be so slight. The ulceration of malignant new-growths may be of actual benefit, by removing portions of the mass, and so diminishing obstruction. In all cases the liability to stenosis must be remembered; and the impaired health of body and mind in chronic ulceration may continue throughout life.

**TREATMENT.**—Since ulcers of the intestine are inaccessible to direct treatment, little can be done for them apart from the general conditions which they may complicate, or the treatment of the symptoms to which they may give rise.

Recognising that an ulcer, when it exists,

may lead to perforation, the object will be to avoid all undue movements of the intestines, and hence aperients are forbidden, and opiates are indicated. The astringents that are likely to be used for the diarrhoea or hæmorrhage may exert a local action on the lesion, and for that purpose bismuth, sulphate of copper, and similar agents are recommended. But rest is probably the only element of treatment that can affect the ulcerative process directly; whilst any improvement of the general condition will necessarily favour the healing—objects which can be best accomplished by the use of diet of the most bland description, or of nutrient enemata.

In the preceding article the diseases to which the intestines are liable have been treated of as affecting the canal as a whole; but it will be observed that when the structure and functions of particular regions of the bowel modify the character of the disease, special reference is made thereto; and, further, that when the affections of any portion require detailed description—as of the cæcum or rectum—the reader is referred to articles under those headings.

WILLIAM HENRY ALLCHIN.

**INTRA-THORACIC TUMOURS.**

Under this general term are included all growths and diseases within the chest, which give rise to tumours or swellings, offering more or less mechanical interference with the functions of the thoracic organs, and for the most part manifesting themselves by external swellings. Aneurysms would thus be comprised in this general definition. The general features and pathology of aneurysmal tumours are, however, so distinct as to require separate consideration (*see* THORACIC ANEURYSM). Other intra-thoracic tumours will be found described under the headings, LUNG, Malignant Disease of; and MEDIASTINUM, Diseases of.

J. RISDON BENNETT.

**INTUSSUSCEPTION** (*intus*, within; and *suscipio*, I receive).—A form of intestinal obstruction, in which one portion of the bowel passes into another portion. *See* **INTESTINAL OBSTRUCTION.**

**INUNCTION** (*in*, on; and *unguo*, I anoint).—**SYNON.**: Anointing.—This is a method of applying certain substances to the cutaneous surface, the object being to promote their absorption, either for the purpose of producing local effects, or of influencing the system generally. Inunction implies more or less energetic friction, the substance employed being rubbed with the hand or fingers into some part of the skin. When used for local purposes, the portion of surface to be anointed must be chosen accordingly; but if it is intended to affect the system, a region should be selected where the cutaneous tissues are thin, such as the inside of the thighs, or the axillæ, so that absorption may take place more rapidly and easily. The

rubbing must be carried on gently, and for a variable time according to circumstances; the process may be aided by heat, inunction being performed before the fire, or the part may be previously fomented.

**APPLICATION AND USES.**—The pharmaceutical preparations which are employed for inunction include glycerines, liniments, oils, ointments, oleates, and compounds made with paraffins or lanoline. If these are used for local purposes, they may be employed simply on account of the oleaginous ingredients, or to allow friction to be more easily carried on; but active ingredients are often combined in the preparations mentioned, varying according to the object sought to be obtained, such as to produce a stimulant or an anodyne effect. Inunction for procuring absorption in order to affect the system, is almost entirely confined to the use of mercury, and on this subject the following observations were written for this work by the late Mr. Gascoyen:—

‘Inunction is an old but very effectual plan of introducing mercury into the system by the skin. Although objected to as a dirty method, and therefore less practised than fumigation, it is often much more convenient, and can be used in many cases where the mercurial vapour is impracticable.

‘From half a drachm to one drachm of strong mercurial ointment, mixed with an equal quantity of lard, should be rubbed into the skin on the inside of the thighs, legs, and arms, before a fire at bedtime, using the different limbs on successive nights. The friction should be gentle and continued for a quarter of an hour to half an hour, when most of the ointment will have disappeared; the surface must not be washed, and the patient should wear the same flannel underclothing night and day. The ointment may be used every night until the gums give evidence of its action, when the quantity and frequency of application must be diminished. Sometimes it will produce an irritation of the skin, especially in fair or hairy persons. If this occur, the surface must be washed clean, and the rubbing discontinued.

‘Inunction is a most convenient way of treating syphilis in young children. The ointment should be spread upon a flannel roller, and the body of the child swathed therein; occasionally the skin should be washed, and fresh ointment applied.

‘Although but little practised now in this country, inunction is still largely employed on the Continent, and particularly in conjunction with the natural sulphur waters, for cases of old-standing syphilis.’

FREDERICK T. ROBERTS.

**INVAGINATION** (*in*, in; and *vagina*, a sheath).—A synonym for intussusception. *See* **INTUSSUSCEPTION**; and **INTESTINAL OBSTRUCTION.**

**INVASION, Modes of.**—This expression signifies the manner in which a disease sets in or commences, and the mode of invasion or onset of an illness is frequently an important factor in forming a diagnosis as to the nature of the complaint. The following are the variations noticed in this respect:

1. The invasion may be absolutely or almost *sudden*, although slight symptoms may have been previously observed, indicating the presence of some morbid condition, but not sufficiently marked to attract attention. Or there may be distinct indications of some disease, but a secondary lesion suddenly occurs in its course. This mode of onset is exemplified by the immediate effects of injuries, apoplexy, syncope, rupture of the heart, cholera, many cases of hæmorrhage, and most forms of colic. Certain diseases of a paroxysmal type are also characterised by the occurrence of attacks, which come on more or less suddenly, such as asthma, ague, and epilepsy. Some cases of fevers, especially typhus and relapsing fever, as well as of inflammatory diseases, begin in a sudden manner. 2. Frequently the onset is *acute*, the symptoms coming on rapidly, and becoming speedily severe, though often preceded for a variable time by premonitory symptoms. This is illustrated by most cases of the various fevers, and the different forms of acute inflammation. 3. A *subacute* mode of invasion is not uncommonly noticed, this being less rapid, and the symptoms less marked than where it is acute. 4. Most affections are *chronic* in their onset, setting in gradually, and often imperceptibly, so that it may be a long time before the patient is aware that there is any deviation from health.

FREDERICK T. ROBERTS.

**IODISM.**—**DEFINITION.**—Iodism is the term within which we include a variety of painful and inconvenient effects, following, under certain circumstances, the administration of iodine and its salts, but more especially the iodide of potassium.

**DESCRIPTION.**—Iodide of potassium being in part, at least, decomposed in the blood, and some of the tissues to which it is carried, we shall endeavour, in considering the symptoms of iodism, to determine which are due to the iodine, and for which the potassium must be held responsible. The physiological action of iodine is mainly directed to the nutritive and glandular functions, to the skin and mucous membranes; whilst the salts of potassium are not only diuretic and purgative, but, experimentally at least, powerfully depress the heart and spinal cord.

Rilliet, the most exhaustive writer on the subject, makes three forms of iodic intoxication: the first consisting of gastric irritation; the second, where nervous troubles come into more special prominence; whilst

cachexia and rapid emaciation characterise the third. But the most orderly and convenient plan to pursue will be to take into consideration, in regular order, the effects produced by iodine salts on the various organs and functions of the body, under various conditions of idiosyncrasy or retarded elimination.

1. *On the nervous system.*—Mental depression and diminution of muscular energy are not infrequently noted in patients taking iodide of potassium; whilst neuralgia, tinnitus aurium, and convulsive movements have also been described. It is possible that the potassium is here the active agent.

2. *On mucous membranes.*—Much mucous irritation is occasionally observed; conjunctivitis, lacrymation, sneezing and running from the nose, frontal headache, and puffy swelling of the eyelids, closely simulating coryza, being the most common symptoms of iodism, and sometimes following a single small or moderate dose. Pharyngeal congestion, and irritable redness of gums and tongue, have also been described. These symptoms are doubtless due to the iodine.

3. *On the skin.*—The eruptions produced by iodide of potassium have always attracted much attention, and they appear under several forms. Erythema has been observed; and most practitioners of experience must have seen the small round petechial spots, situated between the knees and ankles. These do not as a rule cause any inconvenience, and are usually accidentally discovered; but Dr. Stephen Mackenzie records the case of an infant of five months old, suffering from hereditary syphilis, who died of purpura after taking two and a half grains of iodide of potassium in a single dose. See DRUG ERUPTIONS.

A pustular eruption resembling acne, and sometimes so profuse as to simulate small-pox, is not very uncommon, and now and then more serious developments of skin disease may take place. Large tubercular masses may appear on the face, or a copious crop of bullæ, which occasionally burst and become converted into fungoid growths, varying in size from a pea to a shilling. All these evidences of cutaneous irritation are more likely to be produced by small than by large doses, and it is said that the frequent use of soap and the simultaneous administration of arsenic may prevent them.

4. *On the nutritive and glandular systems.*—Patients taking iodide of potassium sometimes complain of nausea, anorexia, and a bitter taste in the mouth; but where cachectic symptoms supervene, indicated by rapid emaciation, nervous palpitation, insomnia, and hypochondriasis, a ravenous desire for food has been observed. Vomiting, diarrhoea, and diuresis have also been described. Salivation is not an uncommon symptom. A time-honoured accusation

against iodine is its supposed tendency to cause atrophy of the mammæ and testicles; of this, fortunately, there is no real proof, the disappearance of the testicle, which occasionally accompanies the absorption of inflammatory products in its substance, being sometimes unjustly attributed to the treatment pursued.

**PATHOLOGY.**—The only reason for the occurrence of many cases of iodism seems to be an individual peculiarity on the part of the patient, or, in other words, that idiosyncrasy which so frequently interferes with our efforts for the treatment of disease. But now and then a more plausible explanation may be given, when we find cardiac or renal disease coinciding with the pustular rash of iodide of potassium. Retarded capillary circulation would naturally detain the drug within the blood; whilst the blocking of its usual means of exit from the system might be supposed to throw the onus of elimination on the glandular structures of the skin. Hence has been derived the plausible theory that iodine-acne is produced by direct local stimulation of the sebaceous structures; but, however true this may be in the slighter cases, Dr. Thin's careful examination of the skin of a patient suffering from a bullous rash has shown the true pathological condition to be one of rupture of blood-vessels at certain localised points, with blocking by coagula, and escape of some of the constituents of the blood into the surrounding tissues. The sebaceous elements and sweat-glands were quite unaffected, and he believes the iodic papule, the so-called acne, the bulla, and the purpuric spot, represent different stages of vascular injury. Sir Dyce Duckworth and Dr. Vincent Harris were unable in their observations to detect any rupture of vessels, but confirm Dr. Thin's report in all other essential respects.

**TREATMENT.**—Coryza, or any skin-eruption, suddenly occurring in a patient taking iodide of potassium, ought to be looked upon with suspicion, and treated by the immediate suspension of the drug. The slighter varieties of iodism are by no means uncommon, and may appear after a single small dose; but although ammonia has been confidently vaunted as a specific against such irregular manifestations of physiological activity, experience has been unable to confirm this, and we should place more faith in encouraging prompt elimination by very free dilution of the remedy, which is best taken on an empty stomach about half an hour before meals.

Fortunately for the reputation of one of our most useful drugs, the graver symptoms of iodism are decidedly rare; and we may all the more congratulate ourselves on this, when we remember how powerless we are to recognise the idiosyncrasy on which they depend. Trousseau, however, pointed out that

iodine is always badly borne in exophthalmic goitre; and Dr. Stephen Mackenzie is inclined to credit syphilis with some share in producing the purpura which proved fatal to his patient above mentioned.

ROBERT FARQUHARSON.

**IRELAND, South of.**—See QUEENSTOWN. Glengariff is also deserving of notice.

**IRITIS.**—Inflammation of the iris. See EYE, AND ITS APPENDAGES, Diseases of.

**IRREGULAR.**—This term is applied to cases of disease which do not run their regular or typical course, such as gout (see GOUT); or to functions when they are disturbed with respect to time, rhythm, or activity—as the pulse, the bowels, or menstruation.

**IRRIGATION** (*irrigo*, I water).—A method of applying cold water as a therapeutical agent, which consists in causing it to fall drop by drop on one spot. See COLD, Therapeutics of.

**IRRITABILITY** (*irrito*, I provoke).—In physiology this word signifies the power of responding to a stimulus, as exemplified by the contractility of muscular tissue. In medicine irritability implies an undue excitability of an organ or tissue, from disease or disorder, such as of the brain, spinal cord, stomach, eye, or bladder.

**IRRITATIVE FEVER.**—The nervous disturbance consequent upon fretting of the system by various sources of irritation, gives rise to a pyrexia which is often called 'Irritative Fever.' The febrile excitement so familiar to the surgeon as a consequence of wounds and injuries may be classed under this head. It may, however, be provoked by any kind of irritation, especially irritation applied to the mucous membrane of the alimentary canal. The rise of temperature which often accompanies irritation of the bowels by scybala or acrid secretions, and the febrile phenomena attendant upon dentition, may be quoted as familiar examples of irritative fever which must be within the experience of all.

The readiness with which pyrexia can be induced by these and similar causes must vary according to the intensity of the irritant, and the constitutional peculiarities of the individual upon whom it operates. As a rule, men are less susceptible than women, and women than children. In children, indeed, with their exalted nervous sensibility, feverishness from this cause is a common symptom. In young subjects mental emotion alone will often produce a rise of temperature, which may be a source of perplexity. In children's hospitals it is a common observation that the bodily temperature on the night of admission is high, even although the illness

affecting the child is one not in itself usually accompanied by fever.

Dentition in young children is so frequent a cause of pyrexia, that the state of the gums should never be overlooked in any case where feverishness is a prominent symptom. Neglect of this precaution may cause some obscurity in the diagnosis. Thus, if a child, while cutting a tooth, has an attack of pulmonary catarrh, the temperature will almost certainly be high. In such a case the cough, combined with fever, rapid breathing, and a quick pulse, might naturally suggest the presence of pneumonia. On examination, however, it will be found that the pulse-respiration ratio is little perverted, the cough is loose and not hacking, there is no movement of the nares in breathing, and the history of the attack is not the history of pneumonia. On searching further for a cause of the pyrexia, tension and swelling of the gums will be noticed, and the difficulty will be at once explained.

Irritation of the stomach and bowels by acrid secretions or indigestible food is another common cause of irritative fever in children. The intense nervous disturbance excited in young babies by an improper meal induces a rapid rise of temperature, and may culminate in an attack of convulsions. Fæcal accumulation, or the irritation of worms in the bowels, may also produce sufficient disturbance to give rise to fever in children and delicate persons.

In children the sensitiveness of the system to irritants varies according to the age of the child, according to the natural impressionability of its nervous system, and also according to the state of its general health. Thus, as a rule, the younger the child, the more sensitive is its nervous system; but even in young babies differences will be found in this respect, some being affected much less easily than others by reflex stimuli. In all, however, slow reduction of the strength, such as is produced by progressive chronic disease, gradually reduces nervous sensibility; and a child, enfeebled by an illness of long standing, may show a complete insensibility to all nervous impressions. In young subjects, irritative fever, like other forms of pyrexia, is usually remittent; but its remissions are not always

found at the same period of the twenty-four hours. There is not, for instance, always a fall of temperature in the morning and a rise at night. One of the peculiarities of this form of febrile disturbance is the irregularity of the fever. A high morning temperature in a young child should always suggest a reflex cause for the pyrexia.

**TREATMENT.**—The treatment of irritative fever must be directed to the relief or to the removal of the irritating sources from which the fever proceeds. The use of febrifuge remedies may be also called for, if constitutional symptoms be marked or persistent.

EUSTACE SMITH.

**ISCHÆMIA** (*ἰσχω*, I restrain; and *αἷμα*, the blood).—Deficiency of blood in a part, short of complete cessation of the circulation: partial anæmia. See CIRCULATION, Disorders of.

**ISCHIALGIA** (*ἰσχίον*, the haunch; and *ἄλγος*, pain).—A synonym for sciatica. See SCIATICA.

**ISCHL**, in the Salzkammergut, Austria.—A sheltered, bracing, mild, rather moist climate. Altitude 1,560 feet. Thermal common saline baths. See CLIMATE, Treatment of Disease by; and MINERAL WATERS.

**ISCHURIA** (*ἰσχω*, I restrain; and *οὔρον*, the urine).—This word properly signifies the arrest of the secretion of urine (see URINE, Suppression of). It is also applied to mere retention of urine.

**ISSUES.**—See COUNTER-IRRITANTS.

**ITALY.**—See CLIMATE, Treatment of Disease by; and NAPLES; PISA; ROME; and SAN REMO.

**ITCH.**—A popular name for scabies. See SCABIES.

**ITCHING.**—See PRURITUS.

**-ITIS.**—A terminal syllable used to indicate an inflammatory disease of a tissue, or organ; for example, Pleuritis, Hepatitis, or Cystitis.

## J

**JACTATION**, or **JACTITATION** (*jactatio*, a tossing about of the body, or marked restlessness).—This is a condition mostly associated with certain severe febrile diseases, but also with some nervous affections, with severe pericarditis, or as a sequence of

copious uterine or other hæmorrhages. A restlessness amounting to jactation may likewise be met with in some patients, when suffering from severe or long-continued pain. It must not be confounded with certain forms of chorea, in which a somewhat similar toss-

ing about of the body may be encountered. The absence of pain and of marked febrile disturbance, together with the history of the patient, will, even in the cases where the general resemblance is closest, speedily enable the latter condition to be recognised.

**JAUNDICE.**—**SYNON.**: *Icterus*; *Morbus Regius*; *Morbus Arquatius* (Celsus); Fr. *Ictère*; *Jaunisse*; Ger. *Gelbsucht*.

**DEFINITION.**—Jaundice may be defined as a yellowness of the integuments and conjunctivæ, and of the tissues and the secretions generally, from impregnation with bile-pigment.

**ÆTIOLOGY AND PATHOLOGY.**—All cases of jaundice may be referred to one of two classes:—

I. Cases in which there is a mechanical impediment to the flow of bile into the duodenum, and where the bile is in consequence retained in the biliary passages, and thence absorbed into the blood.

II. Cases in which there is no impediment to the flow of bile from the liver into the bowel.

These two forms of jaundice have long been recognised; but there is much difference of opinion as to the mode of production of the jaundice in the second class of cases, although these are, perhaps, the most common in practice.

When there is any obstruction to the flow of bile through the hepatic or common duct, the way in which jaundice arises is sufficiently clear. The bile-ducts and the gall-bladder become distended with bile, which is absorbed into the blood by the lymphatics and the veins. If the hepatic duct of a dog be ligatured, and the animal killed after two hours, the lymphatics in the walls of the bile-ducts are seen to be distended with yellow fluid; the fluid in the thoracic duct is also yellow, and so likewise are the intervening lymphatic glands. In patients also who die of obstruction of the bile-duct, the lymphatics of the liver are often found to contain bile. On the other hand, two hours after ligature of the common duct, the serum of blood taken from the hepatic vein contains much more bile-pigment than that of blood taken from the jugular vein, which shows that in cases of obstruction of the bile-duct, bile is also directly absorbed by the veins.

But in a large proportion of cases there is no mechanical impediment to the escape of bile from the liver, and then an explanation of the jaundice is less obvious. Boerhaave and Morgagni long ago suggested that the jaundice in these cases was the result of a *suspended secretion*. They taught that the function of the liver was merely to separate the elements of bile which were already formed in the blood; and that when anything interfered with the function of the liver, the elements of bile accumulated in the blood,

and the result was jaundice of the skin and other tissues. Although this view has been strenuously opposed by several excellent authorities, it was in this country until recently generally accepted. It was advocated, for example, by Dr. George Budd, in his valuable treatise on diseases of the liver, although it is but right to add that Dr. Budd made a special exception with regard to the biliary acids. 'The most skilful chemists,' he says, 'who have recently analysed the portal blood, have failed to detect the biliary acids in it, and have come to the conclusion that these at least are formed in the liver.' This opinion, that the liver manufactures the bile-acids, while it merely excretes the bile-pigment, was adopted by Dr. G. Harley, in his essay on jaundice.

There are, however, weighty objections to this view, some of which may be mentioned.

1. Although bile-pigment appears to be derived from the colouring matter of the blood, it has not yet been satisfactorily shown that bile-pigment, as such, exists ready formed in the blood of persons who have not jaundice. Frerichs denies that it ever has existed. Lehmann, who has investigated with great care the changes which the blood undergoes in passing through the liver, has never been able to detect the colouring matter of bile in portal blood, and infers that this, as well as the bile-acids, must be formed in the liver itself. The blood of the hepatic artery has been examined with a like result. It is obvious that if bile-pigment exists in healthy blood at all, its quantity must be very minute; and when we consider that the quantity of bile secreted by the human liver daily is about two pints, and yet that jaundice is not a normal condition, it seems impossible that all the bile-pigment secreted by the liver can be formed in the blood, and it is not probable that part is formed in the blood and part in the liver. The discovery by a few observers of a small quantity of bile-pigment in what appeared normal blood does not prove that it was formed in the blood. It is quite conceivable that it may have been formed in the liver, and have become subsequently absorbed.

2. The secreting tissue of the liver is often for the most part or entirely destroyed, so that bile is no longer secreted, and yet no jaundice results. If bile-pigment be formed in the circulating blood, it is difficult to explain what becomes of it in these cases.

3. If the constituents of bile are formed in the blood, intense jaundice ought at once to follow the extirpation of the liver in any of the lower animals, in like manner as urea accumulates in the blood after removal of the kidneys. But Müller, Kunde, Lehmann, and Moleschott have repeatedly extirpated the liver of frogs, and have invariably failed to find a trace of the biliary acids, or of bile-

pigment, in the blood, the urine, or the muscular tissue. Whether the same would hold good as regards man is questionable.

These and other considerations make it very doubtful if any case of jaundice can with propriety be attributed to a suppression of the hepatic functions; and it is therefore necessary to seek for some other explanation of those cases of jaundice in which there is no obstruction in the bile-duct.

A solution of the difficulty has been proposed by Professor Frerichs, of Berlin. According to this distinguished observer, a large proportion of the colourless bile-acids formed in the liver is either directly taken up by the blood in the hepatic vein, or is absorbed from the bowel. Under ordinary circumstances, these bile-acids become oxidised and assist in forming the large quantity of taurin found in healthy lung, and the pigments voided in the urine; but if these normal metamorphoses are interrupted by nervous agencies, or by poisons in the blood, the bile-acids, not being sufficiently oxidised, are converted into bile-pigment in the blood, and the result is jaundice. This view seemed to be supported by two experiments intended to show—first, that bile-pigment can be produced artificially from the bile-acids, by the action of concentrated sulphuric acid; and, secondly, that colourless biliary acids, when injected into the veins of dogs, are converted in the blood of these animals into bile-pigment. There is, however, far from being unanimity among different observers as to the results of these experiments; and it has more recently been proved that bile-acids are not, either in or outside the body, converted into bile-pigment. The bile-acids probably act by liberating the hæmoglobin from the red corpuscles, and the dissolved hæmoglobin is converted by the liver-cells into bilirubin. A decision of the points at issue does not appear to be of material importance for explaining those cases of jaundice in which there is no obstruction of the bile-duct, inasmuch as there are good grounds for believing that not only in jaundice, but in health, a portion of the bile-pigment, as well as of the bile-acids, formed in the liver, is absorbed into the blood.

Although the amount of bile secreted daily must vary in different persons, and in the same person under different circumstances, being modified by the quantity and quality of the food, the activity of respiration, and other conditions, there can be little doubt that but a small portion of that which is ordinarily secreted is discharged from the bowel. Observations on the lower animals and on man himself have shown that the quantity of bile secreted by the liver of a healthy adult averages forty ounces. It is generally admitted that the feces contain but a fraction of the bile-acids (altered) corresponding to this quantity of bile, and it

seems equally clear that much of the bile-pigment must also disappear in the bowel. There are grounds for believing that the bile-pigment which so disappears goes to form urinary pigment; while the fact familiar to all clinical observers, that the bile-pigment discharged from the bowel is greatly increased by calomel and other purgatives, without any corresponding increase of secretion of bile by the liver, seems to show that under ordinary circumstances much of the bile-pigment secreted by the liver is not discharged with the feces. It may be added that in carnivorous animals and in snakes, although bile-pigment is secreted in abundance by the liver, the quantity discharged with the feces is even relatively less than in man.

The question as to what becomes of the bile which is not discharged from the bowel has an important bearing on the pathology of the cases of jaundice now under consideration. A large proportion of it is again absorbed, either by the biliary passages, or by the mucous membrane of the bowel. From what is now known of the diffusibility of fluids through animal membranes, it is impossible to conceive bile long in contact with the lining membrane of the gall-bladder, bile-ducts, and intestine, without a large portion of it passing into the circulating blood. The constant secretion and reabsorption of bile is, in fact, merely part of that osmotic circulation constantly taking place between the fluid contents of the bowel and the blood, the existence of which is too little heeded in our pathological speculations and in therapeutics. The quantity of fluid which is being thus constantly poured out from the gastric and intestinal glands, the liver, pancreas, &c., and then reabsorbed, is enormous; in twenty-four hours it probably far exceeds the whole amount of blood and fluid in the body. The effect of this continual outpouring is supposed to be to aid metamorphosis; the same substance, more or less changed, seems to be thrown out and reabsorbed, until it is adapted for the repair of tissue or becomes effete. How many times this cycle of movement is repeated, before the bile is extruded from the system, we have no means of knowing; but in the course of this osmotic circulation, much of the bile appears to become transformed into products which are eliminated by the lungs and kidneys, while at the same time this circulation assists in the assimilation of the nutritive materials derived from the food.

Here, then, we have an explanation of those cases of jaundice where there is no impediment to the flow of bile from the liver. Under normal conditions, the whole of the bile that is secreted is at once transformed, so that neither bile-acids nor bile-pigment can be discovered in the blood or in the urine, and there is no jaundice. But in

certain morbid states the absorbed bile does not undergo the normal metamorphoses; it circulates in the blood and stains the skin and other tissues, and in this way we have jaundice without any obstruction of the bile-duct. The morbid states which, so far as we know, conduce mainly to this result are, for the most part, precisely those in which we might expect abnormal blood-metamorphoses, namely:—

1. Certain poisons, such as those of yellow fever, relapsing fever, pyæmia, and more rarely those of remittent fever, typhus, and scarlatina; also snake-poison, chloroform, &c.

2. Nervous influences, such as a sudden fright, violent rage, great or protracted anxiety, and concussion of the brain.

3. A deficient supply of oxygen, as happens in certain cases of pneumonia, or in persons living in confined and crowded dwellings.

4. An excessive secretion of bile, especially when conjoined with constipation. In this case, unless the bile be removed by purging, the quantity absorbed may be too great to undergo the normal metamorphoses, and the presence in the blood of the untransformed bile causes jaundice.

According to this view, the only pathological difference between jaundice from obstruction and jaundice independent of obstruction of the common bile-duct is that, in the former case, little or none of the bile secreted by the liver can escape from the body with the fæces, and consequently all that is secreted is absorbed into the blood, and the quantity thus absorbed is far too great to undergo the normal metamorphoses; while, in the latter case, bile passes into, and is discharged from the bowel, as usual, but that which is absorbed, which in quantity may not exceed what is absorbed in health, remains unchanged in the blood. As might be expected, the jaundice in the former case is usually much more intense than in the latter, although, when an obstruction of the bile-duct has lasted long, the jaundice often becomes paler, not from any diminution of the obstruction, but from the secreting tissue of the liver becoming destroyed, and comparatively little bile being secreted; while in cases where there is no obstruction of the bile-duct, the intensity of the jaundice will vary according to the amount of bile which is absorbed, and the degree of derangement of the blood-metamorphosis.

Jaundice is by some writers divided into *hepatoginuous* and *hæmatoginuous* jaundice. In obstruction of the ducts the jaundice is clearly hepatoginuous. Regarding hæmatoginuous jaundice there is considerable dispute. In various diseases (*e.g.* acute atrophy of liver, yellow fever, remittent fever, and occasionally typhus and relapsing fever, &c.), and as the result of certain poisons (*e.g.* snake-bite, phosphorus, arsenic, and antimony), in which there is no apparent obstruction of the

ducts, jaundice occurs, and at the same time there is evidence of blood-destruction. How the latter brings about the jaundice has been very carefully worked out experimentally by Stadelmann in regard to the action of toluylendiamine in dogs, cats, and rabbits. Briefly put, the effect of this poison is to act powerfully on the red corpuscles, so that they part too readily with their hæmoglobin. The latter is acted upon by the liver, and converted into bile. The pigment and acids are increased; and if the action is continued, the bile becomes so inspissated that it blocks the small ducts, increases the pressure in the biliary canaliculi, and leads to absorption. Thus, what at first sight appears to be, and is in a certain sense, a hæmatoginuous jaundice is in fact a hepatoginuous jaundice, due to absorption. What is proved with regard to toluylendiamine is probably true with regard to mineral, vegetable, and animal poisons. After transfusion of blood from one animal to another of different species, a dissolution of the foreign blood-corpuscles takes place, and hæmoglobinæmia results. Much of the blood-pigment is eliminated by the kidneys, causing hæmoglobinuria; but some reaches the liver, causing an increase of bile-pigment, which is absorbed and gives rise to a slight degree of jaundice. The same has been observed in some malarial affections, in which both hæmoglobin and bile are present in the urine—malarial hæmoglobinuria. On the other hand, in old extravasations of blood, after a time, disintegration of the blood-corpuscles takes place, and hæmatoidin, which is regarded by many modern chemists as identical with bilirubin, results. The hæmatoidin is absorbed and stains the tissues, and appears in the urine as bile-pigment. What is proved as regards extravasations may occur in smaller blood-disintegrations, and thus the possibility of a true hæmatoginuous jaundice cannot be denied, though it has not been actually proved except in the case of old extravasations. Speaking generally, *hepatoginuous* jaundice corresponds with obstructive, and *hæmatoginuous* jaundice with non-obstructive jaundice. In acute yellow atrophy there is no obstruction. The jaundice is ascribed by some to arrest of the bile-producing functions of the liver-cells, by others to blood-destruction. If the arrest of bile-production in the liver was complete, it is clear that the bile, which in this disease stains the tissues and appears in the urine, must be produced elsewhere, and be hæmatoginuous, or at least extra-hepatic. It is possible that the absorption of bile-acids in obstructive jaundice exercises a destructive influence on the red corpuscles, and thus becomes an additional source of bile-pigment. Niemeyer particularly insisted that 'every hepatoginuous icterus tends to become an hæmatoginuous icterus.'

With these preliminary remarks on the

pathology of jaundice, we may now proceed to enumerate its different causes, which may be classified according to the following tabular form:—

**A. Jaundice from Mechanical Obstruction of the Bile-duct.**

**I. Obstruction by foreign bodies within the duct.**

1. Gall-stones and inspissated bile.
2. Hydatids and distomata.
3. Foreign bodies from the intestines.

**II. Obstruction by inflammatory tumefaction of the duodenum, or of the lining membrane of the duct, with exudation into its interior.**

**III. Obstruction by stricture or obliteration of the duct.**

1. Congenital deficiency of the duct.
2. Stricture from perihepatitis.
3. Closure of the orifice of the duct in consequence of an ulcer in the duodenum.
4. Stricture from cicatrisation of ulcers in the bile-ducts.
5. Spasmodic stricture.

**IV. Obstruction by tumours closing the orifice of the duct, or growing in its interior.**

**V. Obstruction by pressure on the duct from without, by—**

1. Tumours projecting from the liver itself.
2. Enlarged glands in the fissure of the liver.
3. Tumour of the stomach.
4. Tumour of the duodenum or pancreas.
5. Tumour of the kidney.
6. Post-peritoneal, or omental tumour.
7. An abdominal aneurysm.
8. Accumulation of fæces in the bowels.
9. A pregnant uterus.
10. Ovarian and uterine tumours.

**B. Jaundice independent of Mechanical Obstruction of the Bile-duct.**

**I. Poisons in the blood interfering with the normal metamorphosis of bile.**

1. The poisons of the various specific fevers.
  - (a) Yellow fever; (b) Remittent and intermittent fevers; (c) Relapsing fever; (d) Typhus; (e) Enteric fever; (f) Scarletina; (g) Epidemic jaundice.
2. Animal Poisons: (a) Pyæmia; (b) Snake-poison.
3. Mineral poisons: (a) Phosphorus; (b) Mercury; (c) Copper; (d) Antimony; (e) Arsenic.
4. Chloroform and ether.
5. Acute atrophy of the liver.?

**II. Impaired or deranged innervation interfering with the normal metamorphosis of bile.**

1. Severe mental emotions, fright, anxiety, and allied causes.
2. Concussion of the brain.

**III. Deficient oxygenation of the blood, interfering with the normal metamorphosis of bile.**

**IV. Excessive secretion of bile, more of**

*which is absorbed than can undergo the normal metamorphosis.*

1. Congestion of the liver: (a) Mechanical; (b) Active; (c) Passive.

**V. Undue absorption of bile into the blood, from habitual or protracted constipation.**

**SYMPTOMS.**—From what has been stated in the preceding section, it is obvious that jaundice is not a disease, but is a symptom of many different diseases. This view of the matter cannot be too strongly impressed upon the student and practitioner, whose efforts in every case must be directed to discover the fundamental malady. There are, however, certain phenomena connected with jaundice, independent of its cause, which deserve to be mentioned.

1. *Intensity of the jaundice.*—Next to the liver itself, the skin is the tissue of the body which becomes most deeply jaundiced; but before it becomes affected, a yellow tint is usually observed in the conjunctivæ. There must be a certain concentration of bile-pigment to produce a yellow colour of the skin; in the slighter and more temporary cases, the conjunctivæ only may be affected. Although after ligation of the common bile-duct in the lower animals it has been sometimes found that even the conjunctivæ do not become jaundiced for two or three days, in the human subject jaundice of both skin and conjunctivæ is usually observed within twenty-four hours of closure of the duct.

The colour of the skin varies from a pale sulphur or lemon-yellow, through a citron yellow, to a deep olive or bronzed hue. The tint varies according to the cause and its duration. When the cause is obstruction of the bile-duct, it is light at first, and increases in depth the longer the disease lasts; although in advanced cases, as already stated, the colour sometimes becomes pale, not from the obstruction yielding, but from the tissue of the liver becoming destroyed, and very little bile being secreted. In jaundice from obstruction also the depth of tint often varies from day to day, not from any variation in the degree of obstruction, but according to the amount of bile secreted by the liver, and the eliminative activity of the kidneys. It is well to remember that what is called 'black jaundice' may result from any cause of obstruction—from gall-stone as well as from cancer. In these cases the greenish or almost black hue is due to the absorbed bile-pigment being vitiated and dark, or to the visage being also darkened from imperfect arterialisation of the blood, the dark colour resulting from a mingling of the lividity with the colour of bile. When the jaundice is independent of obstruction to the flow of bile, the colour is rarely very deep, and yet these are often the most serious cases. The colour also varies with the age, the natural complexion, and the amount of fat in the individual. It is deeper

in the old, the wrinkled, and the dark-complexioned, than in young persons of fair complexion, and with plenty of fat. Lastly, it is important to remember that the colour often remains in the skin for some time after the cause of the jaundice has been removed, and that then its departure may be expedited by diaphoretics and warm baths.

2. The *secretions* are tinged with bile-pigment, but some much more so than others. This is notably the case with the urine, by which the greater part of the bile-pigment is eliminated from the body, and which acquires a saffron-yellow, greenish-brown, or brownish-black hue, according to the amount of pigment which it contains. The urine usually becomes yellow before the skin, or even the conjunctivæ; and when the cause of the jaundice is transient, it may happen that the whole of the pigment is eliminated by the urine, without any jaundice appearing in the skin. On the other hand, when once the skin has become yellow, it may remain so for some time after bile-pigment has quite or nearly disappeared from the urine.

Other secretions may contain bile-pigment as well as the urine. The cutaneous glands sometimes eliminate it in such quantity as to stain the linen yellow, but the amount discharged in this way is never great. Instances have been recorded where the secretion of the mammary glands has been tinged with bile-pigment, but they are not very common. Still rarer instances have been noticed where the saliva or the tears have been tinged. It is not a little remarkable that bile-pigment is not eliminated in cases of jaundice by the mucous membrane of the respiratory passages, or of the digestive tube. This is a matter of some practical importance, for, were it otherwise, the stools might contain bile-pigment even when there was complete obstruction of the gall-duct. Still, when either of these mucous membranes is inflamed, and throws off an albuminous or fibrinous exudation, the altered secretions may contain bile-pigment. Thus, when pneumonia coexists with jaundice, there is often bile-pigment in the sputa, which may be distinguished by the nitric-acid test from the greenish or yellow colour often presented by pneumonic sputa owing to changes in the blood-pigment independent of bile. Indeed, in cases of jaundice bile-pigment may be detected in inflammatory exudations, as in the serum of a blister, before it appears either in the skin or even in the urine. It is probable that those rare cases where the saliva has been noticed to be yellow admit of a similar explanation; in many of them there has been mercurial salivation, a condition in which the saliva is not normal, but contains much albumen.

3. A *bitter taste* is not infrequently complained of by persons who are the subjects

of jaundice. It may denote the presence in the blood of the biliary acids, for taurocholic acid is intensely bitter. It is at all events not due to bile-pigments, which are tasteless. Moreover, it is a common symptom in biliary derangements where there is no jaundice.

4. *Derangements of digestion*, such as flatulence, constipation, and an altered character of the motions, may be due to the absence of bile from the motions. Bile is an antiseptic, though a very feeble one, and when it is absent the intestinal contents undergo fermentation, gases accumulate in the bowels, the motions become putrid, and from the absence of bile and the presence of fat they present a pale-drab or clay colour. Bile is also the natural stimulant of the peristaltic action of the gut, and consequently, when the supply is cut off, the bowels are usually constipated; but in some cases the putrid fæces act as an irritant and excite diarrhœa. In those cases of jaundice where there is no obstruction of the common bile-duct, the motions may be but little altered.

When bile does not enter the bowel, the digestion of fat is interfered with. Jaundiced patients dislike fat, and do not assimilate it, and the fatty matter in the ingesta may sometimes be detected in the stools. Hence, whatever be the cause of obstruction of the bile-duct, the nutrition of the body suffers: the emaciation may be slow, but it is progressive, until all the fat disappears, and then the weight of the body may remain stationary for many months. With the emaciation there is always more or less muscular debility.

5. *Pruritus*, without any eruption, is a very obstinate and distressing symptom in many cases of jaundice. It is usually worse at night, and, by preventing sleep, may wear out the patient. It is chiefly observed in cases of jaundice due to obstruction of the bile-duct. It is not due to the presence of bile-pigment in the blood, for in some cases it precedes the jaundice, and in others it comes and goes during the persistence of the jaundice. Moreover, in many cases of jaundice it is absent throughout, while it is not uncommon in biliary derangements where there is no jaundice.

6. *Cutaneous eruptions*.—Urticaria, lichen, boils, or carbuncles are occasionally observed in connexion with jaundice; and likewise that remarkable affection of the skin known as *xanthoma*, *xanthelasma*, or *vittiligoidea*. See XANTHELASMA.

7. The *temperature* is not altered in jaundice, except when this occurs as a complication of some acute febrile disease, or when there is inflammatory action in the liver itself.

8. *Slowness of pulse*.—A common result of non-febrile jaundice is retardation of the

heart's action, and diminution of arterial tension. The pulse may fall to 50, 40, or even 20, and sometimes it is also irregular. This slowness of pulse is particularly noticeable when the patient is recumbent. When there has been antecedent pyrexia, the pulse usually falls on the supervention of jaundice. Slowness and irregularity of the pulse are chiefly observed in jaundice from obstruction of the bile-duct, and particularly in those common cases known as catarrhal jaundice; and accordingly they are not unfavourable symptoms, as might have been supposed. So far as the writers' experience goes, patients with this symptom invariably recover. It has not yet been explained why this condition of circulation should be present in some cases of jaundice, and absent in others. The natural explanation would be that it is due to some ingredient of the bile, which does not exist in the blood in all cases of jaundice. Some experiments of Röhrig have shown that the biliary-acid salts paralyse the heart, and retard its action, while bile-pigment has no such effect. It has been shown by Dr. Wickham Legg and others that the slowing of the heart is due to the action of cholalic acid on the cardiac ganglia. Slowness of the pulse, therefore, in jaundice may indicate the presence in the blood of unchanged biliary acids; but so far there are no observations to show that bile-acids are present in the urine in these more than in other cases of jaundice.

9. *Hæmorrhages*.—In many cases of jaundice the blood seems to become impoverished, by a diminution in the proportion of red corpuscles and fibrin; and hæmorrhages take place from the various mucous membranes and into the substance of the skin. This hæmorrhagic tendency is particularly observed, in conjunction with cerebral symptoms, in cases of jaundice where there is no obstruction of the bile-duct; but it also occurs in cases of mechanical jaundice of long standing, from any cause, when the secreting tissue of the liver has in a great measure disappeared.

10. *Xanthopsy or Yellow vision*.—In rare cases of jaundice, all white objects appear yellow to the patient. The administration of santonin internally has also sometimes been followed by yellow vision, which has ceased as soon as the colouring-matter has been eliminated by the kidneys. This fact, as well as the observation that in several cases of jaundice, with xanthopsy, the conjunctival vessels have been preternaturally distended with blood, has led to the belief that the symptom is due to a tinging with bile-pigment of the humours of the eye. On the other hand, the circumstances that the xanthopsy may intermit, without any change in the jaundice; that it is usually absent when there is intense jaundice of the cornea and other tissues of the eye; and the state-

ment that it may occur in typhus fever, and in certain derangements of vision, such as night-blindness, when there is no jaundice, have led most authorities to consider it, as appears to have been proved in regard to the effects of santonin, as due to an affection of the visual nerves.

11. *Cerebral symptoms, and the Typhoid state*.—Patients with jaundice are often irritable in their temper and hypochondriacal; and occasionally they are attacked with acute delirium, stupor, coma, convulsions, muscular tremors, subsultus, carphology, paralysis of the sphincters, a dry and brown tongue, and other indications of the typhoid state. These symptoms are most common in cases where there is no obstruction of the ducts, but they also occur in cases of obstruction, usually of long standing, where all or the greater part of the secreting tissue of the liver has been destroyed. Different opinions are held as to their cause. After death no lesion is found of the brain or its membranes, and they are, therefore, most probably due to some alteration of the blood. They are commonly attributed to poisoning of the blood with bile, either from suppression or reabsorption of the secretion. But the assumption that the elements of the bile are preformed in the blood has been already shown to be probably erroneous; and there is equally little evidence that absorbed bile is possessed of poisonous qualities, or that its presence in the blood, even to saturation, will give rise to cerebral symptoms. Many experiments have been performed on animals to show that bile is a deadly poison; but there is reason for believing that the bad results observed have been due to the injection into the areolar tissue of decomposing mucus contained in the bile. Bile, from which the mucus has been removed, has been repeatedly injected by Frerichs and other observers into the large veins of dogs, without cerebral symptoms or any bad results ensuing, except that death has in some instances been caused by the entrance of air into the veins. But it is scarcely necessary to turn to experimental inquiries on the lower animals for evidence on the matter, and in all these experiments there are sources of fallacy. There is ample proof that the blood of the human subject may be saturated with bile for months, or even years, without any cerebral symptoms resulting. Dr. Austin Flint, jun., is of opinion that the cerebral symptoms of jaundice are due to the retention of cholesterin in the blood, or to what he has designated *cholesteræmia*. Cholesterin is one of the constituents of bile, and Dr. Flint regards it as an excrementitious product of nervous tissue, the elimination of which from the body is one of the functions of the liver, and the retention of which in the blood he believes to act as a poison like urea. But if the non-excretion of all the elements of bile does not give rise to cerebral symp-

toms, it is difficult to understand how they can result from the retention of cholesterin alone. In cases, for instance, of permanent closure of the bile-duct, cholesterin is not discharged from the liver into the bowel, nor does it accumulate in the biliary passages, and yet, if it be retained in the blood, cerebral symptoms rarely occur.

The cerebral symptoms in jaundice are often most severe when the jaundice is slight, and they may occur in diseases of the liver when there is no jaundice. They are best explained by the knowledge which we now possess of the function performed by the liver in disintegrating albuminous matter into less complex substances, such as urea and uric acid, which are eliminated by the kidneys. When this function of the liver is arrested or seriously impaired, urea is no longer eliminated in sufficient quantity by the kidneys; lithic acid and deleterious products of disintegrating albumen even less oxidised, such as leucin and tyrosin, and perhaps others with which we are as yet imperfectly acquainted, accumulate in the blood and tissues; the result is the development of symptoms of blood-poisoning similar to those which arise when the kidneys are unable to eliminate the products of albumen-disintegration, owing to disease of their own structure, or to an excessive formation of urea and other products, as happens in many febrile diseases. In acute atrophy, for example, the structure of the liver is destroyed, and its functions are arrested; leucin and tyrosin take the place of urea in the urine, and are also found in large quantity in the liver, spleen, and kidneys; while cerebral symptoms and the typhoid state are prominent features of the disease. See CHOLEMIA.

DIAGNOSIS.—There is rarely much difficulty in the diagnosis of jaundice, but it is well to remember that certain conditions are sometimes mistaken for it. Such are chlorosis, pernicious anæmia, hæmoglobinuria, the anæmic aspect resulting from organic visceral disease (and particularly from contracted kidneys), from cancer, from exposure to malaria, from Addison's disease, or from lead-poisoning; an undue amount of sub-conjunctival fat; or an unusually dark colour of the ordinary urinary pigment, or the presence in the urine of abnormal pigments, such as those of santonin, turmeric, rhubarb, &c. In every case where there is the slightest doubt, it will be removed by resorting to the nitric-acid test for bile-pigment in the urine. If this gives no result, the case is not one of jaundice.

But it is a more difficult matter to determine the cause of the jaundice, and yet this should invariably be the aim of the medical attendant, before forming a prognosis or proceeding to treatment. The scope of this article does not permit a lengthened analysis of the characters distinguishing the different forms of jaundice according to its cause, but

the following remarks may be of some service.

1. In the first place it is always well to determine whether or not the jaundice be due to obstruction of the bile-duct. According to Dr. G. Harley, this can be done by determining the presence or absence of bile-acids in the urine. Adopting the view that bile-acids are formed by the liver, while bile-pigment is preformed in the blood, he contends that in jaundice from 'suppression' (or independent of obstruction) the liver does not secrete bile, and consequently, no bile-acids being formed, none can enter the circulation or be detected in the urine; whereas in jaundice from obstruction, bile is secreted and absorbed into the blood, and a portion of the bile-acids not transformed in the circulation appears in the urine. But in addition to the strong improbability already urged that any form of jaundice is due to a *suppressed secretion* of bile, clinical experience is entirely opposed to the practical value of the test in question for throwing light on the cause of jaundice. Bile-acids have been found in the urine in cases of acute atrophy of the liver, where there is no obstruction of the bile-duct, and in very many cases of mechanical jaundice they are certainly absent. The ordinary clinical test for bile-acids in the urine (Pettenkofer's) is a very uncertain one. For more accurate methods, see Halliburton's *Text-book of Chemical Physiology and Pathology*. A more reliable indication of obstruction of the common bile-duct is furnished by the stools. When there is no obstruction, the stools almost invariably contain bile; but when the duct is obstructed they are clay-coloured. The rule is not without exceptions, and there are several sources of fallacy. The jaundice usually persists for some time after the duct has become pervious, and thus bilious motions may co-exist with jaundice which has resulted from obstruction; or, if the motions be thin and watery, they may appear to contain bile from the admixture of jaundiced urine; or, not infrequently, when the bile-duct is quite impervious, the motions are of a brownish tinge, owing to the presence of altered blood, which may closely resemble dark bile. Iron, bismuth, and charcoal also colour the motions. A tumour corresponding to the region of the gall-bladder will favour the view that jaundice is due to obstruction of the bile-duct. Lastly, jaundice which persists and is yet slight, is most probably independent of obstruction, for jaundice from persistent obstruction speedily becomes intense.

2. It is always important to note the mode of commencement of jaundice. That which appears suddenly in a person whose previous health has been good, is most probably the result of obstruction of the duct by a foreign body, or it has a nervous origin. The former cause will be distinguished by biliary colic, vomiting, and clay-coloured stools. On the

other hand, jaundice coming on slowly, but ultimately becoming intense, with clay-coloured stools, points to pressure on the duct from without, or to a growth in its interior.

3. A history of previous attacks of jaundice of a similar nature is in favour of a catarrhal origin, or of gall-stones.

4. Pain in severe paroxysms concurring with jaundice points generally to gall-stones or cancer; more rarely to hydatids, or to an aneurysm of the hepatic artery. Cancer is distinguished from gall-stones by there being usually a history of falling health and emaciation before either the pain or the jaundice.

5. Jaundice concurring with enlargement of the liver is most probably due to cancer or cirrhosis; more rarely to pyæmic abscesses, or to waxy liver with large glands in the portal fissure.

6. Jaundice concurring with ascites points to cancer or cirrhosis. The diagnosis of the latter will usually be assisted by the physiognomy, the slightness of the jaundice, the previous habits, and a history of alcoholic dyspepsia; while in cancer there are often darting pains, and the jaundice is usually intense.

7. Jaundice concurring with pyrexia is either secondary to some acute febrile disease; or is due to suppurative pylephlebitis, a suppurating hydatid tumour opening into a bile-duct, or inflammation of the bile-ducts. Temporary pyrexia may also occur during the passage of a gall-stone.

8. Cerebral symptoms associated with jaundice suggest acute atrophy of the liver, poisoning by phosphorus, some specific fever, pneumonia, or nervous shock.

9. Jaundice in a young person, preceded by symptoms of gastric catarrh, is most probably catarrhal.

**TREATMENT.**—There is no special treatment for jaundice; in all cases the treatment must have reference to what is believed to be its cause. The appropriate treatment will therefore be discussed under the head of the several diseases which give rise to it. In a considerable proportion of cases, however, it is impossible to determine the precise nature in the first instance, and pending an exact diagnosis we are obliged to adopt some provisional treatment. Treatment should in no case be neglected, for what may appear to be a trivial jaundice may prove in the sequel to be an early stage of acute atrophy of the liver. There are therefore certain general rules of management which may be laid down. There is no need that the patient should be confined to bed, unless other symptoms indicate such necessity; moderate exercise may be allowed even in the open air, provided the patient is suitably clothed. The clothing should be warm, but light. The food should be simple and nutritious. There is no need to rigidly exclude fatty and saccharine

matter, but no excess should be allowed, and pastry should be avoided. Alcoholic beverages, especially fermented liquors, should be withheld. In some cases of long-continued jaundice considerable quantities of flesh are required, and are digested, to supply the fatty matter lost to the system by the absence of bile in the intestine. Milk is usually well borne, and supplies fat in an emulsified form. The patient should be encouraged to freely drink warm fluids or the natural alkaline waters, as those of Vichy, as the kidneys are the chief eliminators of bile. Warm baths should be used, and occasionally the Turkish bath is of service. At the commencement of an attack a dose of calomel or blue pill (gr. ij.) in combination with colocynth or rhubarb pill, followed by a saline aperient draught on the following morning, may be administered. Mercurials should be cautiously used as long as an uncertainty as to the exact nature of the case remains, but the evil effects of mercury have been exaggerated, and it has been determined that it diminishes, and not increases, the secretion of bile. An alkaline and bitter stomachic is often of much service, especially when the tongue is coated; 20 grains of bicarbonate of potassium or sodium, with 20 minims each of aromatic spirit of ammonia and spirit of chloroform, 5 minims of tincture of nuxvomica, in an ounce of some bitter infusion, as calumba, gentian, cascarrilla, or chiretta, recently prepared, may be given three times a day between meals. In some cases, when the tongue is clean, or has been cleaned by an alkali, acids appear to do good; 10 to 20 minims of diluted nitro-hydrochloric acid in an ounce of a bitter infusion should be given half an hour before meals, and taraxacum may be usefully added—20 grains of the extract, or a drachm of the succus. Aperients are in many cases required, and in most cases useful. They have the effect of lessening portal congestion, and replace the natural purgative quality of the bile. Saline aperients as a rule answer best. A drachm of Carlsbad salt in a tumblerful of warm water, or half a tumblerful of Carlsbad water, to which some hot water has been added, should be taken before breakfast, and its efficacy is increased if it is *sipped* instead of being taken at a draught. Another useful saline aperient is phosphate of sodium, a drachm three times a day in a bitter infusion. In some cases vegetable aperients answer, when colocynth, aloes, rhubarb, or cascara may be given. When diarrhoea is present, salicylate of bismuth (gr. x.), or oil of turpentine (ʒx. to xx.) in capsules, on an empty stomach, should be tried. When jaundice is not due to an insuperable obstruction, euonymin, iridin, podophyllin, and other hepatic stimulants, are sometimes useful; and when there is gastro-duodenal catarrh, ipecacuanha is often of much service. The following is a good

formula: R Euonymin gr. jss., iridin gr. j., ipecacuanhæ gr.  $\frac{1}{4}$ , pilulæ colocynthidis et hyoscyami gr. ij.; ft. pil.; one to be taken every or every other night. For flatulence, which often is troublesome, carbolic acid, creasote, menthol, and turpentine are useful. The following pill may be employed: R Aloin gr.  $\frac{1}{2}$ , acidi carbolici (seu menthol) gr. j., extracti taraxaci gr. ij.; to be taken night and morning. Nitrate of pilocarpine has been highly praised by Witkowski, and is certainly sometimes of great service in relieving the pruritus often present. One-sixth of a grain should be given subcutaneously once or twice a day. For the pruritus which is often such a distressing symptom, warm baths at night, followed by the use of a flesh-brush, are sometimes serviceable. Bicarbonate of sodium, with bromide of potassium or belladonna, sometimes affords relief, or, as already mentioned, pilocarpine hypodermically. The following lotion also relieves the itching: Diluted hydrocyanic acid  $\zeta j.$ , borax  $\zeta j.$ , water to  $\zeta viij.$  When these measures fail, and the night's rest is broken, an opiate may be required. Ox-gall is sometimes useful in supplying the place of the deficient bile. The fel bovinum purificatum (recently prepared) may be given in five- or ten-grain doses in coated pills, three times a day, two hours after meals. Daily enemata of one to two quarts of water at 60° to 90° F., to be retained as long as possible, are extolled by German physicians, especially in catarrhal jaundice. It cannot be too strongly impressed that, whilst using these measures, the physician's aim should be to establish the exact nature of the jaundice, when the correct treatment will be more clearly indicated.

CHARLES MURCHISON. STEPHEN MACKENZIE.

**JEJUNUM, Diseases of.**—See **INTESTINES, Diseases of.**

**JIGGER.**—A popular term employed to designate the sand-worm or sand-flea. See **CHIGOE.**

**JOINTS, Diseases of.**—Diseases of the joints are classified according to the structure primarily or chiefly involved. They may commence in the synovial membrane, in the bone, or in the cartilage. Primary disease of the ligaments is rare, and is not clinically demonstrable. No form of joint-disease remains long confined to one tissue, so that when the disease is of some duration it will be found to implicate, more or less, every element of the joint-apparatus. In this article the diseases of joints will first be generally discussed; and the individual diseases will then be considered separately.

**ÆTIOLOGY AND PATHOLOGY.**—The larger articulations, those in constant use, and more especially the joints of the lower extremity, are the most frequently diseased. Thus the

knee is more often the seat of disease than any other joint; the hip-joint comes next in order; and then the ankle and elbow. All kinds of joint-diseases are frequent in children and young persons. The first year of life appears, however, to be nearly exempt from these affections, and during the second year they are comparatively rare, perhaps because movement and risk of injury are at that period at a minimum. Acute arthritis, however, is occasionally witnessed during the first year of life—during even the first six months. It is unconnected with syphilis or injury; very sudden in its appearance, and rapid in course; dangerous to life; and destructive to the articular ends of the bones by suppurative disorganisation. The causes of joint-disease in general are connected either with disordered nutrition, in which case it usually assumes an inflammatory type; or with disordered function. The latter may depend on the former, or be unconnected with it. Again, the cause may be local in its origin or arise from some constitutional defect. Amongst the *exciting* causes, injury is by far the most frequent. This being often slight, and not followed by any immediate consequences, the connexion is frequently overlooked. A blow, or a fall against the edge of a table or down stairs, may readily bruise the synovial membrane in such exposed joints as the knee or elbow, without causing any external sign. A slight hæmorrhage takes place into the synovial cavity or the sub-synovial areolar tissue, and serous effusion may speedily supervene; in this manner a common variety of acute or traumatic serous synovitis is produced. But although injury is a most fertile cause of joint-disease, the articulations may sustain most severe injury without becoming inflamed. It is rare to find any serious consequences result from dislocation; the joint usually perfectly recovering itself. Penetrating wounds of the joints are always serious injuries; they often occasion acute synovitis, and, if septic changes occur, are followed inevitably by suppuration in the articulation, and danger both to the limb and life of the individual. Fractures often implicate the joint-surfaces, and prove a frequent source of stiff-joint. Plastic synovitis may be thus set up, causing adhesions; or suppuration takes place; or the callus formed for the repair of the fracture may interfere with the joint motion. Gunshot wounds of joints often produce the severest form of inflammation, suppuration being the usual result. When joint-disease follows an injury it is usually confined to one joint; but when joint-disease originates from constitutional causes, more than one joint is often affected; or when only one, the constitutional nature of the cause is manifested in diseased conditions present elsewhere, or by traces of inflammation in other joints due to the same cause. The development of tubercle in the

synovial membrane and bone is a very frequent cause of chronic joint-disease.

Joint-inflammations are of common occurrence in all kinds of fever; and also as sequelæ of the exanthemata. The great frequency of polyarticular serous synovitis in acute rheumatism is well known, as also in purpura and hæmophilia, where it is complicated with blood extravasations. In pyæmia the joints are frequently the seat of seropurulent and purulent effusions; as they also occasionally are in scarlatina. Puerperal synovitis is a variety of the pyæmic. In typhus monarticular arthritis is frequently met with, and the hip is the joint oftenest affected. Endocarditis and polyarthritis are very often associated together, and the endocarditis may precede and give rise to the joint-disorder by embolism. The fact that multiple joint-affectations are met with both in pyæmia and in rheumatism suggests a connexion, but what its nature may be is not clear. Although in articular rheumatism pus-formation is rare, we sometimes witness joint-suppuration in such cases; whilst pyæmia and metastatic abscess may originate from ulcerative endocarditis. In chronic synovitis, affecting two or more joints, the heart should always be examined, for traces of endocarditis will sometimes be found. In the exanthemata, typhus, and diphtheria, metastases in the shape of joint-inflammations more or less frequently take place. Joint-inflammation is of frequent occurrence in dysentery. With gonorrhœa a form of arthritis is associated which is called 'gonorrhœal.' Syphilis in the later stages frequently attacks a joint, the knee by preference, syphilitic deposits taking place in the bone or the sub-synovial connective tissue, but synovial effusion is not common. A suppurative inflammation of the ends of the bone is not rare in children the subjects of inherited syphilis. In gout the joint-structures are affected; as a rule the peri-synovial tissue becomes inflamed owing to deposits of urates. Similar deposits also occur in cartilages of encrustation. The great toe is most often affected, but the other tarsal, digital, and larger joints are frequently diseased.

Some ill-understood form of vaso-motor or trophic irritation appears to occasion arthritis, in locomotor ataxy. Effusion into the joint is preceded by pain; and the knee and shoulder joints are those generally affected. In some cases of the disease changes similar to those in rheumatoid arthritis have been observed, and generally in the knee, shoulder, elbow, or hip. They occur early in the disease; arise suddenly; are often monarticular; and not rarely give rise to dislocation, especially in the shoulder. These characters distinguish the disease from ordinary rheumatoid arthritis. Severe inflammation of the joints of the paralysed limbs has been observed in cases of hemiplegia. The occurrence of joint-

disorder, usually synovial inflammation, is frequent in chronic disease of the spine; and it also occasionally happens in acute myelitis in the form of suppurative arthritis. In both cases the knee is most frequently affected.

**ANATOMICAL CHARACTERS.**—Joint-disease may begin as an inflammation of the synovial membrane, of the bone, or of the cartilage. Fibrous tissue having but slight tendency to inflame, it is improbable that primary disease affecting the ligaments can be otherwise than most exceptional; but these textures very soon become secondarily affected, from their intimate connexion with the synovial membrane. The synovial membrane is perhaps more ready to inflame than any other tissue in the body, and in many joints it is much exposed to injury from without, while excessive joint-movement alone is sometimes sufficient to excite synovitis. Primary disease of the bone comes next in order of frequency. Cartilage is least likely to take on primary disease. Each of these tissues, however, becomes speedily affected by disease which has invaded or commenced in the other.

**SYMPTOMS AND DIAGNOSIS.**—The local symptoms of joint-disease have reference to *impairment of function* and *change in form*; together with *pain*, both local and sympathetic; and certain *physical signs*.

*Impaired function.*—Usually this is great in proportion to the natural mobility and importance of the joint, and most evident in the extremities. The earliest symptom in hip-joint-disease is a slight limp or halt, whilst in other joints mere stiffness occurs; the full range of movement is simply curtailed, before actual pain or swelling takes place. The position of maximum relaxation—namely, that intermediate between flexion and extension—is commonly assumed by diseased joints. Even in the early stages of disease, the interference with movement is often very great, amounting to a sort of vital ankylosis, produced by the action of the muscles, whose tension prevents the joint-surfaces moving upon each other—an effort to avert pain. This form of ankylosis disappears during narcosis. Muscular or vital ankylosis must be distinguished from the rigidity produced by structural changes. Both synovial effusion and peri-synovial infiltration mechanically hinder free joint-movement.

*Changes in form.*—Changes in form are due to the alterations in shape and texture of the joint-structures, and to effusions within its cavity. These changes may be best appreciated by careful measurements, and a comparison with the opposite joint. No true estimate of the amount of departure from the normal is otherwise possible. The practitioner is thus better able to diagnose the special character of the swelling, whether it be due to synovial effusion, and confined to the limits of the capsule, causing it to bulge at the least protected parts; or to chronic

thickening of the synovial membrane, recognised on palpation by its elasticity and more general diffusion; or to disease of the bone and periosteum, when the swelling is deep-seated and hard. By accurate comparison a fluid collection outside the joint, either an abscess or a bursal tumour, may be distinguished from intra-articular swelling.

*Pain.*—The character of the pain is an important symptom in diseases of the joints. In acute synovitis it is severe and lancinating. In bone-inflammation it is a dull aching pain, with marked local tenderness, liable to periodic exacerbations of an intense kind. Often the pain is of a shooting, starting character, wakening the sufferer from sleep. The pain is of this character and most severe in sub-articular osteitis. Pyæmic suppuration and chronic synovitis are generally painless.

*Physical signs.*—When one hand is laid flat upon a diseased joint while the other moves it, certain sensations or sounds are often distinguishable. A peculiar soft crepitation, due to the presence of blood-clot, must not be mistaken for the rougher sensations which adhesions afford, the friction-sounds of movable joint-bodies, or the grating of exposed bone. The rubbing of one granulation-surface upon another may be likened to that of two pieces of velvet. Abnormal movements, such as lateral motion in a ginglymoid joint, usually imply extensive joint-disorder. Displacement or partial dislocation, and alteration in form of the joint-surfaces, occur as the disease progresses. A notable increase of local heat may be felt in all inflamed joints. When fistulous tracks exist around a diseased joint, they do not often, on being probed, afford direct evidence of the condition of the joint, but they generally prove the existence of articular suppuration, and disease of the bone.

With respect to the *general symptoms* of diseases of the joints, it need only be said here that the amount of pain or constitutional disturbance in acute cases varies according to the extent and acuteness of the disease, and the presence and amount of suppuration. In chronic disorders the associated constitutional condition should be investigated.

*COMPLICATIONS AND SEQUELÆ.*—The complications which occur in joint-disease are generally connected with long-continued suppuration. Amyloid degeneration of the viscera is pretty certain to be present when suppuration has existed for a year or more in young people; less certainly in adults. Hectic fever, tuberculosis, or pyæmia may occur at any period. In the absence or failure of treatment, the patient, should he survive, will suffer from contraction, deformity, and imperfect growth of the limb, together with more or less complete loss of function.

*PROGNOSIS.*—The prognosis in diseases of the joints will depend on many circumstances,

and must be considered both as regards life and as regards function. First, with respect to *life*, the gravity of joint-diseases increases with the size of the joint affected. They are more serious in the lower than in the upper limb. Pyæmia is comparatively rare in acute joint-suppurations—why, it is impossible to say. When pus escapes from the interior of a joint into the surrounding tissues, pyæmia may occur. A continuous high temperature, or a large evening increase associated with hectic, are bad signs; the exhaustion, which depends on profuse suppuration with its attendant hectic fever, amyloid degeneration, and tuberculosis, are the most frequent causes of death in joint-disease. The prognosis as regards *function* is often difficult to determine. After an attack of simple acute or subacute serous synovitis, recovery is generally complete. Joint-function is usually absolutely lost after suppuration of traumatic origin, recovery being quite exceptional. When the suppuration is of a pyæmic nature and the patient survives, the effusion may become absorbed, and the joint-motion be preserved or ankylosis ensue. Chronic synovitis with thickening of the sub-synovial tissue, due to infiltration with granulation-material, can seldom be cured except by operation, especially after suppuration has taken place. If recovery should take place, the joint-function is lost, and deformity is always present. Increased mobility—'flail joint'—is a very rare sequence of joint-disease. It is occasionally seen in the shoulder, and also in the knee.

*TREATMENT.*—The treatment of diseases of the joints must be directed to preserve the life of the individual; and, as far as possible, the functions of the limb. Of the first and greatest importance among remedial measures is *rest*, which is best secured by fixation of the joint and limb in an appropriate apparatus. This is of cardinal importance to a diseased articulation, just as motion is a necessity for a healthy one. Immobilisation should not be continued longer than necessary; it will sometimes seriously damage even a previously healthy joint, immobilised for instance on account of fracture of the limb; and a continuance of rest after all diseased action has subsided often exerts a prejudicial influence. Rest, however, should be continued so long as swelling, pain, and increased temperature persist.

*Position.*—The means adopted to secure immobility must be also utilised to obtain the best available position for the future function of the part, should ankylosis become inevitable. In the ankle the foot should be maintained at a right angle; the hip and knee must be kept extended in the axis of the body; the elbow is generally flexed to a right angle, the position in which the limb is most useful. Splints of various forms are used, and we possess in plaster-of-Paris and

starch ready and invaluable means of producing an apparatus which gives uniform and equable support of a simple and very perfect kind.

*Extension* exerts a beneficial influence, as well by immobilising the joint, as by its power to remove contraction and deformity. It relieves pain and abates the symptoms rather by keeping the joint at rest, and changing the surfaces of contact, than by any actual distraction of the joint-surfaces. A much greater weight than a patient could tolerate would have to be used before any such separation could occur. Extension often even increases the intra-articular pressure. By straightening the limb it removes the joint from its position of maximum relaxation, and puts the skin and tendons on the flexor aspect on the stretch, and alters the mutual accommodation of the joint-surfaces.

As there is almost invariably an increase of temperature in the affected joint, the application of *cold*, by means of ice-bags or coils of cold-water tubing, is indicated. Cold acts most beneficially in all acute, and many subacute, inflammations. Even in deep-seated joints like the hip, it will often soothe the pain and abate the symptoms, but it is more applicable to the superficial joints. Cold is both anæsthetic and prophylactic in its action. The sensations of the patient in respect of the continuance of cold applications must be consulted. In most cases they are grateful. In chronic joint-affections when an acute attack supervenes, threatening suppuration, cold should also be applied. Cold applications may in some cases be continued for weeks or months with advantage. When the acute symptoms have passed off, and it is desirable to encourage lymphatic activity and absorption, the cold must be discontinued, and compression, together with friction and warmth, substituted. Cold is not applicable to purely chronic cases without much pain or tenderness.

In some instances of acute and subacute arthritis *local depletion* by means of leeches or scarification is very useful, and this may be combined with hot *fomentations* in cases where cold is not well borne. In chronic inflammation of the bone the *actual cautery* sometimes procures immediate abatement of the pain, and, after a time, the subsidence of the inflammation. The button cautery may be used, or, still better, linear cauterisation, over the most sensitive points. Or an *issue* may be employed instead.

*Compression* by strapping, or with a thick layer of cotton-wool and a tightly applied bandage over it, is applicable to the chronic stages of joint-disease. It must be discontinued if it occasion pain. It is better calculated to remove fluid effusions than the plastic material poured out into the peri-synovial tissue. For these cases the more continuous compression of a properly applied elastic

bandage will prove more efficient; or the strapping known as 'Scott's dressing.'

*Massage* is a most valuable local means for the dispersion of chronic swellings of joints. It both removes the results of diseased action in the joint, and helps to restore its function. It is well suited to disperse serous effusions when the acute stage is over; for cases of plastic synovitis it is also useful, but not for cases of the type known as *tumor albus*. It produces a diminution of the sensibility of the part, and a local increase of temperature, and the lymphatics are stimulated to increased activity. There are several modes of employing massage. The first is centripetal stroking with the palm of the hand from the periphery of the affected part towards the centre of the body, called *effleurage*, one hand following the other in immediate succession. The amount of pressure varies with the circumstances of the case. This will readily disperse fluid effusions both of blood and serum. Friction-massage is another useful method, and is practised by pressing the palm firmly upon the surface, and then rotating it. This plan may be combined alternately with the last method, massage proper—*pétrissage*—which is done by raising up the soft parts vertically from the bone with both hands, and compressing them, always in a centripetal direction.

*Forcible movements*.—Forcible movements, which break down adhesions, are often most useful in cases of stiff joint arising after protracted immobilisation, as after fracture in the vicinity of a joint, or after a severe sprain. Pain will be relieved in this way, and mobility restored in some instances, in a degree quite remarkable. They are less applicable in cases of fibrous ankylosis.

*Constitutional treatment*.—Where any general taint exists, this must be dealt with at the same time. A tendency to tubercle must be met by iron, tonics, good food, and fresh pure air. Gout, syphilis, or rheumatism must, when present as a diathesis, be appropriately treated.

*Operative treatment*.—Puncture alone, or combined with antiseptic washing-out of the articulation, may often be performed with advantage, to evacuate the fluid in a distended joint, or to diagnose the presence of pus and evacuate it when suppuration has taken place. Sufficiently free incisions, however, and the insertion of drainage-tubes, are generally to be preferred in cases of joint-suppuration; and with these should be combined the washing-out the joint-cavity with a three- or five-per-cent. solution of carbolic acid, or other effective antiseptic. It has been proposed to substitute free incisions and drainage for excision of the joint, in certain chronic forms of disease, such as white-swelling, but excision, or arthrorectomy, is probably in most respects preferable. Excision is practised for chronic joint-disease not amenable

to other means; it is not a substitute for amputation, but is intended to obviate its necessity. Sub-periosteal resection, where practicable, possesses many advantages, especially in cases of traumatic origin. The attachments of the muscles and tendons, and the cellular interspaces between them, are thus left undisturbed. The chances of peri-synovial suppuration are diminished, and the bleeding is reduced to a minimum. There is more complete bony reproduction of the joint-surfaces, and in young persons a new joint very similar to the normal is in some instances formed, while in all cases there is a probability of better subsequent function and position. The operation thus performed requires time and skill. It is scarcely applicable to the knee or even the hip, and is unsuited for cases of chronic synovial disease, where it is of the last importance to excise all the diseased granulation-material. The after-treatment of excisions is of great importance. Plaster-of-Paris bandages supply one of the most useful means of immobilisation, especially in those cases where ankylosis is sought for, as in the knee; and the splint should be unchanged, if possible, for four or five weeks. In the elbow, shoulder, and wrist, where mobility is the end aimed at, passive movement should be commenced as soon as the condition of the wound admits of it—namely, in about a week or ten days. Galvanism must be used at a later period to restore the wasted muscular apparatus. Arthroectomy aims at the removal of all the diseased tissue, yet stops short of a formal excision in which healthy parts may be removed.

Amputation is only performed as a last resort. It is very rarely needed for joint-diseases in the upper limb, except perhaps the wrist, when the hand is permanently crippled. In the lower limb, amputation must be performed in those cases in which the patient has lost all strength and healing power, from the drain of a long-continued discharge. It is advisable where amyloid degeneration or incipient tuberculosis exists, or in any case, in short, in which the power of the patient is inadequate to furnish the amount of repair required in the expectant form of treatment, or in case of excision, always a more serious operation than mere amputation. Amputation should also be adopted in those cases in which the local disease, especially of the bone, is too extensive to admit of a good functional result after excision. In the very young formal excision is undesirable, since the epiphyses are almost of necessity sacrificed, and the growth of the limb checked. Resection in some joints is practised to avert ankylosis, or to restore the lost function of the joint, as in the shoulder, elbow, and wrist. Various congenital and other deformities of the joints may be removed by osteotomy of the bones concerned.

The chief diseases of joints will now be separately considered in alphabetical order as follows:—(1) Cartilages, Diseases of; (2) Congenital Dislocation; (3) Immobility; (4) Inflammation, Acute, of the Synovial Membrane; (5) Inflammation, Chronic, of the same; (6) Gonorrhœal Inflammation; (7) Gouty Inflammation; (8) Strumous or Tuberculous Inflammation; (9) Loose Cartilages in Joints; (10) Nervous Affections; (11) Rheumatic or Osteo-arthritis, Chronic; (12) Serous Effusions; and (13) Syphilitic Disease. Rheumatism in its several forms will be considered separately.

**1. Joints, Cartilages of, Diseases of.**—Primary chondritis as a form of joint-disease is neither clinically nor pathologically established. The cartilage has little tendency to active inflammation; it is not sensitive to pain; chronic changes take place in it as the result of impaired nutrition, rather than inflammation. The cartilages of incrustation are the residue, which does not ossify, of the mass of fetal cartilage forming the bone-ends. We meet in the joints of the aged with depressions and fibrous scars, caused by partial atrophy of the cartilage; these changes do not occasion symptoms during life, beyond sensations of creaking and roughness in the joint when moved. In osteo-arthritis changes in the encrusting cartilages occur at a very early period, and may possibly in some cases form the primary change.

Almost all the changes which occur in the articular cartilages are secondary to synovitis and ostitis, and more or less passive. In serous synovitis the cartilage is softened and swollen, and becomes to a certain extent œdematous. In suppurative synovitis it becomes rough, sodden, and yellow; its cells burst; the intercellular substance fibrillates; and portions may necrose. The cartilage-changes in chronic synovitis consist mainly in the gradual invasion of the cartilage by granulations, spreading from the synovial tissue at the margins and from the subjacent articular lamella of the bone.

Young growing bones, like the periosteum covering them, readily inflame; and as soon as the inflammation invades the articular surface of the bone, the cartilage becomes loosened and may be necrosed, or gradually invaded by the granulation-tissue springing from the bone. The cartilage is softened; its capsules burst; fibrillation of the matrix occurs; and at different places it becomes thinned. Perforations occur in it, leading to the bone, giving it a sieve-like appearance. Where the inflammatory process is more acute, the whole of the encrusting cartilage may become at once detached from the bone beneath, and necrose.

In these cases the pain, especially at night, is very severe. Involuntary starting of the limb causes intense suffering, wakening the

patient from sleep with a scream. This symptom was formerly considered distinctive of ulceration of cartilage, a process which is perfectly painless when it occurs without other disease in the joint; but it is probable that the pain is due to pressure upon the inflamed bone, and it may sometimes greatly abate when the cartilage wholly disappears. Disease commencing near the epiphysal cartilage often extends to the adjacent joint. Those joints in which the epiphysal junction is within the capsule, as, for instance, the hip, run a double risk from bone-inflammation.

**2. Joints, Congenital Dislocation of.**—DESCRIPTION.—This is a curious and ill-understood affection of the joints. It is almost exclusively confined to the female sex. The hip-joint is nearly always the one affected, and the displacement is generally double. It has often been erroneously mistaken for morbus coxæ, and treated accordingly.

It probably occurs in early fetal life from defective formation of the joint-surfaces. It is not discovered until the child begins to walk, which it generally only commences to do at a late period. When the hips are the joints affected, the gait is accompanied by a most ungainly swaying of the body from side to side like the waddling of a duck. All that can be done is to supply a well-contrived artificial support to the pelvis and limbs.

*Genu valgum* and *genu varum* (knock-knee and bow-knee) are two forms of disabling and unsightly deformity of the lower limbs. They are often due to an abnormal development of the condyles or shaft of the femur, or the tuberosities of the tibia, and to changes of a rickety character at the epiphysal junction. In extreme cases progression is greatly interfered with. The symptoms are painfully obvious.

TREATMENT.—Treatment of the limb by splints and apparatus and forcible straightening and subsequent maintenance in plaster-of-Paris bandages succeeds in the milder cases, but in those of a severer type, or in persons of ten years or upwards, this plan is most tedious, and is usually attended by complete failure. The subcutaneous division of the internal or external condyle, according to the nature of the deformity, restores the joint-surfaces of the femur to their normal level, and is attended by the happiest results both to the form and function of the limb and joint. It is an easier and in many ways a more successful operation to divide the shaft of the bone with a chisel near its articular extremity, and thus procure the rectification of the limb.

**3. Joints, Immobility of.**—SYNON.: Ankylosis; Fr. *Ankylose*; Ger. *Gelenkverwachsung*.

ÆTIOLOGY.—This condition may be due to

changes in the structures of the articulation constituting a *true ankylosis*; or in those surrounding the joint, a *false ankylosis*. It may be fibrous and incomplete, or bony and complete. False or spurious ankylosis—extra-articular—may depend on muscular spasm or rigidity; on cicatricial contractions; on paralytic or spasmodic affections; or upon prolonged disuse of the joint. It is often difficult, even under chloroform, to distinguish the presence of absolute bony ankylosis, as the fibrous form may be so strong and extensive as almost wholly to prevent movement. The two varieties are but degrees of the same process. Both may result from previous inflammatory changes in the joint, either of the nature of plastic synovitis, or of granulations springing from the bone and other joint-tissues, becoming further organised. Fibro-cartilaginous ankylosis is a common form in young persons. In time it usually becomes converted into true bony ankylosis.

The marked ankylosis which tonic spasm and rigidity of the muscles produce in the early stages of some joint-diseases, as in the hip-joint and knee, may be called *vital* or *physiological*. It is induced by an effort to avert pain; it disappears entirely during narcosis. A joint may become stiff and ankylosed by long fixation on account of some injury or disease elsewhere, especially if it be retained in a flexed position, as the muscles of the flexor side actually shorten when their points of origin and insertion permanently approach one another. The same thing may also happen in the myogenic affections due to paralysis; in which the cartilages and bones atrophy at the same time.

TREATMENT.—The treatment of diseases of joints should, in all cases where it is possible, be prophylactic against the occurrence of ankylosis. When this has taken place in an incomplete degree, an attempt to restore the function of the limb must be made, by breaking down the adhesions by forcible or gradual extension, by passive motion, by massage, and by tenotomy of the tense tendons. Excision is indicated to restore motion in complete ankylosis of such joints as the wrist, the elbow, and the shoulder. In other cases of complete ankylosis, especially in the lower limb, surgical interference should be confined to an attempt to rectify a faulty position either by tenotomy, extension, forcible straightening, excision, or osteotomy. Amputation can only be needed in extreme and otherwise irremediable deformity.

**4. Joints, Acute Inflammation of the Synovial Membrane of.**—SYNON.: Acute Synovitis; Fr. *Synovite Aiguë*; Ger. *Acute Synovitis*.

This is one of the most common of all joint-affections. Probably in half the total

number of chronic joint-diseases the synovial membrane is first affected. The synovial membrane is very rich in vessels and cells; and much exposed to injury, and to the effects of movement. An inflammation beginning at one point soon spreads over the whole synovial sac.

**DESCRIPTION.**—Acute synovitis is a very common result of injury. It also occurs in rheumatism, gout, pyæmia, and other diseases. It may be serous, sero-fibrinous, or purulent. Serous synovitis is the simplest and most common variety. Even a slight external injury is often sufficient to produce it; a sprain of the joint, or excessive movement, may cause it, as well as the constitutional disorders already mentioned. The knee is very often affected, from its exposed position, and the large area of the serous membrane lining it. The synovial membrane becomes injected and thickened, with effusion into the sub-synovial tissue. The natural secretion is increased in quantity, and many cells are shed into the joint cavity, the capsule becoming swollen, tense, fluctuating, or elastic. The least protected parts bulge, from the pressure of the effused fluid, and the normal contour of the joint is lost. It is usually semiflexed. A severe burning, cutting pain is experienced in it. It is exceedingly sensitive to pressure, and painful on the slightest movement. There is usually considerable fever. When the inflammation is more intense, the synovitis becomes sero-fibrinous. Flakes of lymph are mingled with the synovia; layers of false membrane cover the synovial membrane, which is considerably thickened and dull red in colour; and the constitutional disturbance is greater. Finally, it is but a short step from this to suppurative synovitis. If the irritant cause continue its action, the leucocytes filling the meshes of the synovial membrane are shed in larger quantity; the fluid becomes turbid and puriform; the fever and local symptoms increase very much in severity; the external parts become implicated in the inflammation within; and suppuration or abscess is the result.

**TREATMENT.**—The treatment of the first two stages of synovitis is directed primarily to check the progress of the inflammation; and then to procure resolution, and absorption of the effused fluid. Fortunately the synovial membrane possesses very active absorbent powers; and early and efficient treatment, conjoined with removal of the source of irritation, will generally ensure a cure, with complete restoration of function. The chief means are cold applications; immobilisation till the acute stage is over; and then compression and friction, or in suitable cases counter-irritation. Gout, rheumatism, or other diathesis, must be appropriately treated at the same time.

In those cases in which the inflammation

has persisted for some time, and plastic effusion has taken place on the surface and in the substance of the synovial tissue, the joint will remain for a long time stiff and thickened, and its function impaired, after all acute symptoms have subsided; whilst in those cases where the inflammation has continued long enough to invade the other joint-structures, a perfect cure may not be possible.

When suppuration occurs, the joint must be dealt with as any other abscess-cavity. Free incisions must be made into it, and it should be thoroughly washed out with some antiseptic solution, free subsequent drainage being provided for. In the more favourable cases, ankylosis in a convenient position will be obtained. Recovery of function is very rare. In other cases the suppuration continues; the cartilages of encrustation become necrosed and detached; the bone becomes exposed and carious; and either excision or amputation must be performed to save the patient's limb or life.

### 5. Joints, Chronic Inflammation of the Synovial Membrane of.—SYNON.: Chronic Synovitis.

Chronic synovitis may arise as the sequel of the acute disease; or, as is more frequent, it may depend on some constitutional dyscrasia, or at least some continuously acting irritant, although in the first instance it is generally excited by an accidental injury.

**VARIETIES.**—There are three chief varieties of this disease which are often co-existent, namely: *pannus synovitis*, *granulation synovitis*, and *papillomatous synovitis*.

*a. Pannus Synovitis.*—In this variety a delicate membrane will be found, stretching from the inflamed and thickened synovial margins more or less over the surface of the cartilage, to which it may be in whole or in part adherent.

*b. Granulation Synovitis.*—The second, or granulation form, is a more advanced stage of the disease. In it the synovial membrane is often completely replaced by granulation-material, which encroaches on the margins of the cartilages, and, as the disease progresses, invades them, and coalesces after a time with granulations springing from the inflamed bone. These granulations are pink or greyish-red, not so vascular as those in an ordinary wound. They have little tendency to cicatricial change, and are prone to break down and form abscesses.

The synovial membrane often becomes enormously thickened from the granulation-material accumulated in it, and this form of disease is frequently associated with the tubercular diathesis. Bodies identical with military tubercles are very often found in the semi-gelatinous or pulpy synovial membrane; the whole constituting what is known as *tumor albus*, or 'white-swelling.'

**SYMPTOMS.**—The more prominent symptom

associated with the form of chronic synovitis just described is the presence of a uniform semi-elastic swelling, caused by the sero-fibrinous infiltration into the synovial membrane. The skin is whitish, tense, and smooth, whence the name *tumor albus*. The amount of synovial effusion into the joint is usually limited; the pain is seldom severe; the amount of joint-movement is diminished; and there is usually contraction of the limb, and sometimes partial dislocation. When suppuration occurs, there is more pain and fever. The course of the disease is generally very chronic, extending over months or even years, and allowing the patient at intervals to take moderate exercise. Exacerbations take place, however, from slight causes, or without apparent cause; attacks of subacute or acute inflammation supervene; and sooner or later abscesses form, communicating both with the cavity of the joint, which becomes totally disorganised, and with the surface.

Brodie's 'pulpy disease of the synovial membrane' is simply an exaggerated degree of granulation synovitis, in which the sub-synovial tissue becomes enormously thickened by successive attacks of inflammation.

**TREATMENT.**—The disease is most rebellious to treatment, and scarcely curable amongst the poorer classes without operation. Sometimes the diseased tissue is got rid of by suppuration; and, if the patient's strength suffice, a cure by ankylosis, probably accompanied by considerable deformity, will ensue. Generally, however, the symptoms become worse; the general health gives way from the drain of the continuous discharge; and amyloid or some other intercurrent disease kills the patient. At any time, as in a suppurating wound, pyæmic symptoms or hectic may set in.

*c. Papillomatous Synovitis.*—**SYNON.**: Fimbriated Disease of the synovial membrane; Papilloma, or Papillary Fibroma of the synovial membrane; Ger. *Gelenkzotten*.

This is a peculiar form of joint-disease dependent on chronic synovitis, in which numerous pedunculated bodies, cylindrical or fusiform, varying in size from a pin's-head to a large pea, project from the membrane, generally near the cartilage margins, or may cover the entire surface. These bodies are identical in minute structure with the synovial fringes. Some become detached, and fall into the cavity of the joint. The disease is essentially a hyperplasia of the synovial adventitia; and there may be dozens or hundreds of these bodies present in one joint. Occasionally they contain cartilage cells, or osseous particles. The joints of the lower limb are most often affected.

**TREATMENT.**—The disease is scarcely remediable except by arthrectomy or excision of the joint, which may be practised in those cases in which there is serious loss of function.

**6. Joints, Gonorrhœal Inflammation of.**—This disease is often called 'gonorrhœal rheumatism.' It is almost always observed in the male, very rarely affecting the female. Generally one joint, usually in the lower limb, is involved, as the knee or the ankle. It is pyæmic in nature. It may affect several joints in succession. It may occur at any period of a gonorrhœal or even a gleet discharge, of which the patient himself is possibly unaware. The fibrous tissues of the joint seem to be primarily engaged. Sclerotitis and inflammation of the internal layer of the cornea often co-exist; rarely either endo- or peri-carditis. The joint-effusions contain gonococci.

**SYMPTOMS.**—The joint affected is exquisitely tender on pressure; it is swollen rather from subcutaneous œdema than by intra-articular effusion, which is frequently inconsiderable; and one side of the joint is often more affected than the other. The fever is not in proportion to the intensity of the local symptoms, the rise of temperature being slight. The course of the disease is slow and obstinate; but when cured it is not liable to return, except with a fresh attack of gonorrhœa. These characters distinguish it from ordinary rheumatism.

**TREATMENT.**—Rest, cold applications, and the internal exhibition of iodide of potassium constitute the best treatment for gonorrhœal inflammation of joints. See **RHEUMATISM, Gonorrhœal.**

**7. Joints, Gouty Inflammation of.**—Gout frequently occasions synovitis, by the deposit of urate of sodium in the peri-synovial tissue, which excites a synovial effusion. The attacks are very acute and painful; and, as they recur, the joint becomes more and more disorganised, from permanent deposits of urates in the cartilages and bone, as well as in the peri-synovial tissue. The use of the joint is lost; and the 'chalkstone,' as it is called, acting as a foreign body, sometimes produces an abscess, or an ulceration very troublesome to heal. When the collection is quite superficial, it may often be evacuated by incision with great relief. See **GOUT.**

**8. Joints, Strumous or Tuberculous Inflammation of.**—Scrofulous or strumous inflammation of a joint is a vague term, hitherto taken to denote a form of chronic inflammation in ill-nourished persons—nearly always children—who live under bad hygienic conditions, and are prone to the development of tubercle. It is observed most frequently in the knee- and hip-joints.

Recent investigation has shown that many forms of chronic disease of joints are due to tuberculous infection of the constituent parts of the joint.

A considerable proportion of the cases described under the title *tumor albus* are cases of tubercular disease of the synovial membrane; while in others the articular

disease is the consequence of tubercular ostitis of the ends of the joint.

**ANATOMICAL CHARACTERS.**—This disease generally commences in the synovial membrane, which becomes thickened, and by degrees converted into a semi-gelatinous mass of granulation-tissue, yellowish-white or pink in colour. Or the disease may originate in osteomyelitis of the end of the bone, the inflammatory process being essentially chronic and dependent upon the presence in the tissues of miliary tubercle. After the disease has existed for some time, it is difficult to determine in what tissue it may have originated, and it is of little clinical importance to do so, for in any case the later stages of the malady present similar features. The cartilages are encroached upon from their margins and from their deep surfaces by the granulations, springing from the synovial membrane and the articular lamella, whilst active changes occur simultaneously in their substance, similar to those already described. The ligaments soften, and all the structures of the joint become involved. Frequently larger or smaller masses of necrosed bone will be found in the cancellated structure, and the granulations have a great tendency to suppurate. In the thickened synovial membrane, and also in the ends of the bones, miliary bodies, identical with tubercles, may very frequently be detected.

**SYMPTOMS.**—In tubercular inflammation of the synovial membrane the joint is uniformly swollen, tense, elastic, with a white glistening surface, and enlarged veins shining through the skin. The patient can usually move about until suppuration has taken place, as the pain is never very severe in the intervals of the acute attacks of inflammation which supervene from time to time. Enlargement of the lymphatic glands, or marks of strumous ulceration elsewhere, are seldom wanting; whilst sooner or later a large proportion of the individuals affected by this form of joint-disease show signs of general tuberculosis. Sometimes this state precedes, but generally it follows, the local joint-affection. Inherited syphilis appears to be a predisposing cause of tubercular arthritis. When the disease begins in the bone the localised tenderness and deep-seated thickening, conjoined with the general symptoms, may reveal the nature of the process before the more general invasion of all the joint-elements.

**TREATMENT.**—This must be mainly directed to improving the patient's general condition. Any local treatment, short of a complete removal of the diseased structures, is not of the least use when the joint has become disorganised, as before described; and some form of excision should be performed before the viscera become implicated. If other organs be involved, or the local disease be too extensive, then amputation becomes imperative. When not removed, fresh foci of

suppuration form; the patient becomes more and more exhausted; or some intercurrent disease sets in. It is rare for spontaneous cure to happen.

**9. Joints, Loose Cartilages in.**—**SYNON.**: Fr. *Corps Flottants Articulaires*; Ger. *Gelenkmäuse*.

**DESCRIPTION.**—'Loose cartilages' in joints may originate either from chronic inflammation, or from traumatic causes. They may be single or multiple. The knee-joint is most frequently affected, and in it the most serious symptoms are produced. These bodies may be fibrous, lipomatous, chondromatous, or osteo-chondromatous. They may be produced from polypoid growths springing from the synovial membrane in certain forms of chronic synovitis, and in arthritis deformans; and they are then usually of the fibrous or osteoid variety. Lipomatous free bodies are rare, and are derived from the sub-synovial fatty tissue, being produced in a fashion analogous to the appendicæ of the great intestine.

The chondromatous and osteo-chondromatous are the largest and most important varieties of these bodies; hence the common term 'loose cartilage.' Portions of the joint-surface may sometimes become detached, as the consequence of injury, or by a process of quiet necrosis. They thus become loose in the joint. It is stated, but the fact is doubtful, that some of these bodies may obtain nourishment from the surrounding synovial fluid, and that cartilage and even bone can be developed in them subsequently to their detachment.

**SYMPTOMS AND DIAGNOSIS.**—The symptoms of loose cartilages in a joint vary very much. In some instances these bodies cause no inconvenience. In others they produce repeated attacks of excruciating pain, followed by synovitis, laying the patient up for weeks; whilst in the most severe cases the limb may become almost useless. When the knee is the joint affected, the patient experiences great insecurity in walking, the loose body from time to time becoming wedged between the joint-surfaces. The joint is thus 'locked.' The patient may suddenly fall, or faint with pain; an attack of synovitis follows; and with a frequent repetition of this process joint-disorganisation may finally result. The prognosis as regards function is always bad.

**TREATMENT.**—The treatment of loose cartilages may be either *palliative* or *radical*.

The *palliative* method consists in applying support to the joint; limiting its movements; and fixing the loose body in some synovial pouch where it cannot interfere with the articular surfaces.

The *radical* method consists in excising the body—an operation, with few exceptions, almost exclusively practised upon the knee-joint. The body may be removed by a free direct incision into the joint, and squeezing

the body through the wound at once. Or the indirect manner of operating may be adopted. This consists in subcutaneously incising the capsule of the joint with a long, narrow-bladed knife introduced at some distance from the articulation; forcing the body through this incision into the cellular tissue outside; and then closing the small external puncture in the skin. Three or four weeks later the 'cartilage' may be removed by a superficial incision, or left undisturbed, when it often becomes absorbed.

In appreciating the comparative value of these two plans, it may be said that the former has hitherto proved more uniformly successful *quoad* extracting the body, but that it has been more dangerous to limb and life—a danger, however, which antiseptic precautions reduce to a minimum. The indirect method has been attended by a considerable number of failures in the extraction of the loose cartilage, especially if it be pedunculated; but it has hitherto proved a less dangerous operation. The extremity should be immobilised afterwards for two or three weeks.

When some dozens of these bodies are present in a joint, many of them free, many attached, excision of the articulation is often the only remedy. This is a severe measure, and not to be lightly undertaken, in the joints of the lower limb at all events.

**10. Joints, Nervous Affections of.**—**SYNON.:** Hysterical Joint; Neuralgia of Joints; Arthralgia; Fr. *Arthralgie Hystérique*; Ger. *Gelenkneurose*.

**DESCRIPTION.**—*Hysterical affections* present symptoms simulating real joint-disease so closely that the most energetic therapeutic measures have often been resorted to, though in vain, for their cure. Prolonged immobilisation, blistering, the actual cautery, resection, and even amputation, have been practised upon joints in which there was not a trace of organic disease.

The existence of hysterical affections of joints is denied by some; but assuredly they do occur; and most often in young women, well-to-do in life, with disordered catamenia. The same thing occurs, but less frequently, in young men. The disease is not witnessed under the age of puberty. The hip and knee are the joints principally complained of—most frequently the latter. An all-important feature of a hysterical joint is that, while the local symptoms may be intense, the general symptoms are either absent, or in no sort of proportion to the local.

A special character of this disease is that deep pressure is often less painful than superficial pressure; and that the pain and tenderness are vague, shift from one point to another, and will disappear at a given spot when the patient's attention is directed elsewhere. There is pain on movement, but of an indefinite character, and not so limited or

localised as in real disease. Nocturnal startings do not occur; the patient may enjoy uninterrupted sleep for hours. There is never a continuous rise of temperature, either general or local; the co-relation of the symptoms is not the usual one; the function of the joint is much more interfered with than the other features of disease present would appear to justify. There is an exaggerated fear of examination; and the *facies hysterica* is often well-marked. There may be thickening around the joint, and even marked synovial effusion into it; but these conditions are passive in character, and generally due to the treatment employed. The limb is wasted and consequently weak, but never to the same extent as in real joint-disease. Exacerbations occur at the menstrual period. A careful inquiry should be made into the history and antecedents of the case. An examination under chloroform will often afford important evidence; and the patient's symptoms will be improved afterwards by the movements then practised on the joint.

*Neuralgic pain* in the articulations may arise under different circumstances. It may be the referred pain, unattended by local lesion, which is so frequent in the knee in cases of hip-joint disease. Neuralgic pains in various joints are observed in the preliminary or early stages of chronic myelitis. In the first stage of locomotor ataxy the knee may be affected by severe neuralgia when the disease is low down in the cord; or the shoulders when it is at a higher point. Lastly, so-called neuralgia of a joint may really indicate some obscure lesion, as chronic inflammation of the bones entering into the formation of the articulation.

**TREATMENT.**—The methodical exercise of an *hysterical* joint is as plainly indicated as rest is imperative in a case of organic disease. The bowels should be regulated, as also the menstrual flow. Asafetida, iron, and quinine are most important remedies; and healthy mental and moral influences are valuable adjuncts. 'Get up and walk' is a good prescription in many such cases. Very careful and repeated examination should always be made, to exclude any possible form of chronic inflammation, before pronouncing a joint to be hysterical. It must not be forgotten, however, that after slight injuries which produce some inflammatory symptoms, those of hysterical joint may supervene, and persist long after all traces of organic disease have disappeared.

The treatment of *neuralgia* connected with a joint will necessarily vary with its cause. *See NEURALGIA.*

**11. Joints, Chronic Rheumatic Arthritis of.**—**SYNON.:** Osteo-arthritis; Fr. *Arthrite avec Usure des Cartilages*; *Arthrite Sèche*; Ger. *Altersabschleifung*.

**ÆTIOLOGY AND PATHOLOGY.**—The number

of names that have been applied to this disease betrays the obscurity enveloping its pathology. In nature it is, however, essentially a senile degeneration, accompanied by chronic inflammation; and is, in part, perhaps, the result of wear and tear of the joint. It is most common in hard-working people, exposed to the influence of wet and cold, and in the aged. One or many joints may be affected; generally the fingers, the toes, the hip, and the knee. It may be set up by injury, such as a sprain, dislocation, or fracture; or it may arise without known cause. It is difficult to say which of the tissues is primarily at fault, but sooner or later all become involved. The synovial membrane inflames; papillary outgrowths form upon it; the cartilage swells; and the ends of the bones enlarge. After a time the quantity of synovial fluid diminishes; the joint friction increases; the cartilages are rubbed away at the surfaces of contact; and finally the bone itself, which becomes denser by interstitial deposit, disappears. The surface is eburnated, and marked with striæ produced by friction; whilst deposits of new bone, which may often be felt externally, form around the margins of the joints, so that the area of its surfaces becomes greatly increased.

**SYMPTOMS.**—The symptoms of chronic rheumatic arthritis chiefly consist in constant pain, of a dull aching character, and worse at night. Motion becomes more and more difficult and painful as the disease advances; but ankylosis never occurs. Rough crepitus is felt both by the patient and the surgeon when the joint is moved. See RHEUMATIC ARTHRITIS.

**TREATMENT.**—The treatment of osteo-arthritis can only be palliative. It consists in the use of warm douches and other warm applications, and the administration of iodide of potassium internally. The disease is incurable.

**Charcot's Disease.**—This is a peculiar form of osteo-arthritis, affecting usually the larger joints, such as the knee, hip, and shoulder, and occurring in connexion with tabes. The disease, therefore, is of central origin and dependent on sclerosis of the spinal cord. In some cases, however, evidence of a central lesion is absent.

In many instances the changes in the joint are similar to those in osteo-arthritis, while in others there is much more rapid and extensive destruction of bone. Besides the destructive changes which occur in the joint, there is often abundant proliferation and ossification of the cartilage margins, so that new irregularly formed masses of bone are found, it may be to as great or even a greater extent than in osteo-arthritis.

**SYMPTOMS.**—The malady occurs as a rule somewhat early in the progress of a case of tabes. The onset is rapid, the synovial effusion greater than in osteo-arthritis, and the

wasting and wearing away of the cartilage and bones much quicker.

The swelling generally occurs in the affected articulation quite suddenly and without pain; there is no pyrexia or local evidence of inflammation. Most commonly the knee is affected. The general symptoms of incoordination may be strongly marked or altogether absent. It is well to carefully look for them in all cases of extensive chronic enlargement of a joint in the adult. See TABES DORSALIS.

**TREATMENT.**—The treatment consists in giving the affected joint rest, and in the employment of suitable remedies to control the progress of the central nervous trouble. The prognosis in most cases is very unhopeful.

**12. Joints, Serous Effusion into.**—**SYNON.:** *Hydrops Articulii*; *Hydarthrosis*; *Fr. Hydarthrose*; *Ger. Gelenkwassersucht*.

This is a form of chronic serous synovitis, in which there are no obvious inflammatory symptoms. The joint sometimes becomes greatly distended; the ligaments are stretched; and in consequence there is a sensation of tension and feebleness in the articulation. The knee and elbow are most frequently attacked; and the disease is often associated with a gouty or rheumatic diathesis, or with rheumatoid arthritis. It is very difficult to cure. The joint may be punctured and the fluid drawn off; or, still better, it may be injected and thoroughly washed out with iodine (equal parts of the tincture and water), or with a carbolic acid (2½ to 5 per cent.) solution. Relapses, however, are common.

**13. Joints, Syphilitic Disease of.**—This disease may originate in children, in the form of a suppurative osteitis at the junction of the epiphysis and diaphysis. Other signs of congenital syphilis will help to establish the diagnosis. The disease runs a rapid course, and the joint is frequently destroyed. In the adult a chronic plastic synovitis, due to gummatous infiltration of the peri-synovial tissue, or of the bone and periosteum, is the more common form. There is very little fluid effusion within the joint, but considerable impairment of mobility is produced by the thickening outside it. The progress of the disease is slow and painless, except at night or on motion. The history of the case; the presence of traces of syphilis elsewhere; and the effects of treatment, will help in establishing the diagnosis. The internal use of mercury and iodide of potassium, combined with local pressure by means of strapping with mercurial plaster, speedily effects a marked improvement and cure.

WILLIAM MAC CORMAC.

**JUGULAR VEINS, Physical Signs in connexion with.**—The principal physical signs in connexion with the jugular veins are distension, pulsation, and venous hum. See PHYSICAL EXAMINATION.

## K

**KELOID.**—SYNON.: Fr. *Chéloïde*; *Kéloïde*; Ger. *Keloid*.—*Keloid* and *cheloïd* are two words resembling each other in sound, and sometimes used indiscriminately, but differing altogether in origin and signification. *Keloid* is derived from κηλίς, a mark or blemish; whilst *cheloïd* derives its origin from χηλή, a crab's claw. The disease which we now recognise as cheloid was first described by Alibert under the name of *kelis*, with the synonyms *cheloïde* and *cancroïde*, and is therefore sometimes referred to as the 'kelis of Alibert' (see CHELOID). The term 'keloid' has, however, been applied by Addison to a blemish of the skin, resulting from a fibrous degeneration of the derma allied with scleroderma; hence the use of the term *Addison's keloid*. Both diseases are really fibromata; but one, namely cheloid, is a tumour, while the other, kelis, is flat, and often resembles a cicatrix. There is another obvious difference between them: cheloid is restricted to the derma, whilst kelis follows the subcutaneous connective tissue to the deeper textures of the body. The early dermatologists described kelis under the name of *morphæa*, and by that name it is still distinguished by some modern writers.

ERASMUS WILSON.

**KERATITIS or KERATODEITIS** (κέρας, a horn, the cornea).—Inflammation of the cornea. See EYE, AND ITS APPENDAGES, Diseases of.

**KERION** (κηρίον, a honeycomb).—A term applied to a pustular folliculitis of the scalp. The inflamed skin occurs in the form of one or several blotches of a deep red colour, prominent, and dotted over with yellow spots—the apertures of the follicles, from which the hair has been expelled, and which exude a copious muco-purulent fluid. The yellow spots are converted into hollows by the tumefaction of the framework of inflamed skin, and no doubt thereby suggested the idea of a honeycomb, whilst themuco-purulent secretion might in like manner be compared to honey. Another feature of the disease is the elimination of the hair from the inflamed follicles, and the subsequent baldness of the affected part. Kerion is sometimes associated with tinea capitis. See TINEA TONSURANS.

ERASMUS WILSON.

**KIDNEYS, Diseases of.**—SYNON.: Fr. *Maladies des Reins*; Ger. *Nierenkrankheiten*.—The kidney is subject to a number of diseases, which will be considered in the following pages in alphabetical order. At

the outset, however, it will be convenient to present an outline of the abnormal phenomena to which these affections may give rise.

**SUMMARY OF SYMPTOMS.**—The facts upon which the diagnosis is founded in diseases of the kidneys are of three classes, namely: A. Abnormal local conditions. B. Abnormalities of the urinary secretion. C. Abnormalities in other parts of the system, secondary to the local phenomena.

**A. Abnormal local conditions.**—These may be:—

*a. Subjective.*—The patient may experience pain or uneasiness in the region of the kidney on one or both sides; and abnormal sensations may be also referred to the ureter, the bladder, or the urethra. The pain may be influenced by attitude or by exertion; and be either constant, paroxysmal, or periodic. It may be aggravated, relieved, or unaffected by pressure.

*b. Objective.*—Examination of the abdomen and pelvis, by means of palpation and percussion, may reveal the presence of a tumour connected with the kidney. The tumour may be solid or fluid; uniform or lobulated. Tumours of the kidney may generally be made out by palpation, and especially by tilting with the one hand the mass forwards from the lumbar region, upon the fingers of the other hand applied in front. In some cases the absence of the kidney from its normal position may be ascertained by percussion in the lumbar region. By careful observation of the relative form of the two sides at the back and in front, either the presence of tumour, or the absence of the organ, may be rendered distinct.

**B. Abnormalities of the urinary secretion.**—*a.* The urine may be altered in quantity. It may be increased, as in waxy degeneration of the kidney, or in advanced stages of cirrhotic and inflammatory Bright's disease; or diminished, either from obstruction to its escape, or from failure of secretion. The conditions leading to obstruction to outflow are certain diseases of the urethra, prostate, bladder, or ureters; the last named inducing suppression only where both the ducts are simultaneously occluded, or where, one kidney having been previously destroyed, the ureter of the other side subsequently becomes affected. The impaction of calculi, and the pressure of new-formations, are the chief causes of these obstructions. The conditions leading to diminution or failure of secretion are the pyrexial state; obstruction of uriniferous tubules, as by inflammatory products; long-standing passive

congestion, as in cardiac disease; and, probably, some forms of altered innervation.

*b.* The urine may be altered in *colour*, as from the presence of blood, pus, bile, purpurin; or of substances introduced into the system, such as logwood, rhubarb, senna, tar, and carbohc acid.

*c.* The *specific gravity* may be altered, being much raised when the proportion of water is small, or when an excessive amount of sugar or of urea is being eliminated, or when a large proportion of blood or of albumin is present. Diminution occurs whenever the proportion of water is excessive, or the elimination of urea deficient. It is thus an important feature of renal disease. In determining the specific gravity it is important to remember that, when the specimen has stood for some time, it may vary in different parts of the same column of fluid; that it varies with the temperature, being lower in warm than in cold fluid; and, further, that it varies at different times of the day, in relation to the state of the digestion.

*d.* The *reaction* of urine varies from the slightly acid standard of health, by being either too acid or alkaline. The acidity may be excessive when the urine is passed, or may become increased after it has been voided, in consequence of the acid fermentation. It may be alkaline when passed, from the presence either of fixed alkali or of ammonia. The ammonia results from decomposition of urea, and this change constantly occurs in urine which has been kept and allowed to decompose.

*e.* *Albumin* is a common morbid constituent of urine, either temporary or permanent. Several albuminous bodies are met with, but the most important are serum albumin, serum globulin, and peptone. Temporary albuminuria may be artificially produced by the ingestion into the stomach, or by subcutaneous injection, of raw albumen of egg; and it sometimes results from derangement of the digestion, due to the use of indigestible articles of food or from muscular exertion. It occurs in certain blood-diseases, such as scarlet fever, erysipelas, diphtheria, and acute yellow atrophy of the liver, and is probably due to the irritation of the kidneys by the poisons proper to these maladies. It also occurs in some cases where fever is high and persistent, and is then to be explained by alteration of the condition either of the vascular walls, of the renal cells, or of the innervation of the kidneys. It also occasionally results from the use of certain drugs, such as turpentine and cantharides; certainly sometimes from nervous affections, such as exophthalmic goitre, epilepsy, and injuries to, or organic disease of, the brain. Permanent albuminuria is met with in all the forms of Bright's disease: most abundantly in the inflammatory form; to a less extent, but constantly, in the waxy; to a still less extent

(and sometimes altogether absent) in the cirrhotic variety. It also results from passive congestion of the kidneys, due to cardiac disease or other cause; as well as from suppurative nephritis and other diseases of the kidney, and from pyelitis. The ordinary albumin of the blood-serum is the form which usually appears in the urine. Now and then it is found, especially in the course of or after acute febrile diseases, that a variety of albumin occurs in the urine which is unaffected by heat and nitric acid, but which becomes coagulated by alcohol. This may either be from alteration of the substance itself, or from the presence of some material which interferes with the ordinary chemical reaction. The indications to be derived from the presence of peptones in the urine are not yet definitely ascertained. See ALBUMINURIA; ALBUMOSES; and ALBUMOSURIA.

*f.* *Urea* is diminished in quantity wherever there is destruction of the renal epithelium, as in the different forms of Bright's disease, especially the cirrhotic and inflammatory varieties.

*g.* The *physical* and *chemical* characters of the urine are often much modified by conditions other than diseases of the kidneys themselves, and abnormal ingredients may be present, such as sugar or bile, but these alterations do not come within the scope of the present article. See URINE, Morbid Conditions of.

*h.* *Deposits* are also frequently present in the urine, which are due to various causes apart from renal disease, namely, urates and uric acid, oxalate of lime, phosphates, cystin, xanthin, tyrosin, and leucin. Organic deposits are important in many affections of the kidney or its pelvis. In the first place the *epithelium* from the latter may be present in more or less abundance. *Pus* appears as a fine granular yellowish deposit, which becomes viscid and transparent on the addition of liquor potassæ. In ammoniacal urine it is sometimes found that the pus-cells have undergone this change within the bladder. Pus may be derived from the pelvis of the kidney, or from the kidney-substance itself. Deposits very similar in general appearance to pus are sometimes seen in cases of scrofulous kidney, but the microscopic appearances are different, the corpuscles being altered, and often associated with fibrous tissue. *Cancerous* deposits, showing distinct cancer-cells, are also sometimes met with. The presence of *blood* or of its *pigments* gives the urine a smoky, pinkish, or actually bloody appearance. See HÆMATURIA; MICROSCOPE IN MEDICINE; and LEUCIN.

*Tube-casts* are sometimes so numerous as to constitute a deposit quite visible to the naked eye. These casts are solid moulds of the uriniferous tubules, sometimes formed within the free lumen of the tube, but far more frequently within the basement-membrane,

thus including the more or less altered epithelium. The simplest form of tube-cast is the hyaline, a clear structureless cast. Blood-casts are common, containing distinct red corpuscles. Epithelial and desquamative casts are opaque and granular, the granular condition being due for the most part to the abundance of altered epithelium in their substance. Fatty casts are those which exhibit evidences of fatty degeneration of the epithelium. See BRIGHT'S DISEASE; CASTS; and URINE, Morbid Conditions of.

**C. Symptoms occurring in other parts of the body.**—Very important symptoms occur in connexion with renal diseases, affecting the organs of circulation and of digestion, the nervous system, and the s<sup>l</sup>; but these are for the most part associated with Bright's disease. See BRIGHT'S DISEASE; and URÆMIA.

**SPECIAL DISEASES.**—The several diseases of the kidney will now be indicated, and those will be discussed which are not described under special headings in other parts of the work; for example, Bright's Disease and Renal Calculus.

**1. Kidney, Abscess of.**—This is often used as a generic term to include any accumulation of pus in connexion with the kidney, whether in the substance of the organ, in its pelvis, or even around it. Strictly, it applies only to a collection of matter resulting from suppuration in the kidney-structure itself. See 12. Kidney, Embolism of; 22. Kidney, Inflammation of Pelvis of; 28. Kidney, Suppurative Inflammation of; PERINEPHRITIS; and SURGICAL KIDNEY.

**2. Kidney, Acute Atrophy of.**—**DEFINITION.**—This is a rare disease of the kidney, consisting in rapid exudation into the cells of the organ, followed by fatty degeneration and disintegration; caused by unknown conditions; characterised by the sudden occurrence of copious albuminuria, with very numerous tube-casts, and frequently marked uræmic symptoms; and resulting apparently invariably in death.

**ÆTIOLOGY.**—The causes of this affection are unknown, but it appears probable that it depends upon some form of blood-poison, the disease being frequently associated with acute atrophy of the liver. In most cases it would appear to follow the hepatic disease; in some it precedes it. It is much more common in the female sex than in the male; and is most frequent during pregnancy and after childbirth.

**ANATOMICAL CHARACTERS.**—There are two stages in the progress of this disease, namely, (1) that of exudative infiltration and enlargement; (2) that of disintegration and atrophy. In the first stage the organ is enlarged, not markedly congested, the capsule strips off readily, the substance is flaccid, the cortical substance is swollen, the individual tubules are enlarged and white. On section the

vessels are found mostly empty of blood, being compressed by the diseased tubules. The tubules are occupied by dense opaque material; and the individual cells are swollen and granular, their nuclei being hidden by molecular cell-contents. The tubules of the cones, as well as those of the cortical substance, are frequently affected. Many of the cells also are in a state of fatty degeneration, or broken down even at this stage. In the more advanced stage the organ is smaller than natural, and its capsule appears wrinkled. The kidney is pale and flaccid. When cut into there escapes a quantity of *débris*, often containing oil-globules, quite visible to the naked eye. The stroma is intact, and sections can easily be made with a Valentin's knife. The sections show that many of the tubules are denuded of epithelium, and that the shrinking of the organ results from this disintegration. It is easy to find different tubules and cells in various stages of transformation, some showing the early stage of cloudy swelling, others the stage of fatty transformation, and others the disintegrating, almost deliquescent condition. The friability of the cells is quite extraordinary, the weight of a thin cover-glass often sufficing to reduce them to molecular *débris*.

It will be observed that the changes, both in the organ and the individual cells, exactly correspond to those met with in acute atrophy of the liver.

**SYMPTOMS.**—The symptoms characterising this affection have not yet been very fully studied. Diminution of urine, copious albuminuria, with deposit of casts corresponding to the changes in the kidney, are probably the chief renal symptoms. A tendency to hæmorrhages, jaundice, and uræmic nervous affections, and the series of symptoms proper to acute atrophy of the liver, are also observed.

**DIAGNOSIS.**—There is probably no disease with which acute atrophy of the kidneys is liable to be confounded.

**PROGNOSIS AND TREATMENT.**—The prognosis must be unfavourable; and no treatment can be of any avail.

**3. Kidney, Albuminoid Disease of.** See BRIGHT'S DISEASE.

**4. Kidney, Anomalies of.**—The kidneys may present three kinds of anomaly, namely: in number; in form; in situation. Only the first of these will be referred to here.

**Anomalies in Number.**—Sometimes one kidney, with the corresponding ureter, is entirely absent. In such cases the organ which is present is much above the normal size. There is generally no symptom present during life, but diseases of the pelvis of the kidney or of the ureter are made more formidable in persons so affected, than in those normally developed.

Occasionally one or more supernumerary kidneys are present. See 23. Kidney, Malformations of; and 25. Kidney, Malpositions of.

5. **Kidney, Calculus in.**—See **RENAL CALCULUS**.

6. **Kidney, Cancerous Disease of.**—See 24. **Kidney, Malignant Disease of.**

7. **Kidney, Chronic Atrophy of.**—This condition of the kidney arises under a variety of circumstances, but specially as a consequence of hydronephrosis, and of the different forms of Bright's disease. In hydronephrosis the atrophy commences in the cones, and spreads to the cortical substance. In all the forms of Bright's disease it commences at the surface and spreads inwards. In hydronephrosis atrophy is a result of the pressure of the renal secretion, as it accumulates in the dilated pelvis and within the tubules. In inflammatory Bright's disease it is due to interstitial changes, and the gradual absorption of the contents of the occluded uriniferous tubules. In cirrhotic Bright's disease it is a consequence of the contraction of the hypertrophied fibrous stroma, and the consequent destruction of vessels and secreting structures. In the waxy or albuminoid form it is due to the molecular absorption of the hyaline material and altered cells which occupy the uriniferous tubules, as a consequence of the degeneration proper to the vessels.

**ANATOMICAL CHARACTERS.**—Although really atrophied, the hydronephrotic kidney appears large, and may form a mass several times the size of the normal kidney. It is lobulated on the surface, and may often be seen to be little more than a group of cysts containing watery fluid. In the earlier stages no change is observed, except flattening of the cones; in the later the cortical substance also is more or less wasted. This condition is usually seen only on one side, the other kidney being natural, or somewhat hypertrophied.

Atrophy from inflammatory Bright's disease is rarely far advanced when the fatal result occurs. Both kidneys are affected, and usually to the same extent. The capsule strips off readily. The surface presents a finely granular appearance. On section, the cortical substance is found relatively diminished. Many of the tubules are occupied by sebaceous-looking material. Many of them are diminished in size, and irregular in outline from absorption of their contents. The stroma is relatively increased, the vessels are little altered.

In the cirrhotic form the atrophy is often more advanced, and is commonly equal, or nearly so, on the two sides. The capsule is adherent, the surface granular and uneven. On section, the cortical substance appears diminished, and it often contains many cysts. On microscopic examination the fibrous stroma is found markedly increased, many of the tubules and vessels are destroyed, while the smaller arteries are thickened.

In the waxy or albuminoid form the kid-

neys have in some cases been found greatly and nearly equally diminished in size. The capsule strips off readily; the surface is finely granular. On section the cortical substance is found diminished, the degenerated Malpighian bodies remaining singularly prominent, especially towards the surface, the stroma appearing relatively somewhat increased, and the hyaline contents of the tubules in process of absorption.

**SYMPTOMS.**—No definite group of symptoms indicate the existence of atrophy. Those met with in the atrophic stages of the different affections are described under each disease.

**Partial Atrophy of the Kidney** results from embolism, new-formations, and other like causes; and is in many instances unattended by symptoms.

8. **Kidney, Cirrhotic.**—See **BRIGHT'S DISEASE**.

9. **Kidney, Congestion of.**—See 18. **Kidney, Hyperæmia of.**

10. **Kidney, Cystic Disease of.**—**DEFINITION.**—A chronic morbid state of the kidney, caused by conditions not fully ascertained; characterised in some cases by no symptoms, in others by the presence of tumours, and by symptoms resembling those of the cirrhotic form of Bright's disease; resulting in permanent change; and not amenable to treatment. The formation of cysts in the kidney may be unimportant, either from the small number of cysts, or from the co-existence of much graver disease of the kidney; but it may constitute a serious disease.

**ÆTIOLOGY.**—Nothing is known as to the causes of cystic degeneration of the kidneys. It may be congenital, or may come on during adult life. The mode of origin of the cysts appears to be from the dilatation above obstructed points in the course of the uriniferous tubules, or at their points of origin in the Malpighian bodies. Sometimes the cysts are new-formations in connexion with epithelium; and sometimes they arise from the fibrous stroma of the organ.

**ANATOMICAL CHARACTERS.**—Renal cysts vary greatly in size, from minute, almost microscopic, cavities, to spaces capable of holding several pints of fluid. The true cystic kidney is large; its surface is uneven, and in colour it resembles a piece of conglomerate. The capsule strips off, but often with some difficulty. On section its substance is found replaced by multitudes of cysts, scarcely any proper tissue remaining. The contents may be watery, or contain urinary constituents, or are tinged with blood, or sometimes are gelatinous or colloid.

**SYMPTOMS.**—In many cases cystic disease of the kidneys is unattended by any symptoms; and even when the disease is extensive and severe there are, as a rule, no constitutional symptoms until the case draws near its termination. Among local signs the most

important is enlargement of the organs, which may sometimes be made out by means of palpation and percussion, in cases which are advanced, and in emaciated subjects. Both organs are generally equally enlarged. The urine is secreted in natural or in excessive quantity; its specific gravity is low; and it contains albumin, and sometimes blood. The termination of these cases is not infrequently abrupt, with uræmic convulsions and coma.

**DIAGNOSIS.**—The points which are of importance are the presence of bilateral tumour; with copious discharge of urine of low specific gravity, or containing albumin.

**PROGNOSIS.**—The prognosis is always unfavourable.

**TREATMENT.**—Treatment can be directed only to the relief of symptoms.

**11. Kidney, Dropsy of.**—This is a synonym for hydronephrosis, in which urine, more or less altered, accumulates in the renal pelvis, as the result of obstruction of the ureter. See **HYDRONEPHROSIS**.

**12. Kidney, Embolism of.**—The impaction of emboli in the branches of the renal arteries gives rise to various lesions, of which the most common is the hæmorrhagic infarction; next to this inflammation and secondary abscesses; and more rarely gangrenous inflammation. These affections often do not manifest themselves during life by any distinct symptoms; or their existence may be revealed by sudden albuminuria and hæmaturia, and sometimes by general constitutional disturbance and local pain.

**ÆTIOLOGY.**—The chief cause of renal embolism is disease of the valves of the heart. The emboli may be composed of coagulated fibrin; or of fragments of the tissue of the valve, which have been separated by ulceration. More rarely embolism is caused by fibrin which has coagulated between the meshes of the fleshy columns of the heart, or by coagula which have formed on the roughened inner coat of arteries. Among the remoter causes are those of endocarditis and endarteritis. This, in so far, corresponds with the name which Rayer applied to these infarctions—'rheumatismal nephritis.'

**ANATOMICAL CHARACTERS.**—(a) *Of hæmorrhagic infarction.*—This may, for convenience, be described as passing through three stages: (1) that of red consolidation; (2) that of fawn-coloured transformation; and (3) that of absorption or atrophy. The form of the masses is usually conical, or, as seen on section, wedge-shaped, the base being towards the surface. At first a patch is deeply congested, and presents a dark red colour. On microscopic examination the vessels are found congested, many ruptured; and blood is extravasated into the tubules. In the second stage this redness has passed away; a buff or fawn-coloured mass represents the red patch of the earlier stage. On microscopic examination the cells of the tubules

are found to be destroyed, and within them, as well as in the stroma, blood-pigment may be found. In the third stage there is commonly a depression of the surface of the organ; and, on section, what had been the conical patch is represented by a fibrous cicatrix.

(b) *Of abscesses.*—In certain cases, especially in the course of pyæmia, emboli lead to the formation of abscesses. Such abscesses will be found throughout the kidney, and may be traced in various stages, which might be described as red consolidation, ashy-grey consolidation, and suppuration. In this condition the clot will not be found at the apex of the cone of disease, but imbedded within it. Between the simple infarction and the abscess there is an intermediate state—a certain degree of suppuration occurring at the margin of the affected area.

(c) *Of gangrenous patches.*—In consequence of the impaction of an embolus, though very rarely, gangrene of the affected district occurs, with more or less suppuration.

**SYMPTOMS.**—The symptoms of the embolic infarction in the kidney are often very indistinct, but the condition may sometimes be diagnosed. For example, if in a case of valvular disease of the heart or extensive calcareous affection of the arteries, sudden albuminuria or hæmaturia, with some degree of fever, and pain in the region of the kidneys, occurs, there is every reason to conclude that an embolus has been impacted. This condition is rarely one of importance in the case, for much graver maladies co-exist with it. The occurrence of embolic abscess in the kidney may sometimes be surmised when sudden albuminuria or hæmaturia becomes superadded to the other symptoms of pyæmia. Gangrene of the kidney or a portion of it is not likely to be capable of diagnosis during life.

**DIAGNOSIS.**—The diagnosis of renal infarction turns upon the points above referred to. It is important to distinguish it from Bright's disease, and from passive congestion. From the former it is distinguished by the suddenness of the onset, the shortness of its duration, the absence of renal dropsy, and the presence of cardiac or vascular disease; from the latter by the suddenness of its development, and by the absence of signs of stasis in other organs.

**PROGNOSIS.**—The prognosis in cases of infarction is favourable so far as the kidneys and their functions are concerned, but unfavourable in this respect, that there is a tendency to the impaction of emboli in other more important parts, particularly in the brain.

**TREATMENT.**—No special or particular treatment can be directed to this condition.

**13. Kidney, Fatty Disease of.**—**DEFINITION.**—A chronic affection of the kidney, consisting in simple fatty degeneration

of, or infiltration into, the renal epithelium, without inflammation; characterised by no symptoms as yet clearly ascertained.

**ÆTIOLOGY.**—Fatty kidney results in some cases from long-continued exhausting disorders; from senile marasmus; from starvation; from poisoning with phosphorus; and perhaps from excessive indulgence in fatty food.

**ANATOMICAL CHARACTERS.**—The kidneys are of about the natural size, their surface is smooth, and the capsule strips off readily. There is no congestion, and scarcely any stellate veins are visible. The organ is more soft and flexible than natural, and the surface is mottled with numerous deposits of sebaceous-looking material. On section the relative size of the cortical substance and the cones is seen to be preserved; and beyond a general pallor there is no change except the abundant deposition of sebaceous-looking material, mostly in the tubules of the cortical substance, but also in those of the cones.

On examining a section with a low power of the microscope, the characteristic fatty opacity is well-marked, and by careful scrutiny it may be generally made out that the fatty material is not in the canal of the tubule, but within the epithelial cells. The Malpighian bodies, the vessels, and the stroma, under a higher power, appear natural; and, in transverse section of the tubules, a clear lumen may be made out. It will thus be observed that there is no inflammatory desquamation of the cells, nor exudation filling up the lumen of the tube.

**SYMPTOMS.**—Little is known of the clinical features of this affection. But the quantity of the urine appears to be diminished. There is certainly no albuminuria, and apparently no other important change in its composition. Dr. Lang, of Dorpat, has shown that a little free oil is sometimes found in the urine.

**DIAGNOSIS.**—It is scarcely possible to diagnose this affection with certainty; but diminution of the urine, without albuminuria, and with the presence of oil in the urine, especially if associated with the signs of fatty liver, and with any of the known causes of fatty degeneration, may lead to the establishment of the diagnosis.

**PROGNOSIS.**—The renal affection is not generally an important element in the prognosis, other conditions of more importance being present.

**TREATMENT.**—General tonic treatment, and the removal of the cause when known, are the only indications.

**14. Kidney, Gouty.**—This is a form of contracted granular kidney, occurring in gouty subjects, and attended with the deposit of urates in the renal tubules. See BRIGHT'S DISEASE; and GOUT.

**15. Kidney, Granular.**—A synonym for a chronic form of Bright's disease, where the

kidney presents a granular appearance. See BRIGHT'S DISEASE.

**16. Kidney, Hæmorrhage in connexion with.**—Blood may escape into the substance of the kidney, as the result of embolism or injury; into the tubules, giving rise to blood-casts; or into the renal pelvis, especially from injury to the mucous lining by calculi. The only diagnostic indication of this event is the presence of blood in the urine, intimately mixed with it, or sometimes in clots. A coagulum of blood may block up the ureter. See HÆMATURIA; and HEMOGLOBINURIA.

**17. Kidney, Hydatid-Disease of.**—**DEFINITION.**—A chronic parasitic disease of the kidney, caused by the reception into the system of the ova of the *Tænia echinococcus*, and the development in the kidney of the corresponding cystic form; consisting in the formation of hydatid cysts—'echinococcus hominis'—in the substance of the organ; characterised in some cases by no symptoms, in others by renal tumour, or by the discharge of cysts with the urine, after symptoms resembling those of renal calculus; and resulting sometimes in recovery, sometimes in death, either by perforation into the lung, intestine, or other part, or by suppuration.

**ÆTIOLOGY.**—See ENTOZOA.

**ANATOMICAL CHARACTERS.**—The affected organ is enlarged, sometimes greatly. It is often connected by adhesions to neighbouring parts. A globular tumour projects from the surface, and extends into the substance of the kidney, inducing corresponding atrophy. The cyst has an outer covering of fibrous tissue derived from the organ; and an inner coat—the cyst proper, which may be barren, that is, devoid of daughter-cysts, or may contain within it numerous smaller cysts and processes growing inwards from the walls, containing scolices which may give origin to the corresponding tape-worm. In either case the cyst-wall is somewhat tensely expanded by a clear liquid, rich in chloride of sodium. The cyst enlarges gradually, and may burst in various directions, but most frequently into the pelvis of the kidney, or into the lungs and bronchi. Sometimes suppuration of the cyst occurs, and accordingly one finds on *post-mortem* examination the remains of a shrivelled and sunken cyst, with caseated contents, in which are imbedded remains of daughter-cysts and hooklets from the scolices.

**SYMPTOMS.**—The course of hydatid-disease of the kidney is always chronic. It may be prolonged for many years. The advance is insidious. Attention is sometimes drawn first to the presence of a tumour, sometimes to the evidences of its rupture. When rupture takes place into the pelvis of the kidney, daughter-cysts passing along the ureter give rise to symptoms resembling those of renal colic; but the discharge of the cysts, and the results of the microscopic examination, reveal

the true cause of the irritation. After such a discharge the cyst may atrophy; sometimes it happens that a second or even a third discharge occurs after a shorter or longer interval. When the discharge is by the lung, pain and cough occur, due to irritation of the pleura; then the expulsion of the hydatids takes place; sometimes this also results quite favourably. The special features of a hydatid tumour are its globular form and its elasticity. When suppuration occurs, fever supervenes, attended with local pain.

**DIAGNOSIS.**—The diagnosis of hydatid of the kidney depends upon the presence of a tumour of a special kind; and is made certain by the discharge of cysts or hooklets.

**PROGNOSIS.**—The prognosis is always doubtful.

**TREATMENT.**—Medicine is of no avail. The best treatment is the removal of the fluid contents of the cyst by aspiration. When such removal is successfully effected, the parasites in many instances die, and the cyst shrivels up.

### 18. Kidney, Hyperæmia of.

**DEFINITION.**—An acute or chronic affection of the kidney, consisting in active or passive congestion of its vessels, with secondary changes; characterised by the appearance of albumin, and sometimes of blood and of hyaline tube-casts in the urine, the quantity of urine being generally diminished, and its specific gravity natural; resulting in recovery if the cause be removed, but in the passive form commonly continuing or recurring till the fatal result is induced, partly by the original, and partly by other causes.

**ÆTIOLOGY.**—*Active* congestion—that is, congestion due to increased influx of arterial blood—may be caused by inflammation; by various blood-poisons, such as those of scarlet fever, measles, typhus; and by some medicinal substances, such as cantharides, turpentine, cubeb; also probably by agencies which paralyse the muscular fibres of the small arteries—as is sometimes seen in the course of exophthalmic goitre, or which increase the blood-pressure in the renal arteries. *Passive* congestion, which implies congestion due to hindrance to the efflux of venous blood from the organ, may be caused by any obstruction to the circulation. It is most commonly met with in cases of cardiac disease where the right chambers of the heart are dilated. It also results from such diseases of the lungs as are followed by dilatation of the right side of the heart—for example, emphysema. It also sometimes arises, though much more rarely, from obstruction in the course of the inferior vena cava, or in the renal veins, as from pressure of aneurysmal or other tumours, or from the formation of a thrombus.

**ANATOMICAL CHARACTERS.**—In the *active* form of renal hyperæmia, the anatomical changes are probably less marked after death

than during life. The kidneys are generally of fully the normal size; the capsule strips off readily; and the surface is smooth. On section the vessels are found congested; the Malpighian bodies frequently standing out prominently, being distended with blood. The vessels of the cones are also overfilled. More or less evidence of inflammatory change is to be found in the tubules, the epithelium being granular and opaque, and the lumen of the tubules, especially those of the cones, being filled up with coagulated fibrin. Blood is sometimes found extravasated into the convoluted tubules. The stroma is unaltered. Sometimes there is congestion of, or even extravasation into, the mucous membrane of the pelvis of the kidney and of the ureter. In *passive* congestion the anatomical changes are more marked, and vary with the duration and intensity of the affection. In the slighter forms, or in those of short continuance, the kidney is of fully the natural size; its capsule strips off readily; the surface of the organ is smooth; and there is evidence of congestion. On section, the congestion is seen to occupy the veins and the Malpighian bodies; sometimes there are evidences of extravasation of blood; and sometimes fibrinous coagula are found in the tubules.

In the more chronic forms, although the capsule strips off readily, the surface is somewhat uneven; congestion is still marked on the surface, but scarcely so distinctly as in the earlier stage. On section the organ feels firmer than natural; it is in a condition which may be best described by the term 'induration.' Its small veins and Malpighian bodies are dilated and full of blood; the fibrous stroma is relatively increased, especially towards the surface of the organ; some of the tubules are wasted, some are blocked up with exuded material, and some exhibit evidence of disintegration and fatty degeneration of the epithelium. The condition of the stroma thus approaches that of cirrhosis of the kidney; the condition of the tubules approaches that of inflammation. There is no definite boundary line between the conditions anatomically; still the combination of the changes confirms the inference which must be drawn from the study of the clinical history, that these hyperæmic changes, though approaching to, are not identical with, the processes properly included under the term 'Bright's disease.'

**SYMPTOMS.**—The symptoms of *active* congestion are the presence of albumin in the urine, occasionally accompanied by hyaline tube-casts, and sometimes by blood in greater or less quantity. It occurs commonly as a transient, or occasionally as a recurring condition; and, unless when it betokens a commencing inflammatory action, is rarely of much practical importance.

In *passive* congestion albuminuria is again the leading symptom; the urine is generally

somewhat reduced in quantity, of about normal specific gravity, often of acid reaction, depositing urates. The amount of urea is little below the normal. Tube-casts are usually scanty, and may be wanting; when present they are hyaline or sometimes bloody, and occasionally contain altered epithelium.

**DIAGNOSIS.**—The question relating to diagnosis, which is of most practical importance, lies between hyperæmia and inflammatory Bright's disease. In making this distinction, the points to which we have to attend are the general condition of the patient, in respect to the possible causes of such an affection, the presence of cardiac or pulmonary disease, or of venous obstruction. In congestive affections the urine is scanty, as it often is in Bright's disease, but it is of high colour, of natural specific gravity, and rarely contains blood, renal epithelium, or tube-casts.

**PROGNOSIS.**—The prognosis depends entirely upon the conditions inducing the congestion. In the active form it usually rapidly subsides; in the passive form, it is persistent, or at best, if temporarily got rid of, is almost sure to recur.

**TREATMENT.**—When the renal congestion is very intense, dry cupping, local blood-letting, the hot air or the warm vapour bath, or warm applications over the kidneys, may be indicated; but the treatment is mostly that of the diseases which are inducing the congestion. Thus, in the case of cardiac disease digitalis and iron, in the case of pulmonary disease digitalis with squill, and, if necessary, a little blue pill or carbonate of ammonium, are indicated. The general management should be that proper to the obstructive disease which has induced the congestion.

**19. Kidney, Hypertrophy of.**—True hypertrophy, that is to say, increase of all the elements, or of the essential elements of the kidney, occurs only in one organ as a rule, and that by way of compensation for atrophy of the other.

Simple increase of bulk of the kidneys often results to a certain extent from congestion, inflammation, the various forms of Bright's disease, new-formations, and accumulation of the secretion.

**ANATOMICAL CHARACTERS.**—These present no peculiarity beyond the enlargement, the organ weighing sometimes eight or nine ounces, the renal artery and vein being proportionately enlarged, with a corresponding coarseness of structure.

**SYMPTOMS.**—Hypertrophy of the kidney is without symptoms, but it might so happen that the enlargement of the organ could be detected on physical examination.

**20. Kidney, Infarction in.**—See 12. Kidney, Embolism of.

**21. Kidney, Inflammations of.**—Inflammation of the kidneys and their pelvis presents many varieties. The kidneys them-

selves exhibit, first, tubular inflammation, acute or chronic; second, inflammation of the stroma, acute or chronic; third, suppurative inflammation of the substance of the organ, septic or non-septic. The renal pelvis is also liable to acute or chronic inflammation. It will serve no useful purpose to discuss these in a general article, and therefore the reader is referred to the several special articles. See BRIGHT'S DISEASE; 28. Kidney, Suppurative Inflammation of; and 22. Kidney, Inflammation of Pelvis of.

**22. Kidney, Inflammation of Pelvis of.**—SYNON.: Pyelitis; Fr. *Pyélite*; Ger. *Nierenbeckenentzündung*.

**DEFINITION.**—An acute or chronic disease of the pelvis of the kidney, caused by extension of inflammation or irritation from the neighbouring parts, by renal calculus, by cold, or by blood-poisoning; consisting in inflammation of the mucous membrane, frequently associated with changes in the other coats and in neighbouring parts; characterised by the presence of mucus or pus in the secretion, with local pain, and more or less constitutional disturbance; sometimes resulting in recovery, sometimes in long-continued illness, and occasionally in death.

**ÆTIOLOGY.**—Pyelitis is caused by:—(1) extension of inflammation from neighbouring parts of the urinary tract—sometimes from the kidneys, sometimes from the bladder; (2) stagnation and decomposition of the urine in the renal pelvis; (3) mechanical irritation, as from calculi and gravel; (4) exposure to cold; (5) certain blood-poisons, such as those of septicæmia, diphtheria, and typhus; and (6) the action of certain other poisons.

**ANATOMICAL CHARACTERS.**—Three types of pyelitis may be recognised, namely:—(a) the acute; (b) the chronic; and (c) the calculous.

(a) *Acute.*—The mucous membrane is congested, and its surface coated with mucus, sometimes with a bloody, sometimes with a diphtheritic, layer. The membrane itself may be more or less extensively destroyed; and the characteristic tailed epithelial cells of the pelvis of the kidney may be thrown off in large numbers. Besides these cells, the cavity contains mucus or muco-purulent material in quantity.

(b) *Chronic.*—In this condition the mucous membrane is much thickened, often of a slate-grey colour, with ecchymoses, and sometimes with ulcerative abrasions of the surface. The other coats of the pelvis and the ureter may also be distinctly thickened, and the lumen of the ureter may be more or less narrowed. The cavity contains purulent material, with *débris* of broken-down mucous membrane; and sometimes, the ureter being obstructed, great accumulation of pus takes place, so as to expand the pelvis and lead to partial atrophy of the kidney (*pyonephrosis*).

(c) *Calculous.*—In this form the mucous

membrane may present either of the conditions above described, but one always finds mingled with the other contents calculi of greater or less size.

**SYMPTOMS.**—The symptoms of *acute pyelitis* may be either well-defined or masked. There may be uneasiness or acute pain in the loins and along the line of the ureter; sometimes distinct rigors, with other febrile symptoms, occur; and the urine is cloudy, depositing mucus or muco-purulent material, or sometimes blood. The most characteristic feature is the presence in the urine of the angular tailed cells which line the pelvis of the kidney. The condition may gradually subside, or may become chronic; or in rare cases, and where important complications exist, it may prove fatal.

In *chronic pyelitis* there is often an aching feeling, or well-defined pain, in the region of the ureters. There is constitutional disturbance, debility, fever, hectic; the urine is opaque, and deposits pus—generally grey, sometimes tinged with blood. This condition may go on for long periods, sometimes terminating in recovery, but often persisting and proving fatal by exhaustion, by extension to the kidney-substance or to the surrounding cellular tissue, or by concomitant complications.

The *calculous* differs from the other forms in respect of its cause; and in being attended by more pain, by more tendency to hæmorrhage, and sometimes by the presence of crystals, gravel, or calculi in the deposit.

If the escape of the pus should be prevented in any of the three forms of pyelitis, owing to obstruction of the ureter, and pyonephrosis result, a fulness or fluctuating tumour may be detected in the renal region, and this in some instances subsides at intervals, with a copious discharge of pus in the urine.

**DIAGNOSIS.**—From cystitis, pyelitis is distinguished by the absence of vesical pain, and of frequent calls to micturition; and by the presence of the lumbar uneasiness, and the more intimate admixture of the foreign materials with the secretion. From renal inflammation it is distinguished by the absence of tube-casts; the seat of the pain; and the presence of the characteristic cells of the renal pelvis. From strumous kidney it is sometimes almost impossible to differentiate simple pyelitis. Indeed, the two conditions are not infrequently associated together; but the presence of other evidences of strumous disease, the enlargement of one or both kidneys, and the discharge of copious *débris*, in addition to the pus, often suffice to distinguish the one from the other.

**PROGNOSIS.**—In the slighter and acute forms of pyelitis, the prognosis is generally favourable. In the chronic variety it must always be guarded, the amount of danger being determined in some measure by the

cause, the constitutional conditions, and the complications.

**TREATMENT.**—The first essential is that the patient should have rest, and that the urinary secretion should be copious and bland. In order to secure these ends, a diet largely composed of milk and simple diluents, or in some cases exclusively of milk, and the avoidance of stimulating foods and drinks, are to be insisted on. As to medicine, if the urine be excessively acid, alkalis should be administered; if it be alkaline, mineral acids, or benzoic acid, or a benzoate should be given. Various remedies which appear to diminish irritation, such as *uva ursi*, *pareira brava*, *buchu*, *triticum repens*, *copaiva*, and *sandalwood oil*, ought to be employed. In the more acute cases the application of poultices to the loins, and the internal administration of henbane or opium, or morphine hypodermically, are to be recommended.

In the chronic forms of pyelitis a similar line of treatment should be perseveringly followed; and in cases which owe their origin to the presence of calculi, the remedies appropriate to the diathetic condition should be employed. Astringents may possibly be useful in checking too copious a discharge of pus.

In *calculous pyelitis* our first effort must be to obtain the removal of the calculus by a copious flow of urine, and to prevent the formation of others by correcting the faults of the system to which their formation is due. But if we are unable to get rid of them, surgical interference is fully warranted, and in many cases this suffices at once to relieve the accumulation of pus, and get rid of the calculi.

**23. Kidney, Malformations of.**—The commonest malformation of the kidneys is *lobulation*, which is a relic of the fetal condition. Next comes the undue development of one organ. Sometimes there are two pelves belonging to each kidney, or two ureters. A not very rare anomaly is the *horse-shoe kidney*, which consists simply in the union of the two kidneys, by a band of renal tissue, at either end, usually the lower. This abnormality is often attended by anomalies in the arrangement of the ureters and vessels. A very rare condition is that in which there is a *central union* between the two organs, owing to the development of supplementary renal structure opposite their pelves. None of these malformations lead to any important symptoms, except by pressure upon the duct or vessels, under superadded abnormal conditions of the organs themselves, or of neighbouring parts.

**24. Kidney, Malignant Disease of.**—**DEFINITION.**—A chronic disease of the kidney, caused by the circumstances which induce cancer elsewhere; consisting in the formation of nodules of cancer, or the infiltration of the organ with the new-formation; charac-

terised by a renal tumour, cachexia, and frequently by alteration of the urine; and resulting in death.

**ÆTIOLOGY.**—*Primary* renal cancer arises from causes not yet ascertained. It occurs at two epochs of life—namely, in early childhood and in adult age. Children under four years appear specially liable. The male sex is more frequently affected than the female; the right kidney more commonly than the left.

*Secondary* cancer of the kidney is most frequently associated with carcinoma of the liver, the stomach, the mamma, the testicle, or the uterus; sometimes of the supra-renal bodies, or the mesenteric glands.

**ANATOMICAL CHARACTERS.**—All the varieties of cancer have been met with in the kidney, but the medullary is by far the most common. It is sometimes primary, sometimes secondary. The *primary* affects usually one kidney, most commonly the right; the organ is often much enlarged, weighing sometimes as much as sixteen or seventeen pounds, and this even in young children. In ten children Sir William Roberts found the average weight  $8\frac{3}{4}$  lbs.; in ten adults he found it  $9\frac{3}{4}$  lbs. Such large tumours occupy a great part of the abdomen, and push the colon forward. The cancer is in some cases scattered in separate nodules; in others it is infiltrated through the mass. It commences always in the cortical substance, and is developed from the fibrous stroma. In the scattered cases the remaining portions of the kidney are quite sound. The cancer may involve the sub-mucous tissue of the mucous membrane of the pelvis, the ureters, and the veins. The lymphatic vessels and glands also become secondarily affected. Sometimes it affects the peritoneum and colon, and it has even involved the skin.

When the renal affection is secondary, it usually involves both organs, and rarely leads to such enlargement as is seen in the primary disease. It occurs in the form of numerous nodules, developed in the stroma or along the vessels. The remaining renal tissue is commonly healthy, but it may be inflamed or otherwise altered.

*Kidney, Sarcoma of.*—It is probable that a considerable proportion of the cases from which the description of carcinoma of the kidneys is drawn was really referable to one or other of the varieties of sarcoma—either the more common alveolar form, or the rarer one with striped muscular fibres—*rhabdomyoma*. At the present time it is not possible satisfactorily to mark off a definite clinical history of this group of new-formations.

**SYMPTOMS.**—The symptoms of primary cancer generally become quite distinct when the disease advances, but in the earlier stages they are very indistinct. The urine itself is, as a rule, natural in quantity, of acid reaction, normal specific gravity and colour; but from

time to time blood appears, its amount varying from a mere trace to a very serious hæmorrhage. Sometimes the blood is in clots, and this bleeding may be the earliest symptom. It occasionally happens that carcinomatous elements may be discovered in the urinary deposit, but it is very difficult to identify them.

Examination of the abdomen reveals the presence of a tumour, occupying and extending from the region of the kidney. The tumour is generally nodulated, of tolerably firm consistence, and dull on percussion. The colon lies in front of the mass, which is capable of being tilted forward by pressure on the renal region. When the left kidney is affected, the spleen is displaced upwards. As a rule, there is persistent constipation, and some pain; together with the general symptoms of the carcinomatous cachexia.

**DIAGNOSIS.**—Carcinoma of the *left* kidney may be confounded with enlargement of the spleen; with perinephric abscess; perhaps sometimes with disease of the mesenteric glands; or with obstruction of the colon, and retention of fæces. From splenic tumour it is distinguished by its lower position, by the absence of the splenic notch, the normal condition of the blood, the presence of blood in the urine, the nodulated character of the tumour itself, and the position of the colon in relation to it. From perinephric abscess cancer of the kidney is distinguished by the absence of fever, and of fluctuation; as well as by the less rapid advance of the disease. From tumours of the mesenteric glands renal cancer is distinguished by its situation, being more towards the side and the lumbar region. The mass also is less nodular than in mesenteric growths, which are composed of groups of glands. From carcinoma of the intestine, with accumulation of fæces above it, it is distinguished by the position of the mass, and by its characters on palpation; as well as by absence of the signs proper to the intestinal disease.

Carcinoma of the *right* kidney may be confounded with tumour of the liver, especially in children; but the presence of a space of clear percussion, more or less extensive, between the liver and the tumour, should remove all doubt. If the diseased kidney touches the liver, reliance must be placed on the symptoms proper to renal or to hepatic disease respectively.

Cancer of the kidney is to be distinguished from tumour of the ovary by its more fixed position, and the history of its growth.

**PROGNOSIS.**—The prognosis is in all cases unfavourable; the duration varies with the form of cancer.

**TREATMENT.**—Treatment is, of course, merely palliative—morphine, belladonna, henbane, applied externally or injected subcutaneously, to relieve pain; ergotin and other preparations of ergot, acetate of lead, and

gallic acid to check hæmorrhage; and iron as an astringent and blood-tonic. The bowels require careful attention; and sometimes there may be so much ascites as to warrant tapping. Excision of the affected organ may be attempted in some cases.

**25. Kidney, Malpositions of.**—The kidney may be congenitally displaced, but the important anomaly coming under this head is the *movable kidney*, which demands special consideration.

**DEFINITION.**—The movable kidney is a condition especially affecting women; consisting in the undue mobility of one, or rarely of both kidneys; characterised in some cases by no symptoms, in others by uneasiness or pain, with general nervous disturbance, and by the presence of a tender reniform tumour, with clear note on percussion in the renal region of the affected side; resulting, as a rule, in frequent recurrence of the symptoms, without danger to life.

**ÆTIOLOGY.**—Movable kidney is more common in the female sex, and especially in those who have passed through many pregnancies, but it is not exclusively associated with women, for it occurs (although rarely) in males, and also in children. The right kidney is much more frequently affected—65 out of 91 cases (Ebstein). Its occurrence is probably mainly due to laxity of the abdominal parietes, and unusual length, or irregular distribution, of the renal vessels.

**ANATOMICAL CHARACTERS.**—The kidney is not necessarily changed in its structure, but its position may be altered in any direction.

**SYMPTOMS.**—In many cases no symptoms occur in movable kidney. But in some cases, whenever the displacement occurs, much uneasiness or even considerable pain is experienced. The writer has known a man unable to work in consequence of the pain induced by the displacement, and losing a day's work regularly once a week or once a fortnight. The sensations are generally rather of the nature of vague uneasiness than of actual pain, except when the organ is touched, and then there is pain of a peculiar and sickening kind. The urine sometimes becomes altered during the attacks, depositing mucus, and in one case in the writer's practice a little blood. On percussion over the renal region posteriorly a clear note may be elicited on the affected side, and sometimes a flattening may be made out at the part. Careful palpation reveals a tumour, of characteristic renal form; and now and then pulsation of the renal artery may be felt. The morbid condition may recur at intervals during many years; in some cases it is apparent for a time, and then absent for a very long period. It is liable to be brought on by effort, but often appears without discoverable cause. Occasionally it is found that the displaced kidney owes its position to the existence of carcinoma or other disease of the organ.

**DIAGNOSIS.**—The malady may be confounded with tumour of a malignant nature, originating either in the abdomen or in the pelvis. The diagnostic points are—the peculiar tenderness; the mobility; the occasional disappearance; the unchanging character of the tumour; and the occurrence of a clear percussion-note with flattening in the renal region of the affected side.

**PROGNOSIS.**—The prognosis is favourable.

**TREATMENT.**—The treatment should be by means of bandages or trusses to support the abdominal walls, and keep up a pressure upon the kidney, so as to retain it in its normal situation. In the case of the working man above referred to, complete immunity from the displacement was obtained by the use of a bandage with a pad so arranged as to keep up a pressure upon the organ.

**26. Kidney, Morbid Growths of.**—The only really important morbid growths of the kidney are cancer and tubercle. Syphilitic new-formations occasionally occur, but give rise to no characteristic symptoms. Growths of fibrous, fatty, bony, muscular, and glandular tissue have all been met with in a few cases. Hydatid-disease may also be mentioned.

**27. Kidney, Parasites of.**—The parasites which have been described as existing in the human kidneys are hydatids (*see* 17), *strongylus gigas*, *pentastoma denticulatum*, *bilharzia hæmatobia*; also the larval form of the *filaria sanguinis hominis*, which inhabits the lymphatic vessels, either of the kidneys themselves, or of some lower part of the urinary tract.

**TREATMENT.**—In the treatment of patients affected with *bilharzia hæmatobia*, the internal use of oil of turpentine, and of the extract of male fern, is recommended. It is stated that, when the bladder is affected, injections of iodide of potassium, twenty or thirty grains dissolved in tepid water, repeated every second or third day, have been found useful. *See* ENTOMOZOA; CHYLURIA; and 17. Kidney, Hydatid-Disease of.

**28. Kidney, Suppurative Inflammation of.**—**DEFINITION.**—An acute or sub-acute disease of the kidneys; caused by injuries, extension of disease from the bladder, and perhaps exposure; consisting in inflammation and suppuration in the kidney; characterised by constitutional disturbance, with local pain or tenderness, and various alterations of secretion; and usually resulting in death.

**ÆTIOLOGY.**—The commonest causes of this disease are renal calculus, leading to inflammation of the pelvis of the kidney; and inflammation of this part, propagated upwards from the bladder or urethra. Next in frequency is pyæmia, which induces metastatic abscesses. Comparatively rarely the inflammation is a result of embolism of the renal arteries; of injuries; and perhaps of exposure to cold.

**ANATOMICAL CHARACTERS.**—The affected organs are generally above the natural size. The capsule may strip off readily, but often this process leads to tearing of the substance, and liberation of pus. The surface is frequently discoloured in patches. The abscesses may be described as passing through several stages. There is first the stage of congestion, with exudation into the stroma of the organ; secondly, the stage of grey consolidation; and thirdly that of suppuration. Occasionally sloughing occurs. Sometimes perinephric abscess results, from perforation of the capsule. Drying up of the pus, with shrivelling of the affected area, is sometimes met with.

Microscopic examination reveals in some cases at an early stage the presence of colonies of bacteria in certain districts within the tubules, causing irritation first in them, then in the stroma, and thus inducing suppuration.

**SYMPTOMS.**—The most important clinical features of suppurative nephritis are the constitutional disturbance, accompanied by pain in the region of the kidneys, and tenderness on pressure, with scantiness of secretion; the urine being albuminous or bloody, or sometimes purulent, and depositing tubecasts.

**DIAGNOSIS.**—From pyelitis, suppurative inflammation of the kidneys is distinguished by the presence of tube-casts, and the absence of the characteristic angular cells of the calices of the pelvis. From perinephritis, it is diagnosed by the absence of distinct tumour, and by the history of the case.

**PROGNOSIS.**—The prognosis is generally grave.

**TREATMENT.**—The strength should be supported by suitable food, by tonics, and stimulants when necessary; and in some cases benefit may be derived from poulticing, fomentations, or the application of leeches. Under suitable conditions it might be desirable to open a renal abscess, and evacuate the pus.

**29. Kidney, Syphilitic Disease of.**—Syphilis may produce in the kidney, as in other organs, congestion; inflammation—either simple or gummatous, with the cicatrices and nodules resulting therefrom; and waxy or amyloid degeneration.

**ANATOMICAL CHARACTERS.**—There is scarcely ever an opportunity of studying the appearances of the syphilitic kidney in cases of congestion—probably over-filling of the vessels, with slight inflammatory conditions of the tubules, is all that would be found. The simple interstitial inflammation is characterised by thickening and swelling of the fibrous stroma, in patches here and there. Gummatous inflammation is rare; but when it does occur, it forms masses of the ordinary gummy character. Either of these conditions may lead to the formation of syphilitic

cicatrices, which may appear on the surface of the organ, or be imbedded in the cortical substance. Their formation is attended by the destruction of tubules in the affected parts.

**SYMPTOMS.**—Albuminuria, slight in amount and of temporary duration, occurring along with other syphilitic congestive affections, has appeared to the writer to indicate renal congestion. Various slight cases of inflammatory Bright's disease have appeared to be due to the syphilitic poison. The symptoms of the interstitial and gummy inflammations are not ascertained, although probably albuminuria attends them also. For the symptoms of waxy degeneration, see BRIGHT'S DISEASE.

**DIAGNOSIS.**—The diagnosis of syphilitic disease of the kidney depends upon the co-existence of renal symptoms with evidences of syphilis, while other diseases of the kidney are excluded.

**PROGNOSIS.**—This is favourable so far as danger is concerned, except in the case of severe waxy degeneration.

**TREATMENT.**—Iodide of potassium has been found to be useful in this as in other syphilitic affections, at least in the congestive and inflammatory conditions. Should it fail to give relief, the perchloride of mercury may be given in moderate doses, and continued cautiously even when albuminuria is present.

### 30. Kidney, Tuberculosis of.

**DEFINITION.**—A chronic disease of the kidneys and ureters; caused by tubercular infection, or by strumous inflammation of the structures involved; consisting in the formation of nodules of tubercle, or in strumous inflammation of the substance of the gland, and of the mucous membrane; characterised by some degree of constitutional disturbance, sometimes by renal tumour, and by various alterations of the urine, particularly deposit of caseous purulent *débris*; and resulting usually in death.

**ÆTIOLOGY.**—The direct causes of tubercular disease of the kidney are unknown. It is more common in children and young people than in those more advanced in life, but it—especially the scrofulous form—may occur later. Men are decidedly more frequently affected than women. The kidneys are rarely equally involved; the right is commonly worse than the left. One organ may be quite free from disease.

**ANATOMICAL CHARACTERS.**—Under renal tuberculosis are included both tubercle proper and strumous inflammation. Tubercle proper occurs in the form of minute miliary nodules scattered throughout the substance of the organ, as a local manifestation of a general true tuberculosis. Strumous inflammation leads to the formation of larger masses, involving either the mucous membrane of the pelvis of the kidney, or the cortical substance. When the former is its seat, it leads to a thickening of the mucous membrane,

commencing in patches which gradually extend and ultimately undergo ulceration. When the cortical substance is affected, the organ becomes enlarged; presents a markedly lobulated surface; and on section conical masses of altered tissue are found to correspond with the prominences of the lobules. Some of them are solid and cheesy; others are softened in the centre; while others are completely softened, so that on section a quantity of puriform *débris* flows out, leaving a cavity with white walls, rendered shaggy by the shreds of fibrous tissue which project from them. Sometimes scarcely any renal structure is left. Occasionally what remains shows the characters of waxy degeneration. The disease commences in the stroma of the organ; the tubules are compressed, but are rarely the seat of inflammatory changes. When the mucous membrane of the pelvis of the kidney and the ureter is affected, the membrane is thickened at certain parts, and afterwards becomes ulcerated; and the lumen is diminished, or completely choked up by granular *débris*. Frequently both the mucous membrane and the substance of the kidney are affected. It occasionally happens, when one kidney is exclusively affected, that shrinking of the gland takes place; and a putty-like material, rich in cholesterin, or perhaps even calcareous nodules are found, occupying the smooth-walled cavities produced by the disease. Tuberculosis of the ureters, prostate, vesiculæ seminales, bladder, and testicle not infrequently co-exists.

**SYMPTOMS.**—When tubercle occurs in small nodules in the kidneys it produces no symptoms, and even in the inflammatory form the constitutional symptoms are, in the earlier stages, not very well-marked; but as the disease advances, fever, passing gradually into the hectic type, is developed. The local symptoms may be negative, but there is frequently pain in the affected organ, with tenderness on pressure; and in some cases a tumour may be felt in front, or percussion may reveal an increased area of dulness in one or both renal regions. The secretion may be normal, or even sometimes excessive in quantity, when the disease is not far advanced. It may be acid or alkaline, of fair specific gravity, albuminous, and sometimes bloody. It often contains a puriform material, with *débris* of renal tissue. Sometimes there are masses of cheesy material, which are eminently characteristic, occurring in no other form of disease of the urinary tract. Tubercle bacilli are sometimes demonstrable in the deposited *débris*. Occasionally the urine becomes suppressed, and symptoms of uræmia precede the fatal termination.

**DIAGNOSIS.**—The evidences on which we rely in the diagnosis of tubercular disease of the kidney, are the presence of pyelitis,

combined with signs of tubercular disease in other parts, and above all the deposit in the urine of the characteristic fragments of cheesy tissue above described, and the occurrence of bacilli, although the failure to demonstrate them cannot be held to forbid this diagnosis.

**PROGNOSIS.**—The prognosis is very unfavourable, on account both of the local and of the constitutional conditions.

**TREATMENT.**—The treatment is merely palliative—to relieve pain or uneasiness; and to seek to improve the general health, by administering remedies which are useful in strumous affections.

**31. Kidney, Tumour of.**—Any enlargement connected with the kidney, which reveals itself on clinical examination, is regarded as a renal tumour. This may be due to mere hypertrophy of the organ; any form of cystic disease; accumulation of any fluid in the renal pelvis, or in the kidney itself; or a solid new-growth, especially malignant disease. For a description of the signs of these several conditions, the reader is referred to their respective headings in this article; and to other appropriate articles.

T. GRAINGER STEWART.

**KIESTEIN** (κίω, I am pregnant; and ἐσθής, a pellicle).—**SYNON.**: Fr. *Kyestéine*; Ger. *Kyestein*.—This substance was formerly believed to be peculiar to, and always present in, the urine of women in pregnancy, and it was held, therefore, to be significant of that condition. It is usually detected between the second and seventh months. It may, however, be absent during the whole period of pregnancy, as also at the beginning and at the end of pregnancy. It is also met with in the urine of non-pregnant, anæmic patients, under other conditions apart from gestation; and even in the urine of men. It therefore possesses no diagnostic value.

If urine capable of yielding kiestein be set aside in a tall glass, a cotton-wool-like flocculent precipitate becomes visible in the middle of it on the second or third day. This soon rises to the top, and an iridescent pellicle is seen forming on the surface. When this has fully formed, it begins to fall through the fluid in the form of flocculi, until the whole is deposited at the bottom as a whitish layer. Another pellicle containing triple phosphates succeeds this, and putrefactive changes proceed. Fat, mucus, crystals of triple phosphate, vibriones, and granular matter have been detected in kiestein.

ALFRED WILTSHIRE. JOHN HAROLD.

**KINÆSTHESIS** (κινέω, I move; and αἴσθησις, sensation).—The sense of movement.

This endowment is one of great importance in reference to the mode of production of voluntary movements; in reference to the

real nature of the functions performed by the so-called 'motor area' of the cerebral cortex; and also for the proper comprehension of aphasia and other speech defects.

Every time a movement is executed we receive a group of sensorial impressions occasioned by and peculiar to the particular movement. This group of kinæsthetic impressions is made up in part of impressions emanating from the muscles in action, of others emanating from the joints moved, and of others coming from tendons, fasciæ, and skin. As some such complex group of sensations or impressions occurs in association with every movement, they may best be described as 'sensations of movement,' with the understanding that they pertain to a distinct kind of sensorial endowment—a sense of movement—kinæsthesia.

These groups of sensations are registered as groups in definite portions of the cerebral cortex, and are capable of being revived in memory like other sensorial impressions. When so revived they constitute (with other related revivals in the visual centres) our 'ideas of movements.' A revival of such an idea must always constitute the first stage in the re-initiation of any voluntary movement that we may desire to perform.

Part of the group of kinæsthetic impressions belongs to what is known as touch and common sensibility, but the most important part of all is composed of what are called 'muscular sense' impressions, coming from the different muscles, and giving information as to their several degrees of tension or contraction. Thus, it is by means of kinæsthetic impressions (*a*) that we are made accurately acquainted with the position and movements of our limbs, (*b*) that we judge of weight and resistance, (*c*) that we derive information and guidance during the performance of movements, and (*d*) that we are enabled volitionally to re-initiate similar movements by the ideal recall of impressions excited by past movements.

Kinæsthetic impressions pass up through the spinal cord and the bulb; thence they pass through the posterior part of the internal capsule, and, as the writer has endeavoured to show elsewhere, they are registered in the Rolandic area of the cortex. The so-called 'motor centres' in the cortex are, in fact, rather sensory centres of kinæsthetic type, in which the effects of movements are registered, and in which the ideal recall of movements occurs as an absolutely necessary preliminary to the production of voluntary movements.

**Disorders of Kinæsthesia.**—From what has already been said, it will be clear that very different disabilities should result (A) from lesions which affect the *paths* for kinæsthetic impressions, either in the cord or in the brain; (B) from those which are produced by disease of the kinæsthetic *centres* in the

cortex. The effects of disease of the centres, again, will vary according as they are of a destructive or of an irritative nature. Not infrequently, and especially in hysterical subjects, we get a further complication (C), due to the simultaneous involvement of the cerebral paths and of the centres for kinæsthesia.

A. Where the *paths* for kinæsthetic impressions are damaged, the following defects are more or less marked:—

1. Difficulty in specifying the nature and extent of passive movements, and also as to the position in which affected limbs are left.
2. Difficulty in discriminating differences in weight and resistance when affected muscles are called into play.
3. Difficulty in accurately performing prescribed movements when the eyes are closed, *e.g.* from the abducted position of the arm touching the tip of the nose with the forefinger. These are the kinds of defect which are met with in locomotor ataxy, owing to implication of kinæsthetic paths in the cord; and such paths are sometimes affected altogether out of proportion to other sensory channels. Very similar defects are also met with in some cases of cerebral hemianæsthesia, due to lesions or functional derangements in the region of the posterior part of the internal capsule—that is, in cases where there is a loss of deep as well as of superficial sensibility in the affected half of the body. Paralysis, it should be observed, is not a symptom belonging to either of these conditions.

The fact of the existence of a loss, partial or complete, of kinæsthetic sensibility can often only be established when great care is taken; and the difficulties are greater in the way of our diagnosing such a defect in the leg than they are in the arm and hand. The kind of tests to which we must submit the patient (who must be blindfolded) for the recognition of such defects, are these: We must investigate (*a*) his ability to discriminate differences in weight; (*b*) his ability to recognise and describe the position in which a limb is left after passive movements; this may sometimes best be judged by his ability to put the opposite sound limb in a similar position, or by telling him to touch with the sound hand some named part of the affected limb; (*c*) his ability to move the affected limb quickly and accurately in some specified manner.

B. Disease affecting the kinæsthetic centres in the cortex rather than the mere paths for kinæsthetic impressions gives rise to all the symptoms produced by affection of the paths for transmission *plus* certain others, which will vary according as the lesion is of a destructive or of an irritative nature. Thus, a *destructive* lesion in some of the kinæsthetic centres will cause loss of muscular sense in the related parts, together with a motor paralysis in the same parts. On the other hand, *irritative* lesions of the same kinæsthetic

centres will cause diminution of muscular sense, together with spasms or convulsions in related parts, of the kind met with in 'Jacksonian epilepsy.'

Where this morbid condition of the kinæsthetic centres is functional rather than structural, the effects are most frequently akin to those resulting from destructive lesions of the same parts, and we then have to do with various kinds of functional or *hysterical paralysis of cerebral type*, with which there is the association of loss of muscular sense in the affected limbs.

If the kinæsthetic centres are the seats for the registration of the sensation resulting from movements and for the ideal recall of movements, and if such ideal recall is the first step in the initiation of voluntary movements, it seems clear that disease of these centres, whether structural or functional, when it renders such recall impossible, must necessarily render the performance of the corresponding movements also impossible. Similarly, in accordance with the above-mentioned views, the irritation of such centres should lead to the production of spasms or convulsions in related parts of the body.

C. In hysterical subjects, however, another grouping of symptoms is frequently met with, because in them a simultaneous affection of the cerebral paths and of the centres for kinæsthetic impressions is common. That is, we seem to have the more common affection of the cerebral paths for kinæsthetic and other sensory impressions (due to functional disabilities in the posterior part of the internal capsule) associated with a similarly degraded condition of functional activity in the kinæsthetic centres themselves, or some of them, on the affected side. The result is the co-existence of a well-marked hemianæsthesia (superficial and deep) with paralysis of one or both limbs on the affected side. In one very peculiar sub-class of these cases the paralysis only exists when the eyes are

closed; the simplest movements cannot then be made, though they are at once capable of being performed as soon as the eyes are opened.

TREATMENT.—Where kinæsthetic defects are associated with organic disease, either of the spinal cord or of the brain, the treatment to be adopted is that suitable for the diseases in question and the general condition of the patient; and, similarly, when kinæsthetic defects constitute part of the symptomatology of some functional or hysterical malady, we must address ourselves to the relief of the patient's general condition as the best means of ameliorating the special symptoms. We must not forget, however, the special potency of applications of the wire-brush for the restoration of all modes of defective sensibility in cases of functional hemianæsthesia.

H. CHARLTON BASTIAN.

**KIN-COUGH** (Dutch, *Kinkhoest*). Also *Chin-cough*.—Both these words are synonyms for whooping-cough. See WHOOPING-COUGH.

**KING'S EVIL**.—A popular name for scrofula, originating in a belief formerly held that the disease could be cured by the king's touch. See SCROFULA.

**KISSINGEN**, in Bavaria.—Common salt waters. See MINERAL WATERS.

**KLEPTOMANIA**.—Insanity characterised by an irresistible impulse to steal. See INSANITY, Varieties of: 9. Legal Insanity; and CRIME, Irresponsibility for.

**KREUZNACH**, in Germany.—Common salt waters containing iodine. See MINERAL WATERS.

**KYPHOSIS** (κνῆσις, bent).—A synonym for angular deformity of the spine. See SPINE, Diseases of.

## L

**LABIO - GLOSSO - LARYNGEAL PARALYSIS** (*labium*, a lip; *γλῶσσα*, the tongue; and *λάρυγξ*, the throat).—There are two common forms of this disease, which have to be considered separately, namely (A) a **chronic** form; and (B) a **sudden** form—the latter is often termed 'acute,' but is, as a rule, sudden in onset. (C) A true **acute** (not sudden) variety is met with in rare instances. A fourth variety (D), **pseudo-bulbar** paralysis, presents many of the symptoms of bulbar disease, but the lesion is seated in the cerebral hemispheres. It will

be convenient to describe the common chronic form first.

(A) **Chronic Labio-Glosso-Laryngeal Paralysis**.—SYNON.: Fr. *Paralyse Glosso-labio-laryngée* (Trousseau); Ger. *Progressive Bulbarparalyse* (Wachsmuth).

DEFINITION.—A progressive symmetrical paralysis of the lips and adjacent facial muscles, of the tongue, pharynx, and sometimes also of the larynx; with or without conspicuous wasting; and often associated with muscular atrophy elsewhere.

HISTORY.—First described by Du Mesnil

in 1857, and by Duchenne in 1860, the disease was made generally known by Trousseau's *Lectures*, published in 1863. By the latter writer it was termed 'labio-glossolaryngeal paralysis,' but it is now generally known by the name, first given to it by Wachsmuth in 1864, 'progressive bulbar paralysis.'

**ETIOLOGY.**—Of the causes of the affection little is known. It is a disease of later life, being almost unknown under forty. Exceptional cases have been met with in early adult life, and even in late childhood. In the latter instances several members of a family have been known to suffer, and a congenital tendency to early failure of vitality in the structures concerned may reasonably be assumed. In the common form, males are affected more frequently than females. The disease, as such, does not appear to be inherited, but in many cases there is a family history of other affections of the central nervous system. Of immediate causes, exposure to cold, mental anxiety, and defective nourishment have been supposed to exert an influence in some cases.

**ANATOMICAL CHARACTERS.**—When, as is commonly the case, there is wasting of the paralysed parts, their muscles are reduced in size, and present changes similar to those met with in the limbs in chronic spinal atrophy. Their substance is pale; fatty tissue may be in excess; and the muscular fibres often, but not always, present granular degeneration. They are frequently narrowed, and the tissue between them may be increased in quantity, and may contain pigmentary and other products of degeneration, and an increased number of nuclei. When there is no apparent wasting, this may be merely because the interstitial fat compensates for the reduction in bulk of the muscular fibres. Very rarely the structure of the muscles presents little alteration. The motor nerve-fibres are grey, translucent, and, under the microscope, degenerated. Their nuclei of origin in the medulla oblongata are also diseased. The motor cells are shrunken and atrophied; their processes are lost; and the intermediate tissue is degenerated. The hypoglossal nucleus is examined most readily, and the change in it is striking, as well as in the nucleus of the spinal accessory (palate and larynx); it may often also be seen in the nuclei of the glosso-pharyngeal, the vagus, and the 'nucleus ambiguus.' The anatomical change which underlies the affection of the lips has not yet been detected. There is still uncertainty regarding the precise origin of this part of the facial nerve. Most of its fibres diverge from the middle line, close to which the hypoglossal nucleus is situated, and some at least pass towards the lower part of the column of cells, which, above, gives origin to the motor fibres of the fifth nerve. It is certain, however, that a

large number of the fibres of the facial descend towards the level of the hypoglossal nucleus. Further, the physiological association of the movements of the tongue and lips is most close. We cannot narrow the tongue without contracting the orbicularis. Hence it is certain that the nucleus for the lower facial muscles and the hypoglossal nucleus, whether blended, contiguous, or distant, are closely connected and are alike predisposed to disease, so that this connected part of the facial suffers in the same manner as the hypoglossal. The fibres passing to these nuclei from the cerebral hemispheres may also be degenerated, and it is probable that these are alone affected in the cases in which there is no change in the muscles. When there is muscular atrophy in the limbs, a corresponding degeneration may be found in the anterior cornua of the spinal cord, often conjoined with sclerosis in the lateral columns, and in the anterior pyramids in the medulla. There is every reason to believe that, in the chronic form, the atrophic changes in the nerve-elements are the primary alteration.

In very rare cases, no morbid appearance has been observed in the medulla. Some of these may have been cases of peripheral neuritis, local and degenerative. In others, in which even the nerve-endings were normal, the degeneration of cerebro-nuclear fibres may have escaped observation, or some toxic influence may have abolished function without causing destructive degeneration.

**SYMPTOMS.**—The symptoms have the distribution indicated by the name given to the disease by Trousseau, the affected parts being the lips, tongue, throat, and larynx. They are, so to speak, arranged about the tongue as a centre. It is in this organ that the earliest symptoms commonly present themselves, as a trifling indistinctness of speech, due to an imperfect articulation of those sounds in which the tongue is most concerned—especially the dental and palatine explosive sounds, in which the tongue has to be pressed against the teeth or hard palate, *t, d, k, g* (hard), &c. The tongue can be still protruded, although perhaps not quite so far as normal. The lips then become weak, and sounds in which the lips are concerned are imperfectly articulated. The vowels *o* and *oo*, in the pronunciation of which the orbicularis contracts so as to protrude the lips, cannot be well sounded. The lips are not brought together so perfectly, or separated so promptly, as in health, and the labial explosives, *b* and *p*, become *f*, and so does the labial resonant *m*. Whistling is impossible. The lower part of the face loses its expression, the lips are habitually separated, and the saliva cannot be perfectly retained. The difficulty in articulation is soon increased by the weakness of the palate, which ceases to shut off the nasal cavity, so that a nasal resonance accompanies sounds from which

it should be absent. The paralysis of the tongue increases until the organ can no longer be protruded. Deglutition becomes impaired, partly (according to Duchenne, wholly) from the weakness of the tongue, but probably in part also from that of the constrictors of the pharynx. The soft palate ultimately hangs motionless, or, if it can be raised a little in an attempt to phonate, during the act of swallowing does not close the posterior nares, so that liquids regurgitate into the nose. Food is apt to lodge in the upper part of the pharynx, and crumbs or liquid to get into the larynx. The laryngeal muscles subsequently become weak, and the glottis cannot be closed. The vocal cords may be equally affected, or one may be paralysed in greater degree than the other. Coughing is necessarily imperfect; air is driven through the larynx, but there is no sudden opening of a previously closed glottis, and hence no explosive cough. In proportion as the glottis is paralysed, phonation is interfered with, but the ability to produce some sound is rarely altogether lost. As the disease progresses, the speech becomes almost unintelligible, being reduced to unarticulated vocal sounds. It is to be noted, however, that the habitual articulation is rarely the best possible. Words can be distinctly articulated by a deliberate effort which are scarcely at all articulated in ordinary speech. The saliva can neither be swallowed nor retained within the mouth, and is constantly dribbling over the lower lip, below which the patient has to retain a handkerchief continually. It has been thought that the quantity of saliva is increased, but the evidence of this is insufficient. The condition of the tongue varies much in different cases. In some it is large, broad, flabby, and soft to the touch throughout. In others it is conspicuously wasted, and covered with wrinkles and furrows from the shrinking. In some cases the lips retain their normal size; in others they are distinctly thinner than natural. This striking contrast between different cases (already referred to) led Duchenne to distinguish two varieties, the *atrophic* and the *paralytic*, but in some of the cases of the latter class it is merely the interstitial fat that prevents wasting from being conspicuous during life. In the affected muscles the electrical irritability is usually little changed: they still contract to the faradic current, even when the atrophy is conspicuous. Sometimes there is an indication of the reaction of degeneration, in undue readiness of contraction to the anodal (positive) closure, or this reaction may be well marked, faradic irritability being much lessened. Other muscles of the head are rarely affected. Those in the upper part of the face always escape, and the zygomatic muscles, with the elevators of the upper lip, are so little affected that their unopposed contracture

produces a peculiarly deep and characteristic naso-labial furrow. In many cases muscular atrophy in the limbs, in greater or less degree, is associated, with or without 'contracture.' So, too, in cases of ordinary muscular atrophy, commencing in the limbs, the lips, tongue, and throat are often affected towards the end of the case, in the same manner as in the primary form now under consideration.

Death, in labio-glossal paralysis, is usually the result of asthenia, due, it may be, in part, to the difficulty in deglutition, or to chronic lung disease produced by the repeated entrance of particles of food into the air-passages. Sometimes the patient dies in a paroxysm of coughing, occasioned by an ineffectual attempt to swallow liquids or saliva. There may be a fatal extension of the cerebral mischief.

DIAGNOSIS.—Before labio-glossal paralysis was well known, the difficulty in swallowing was ascribed to a chronic inflammation of the fauces, but this mistake is now scarcely possible. The symptoms have to be distinguished from those due to other diseases of the medulla, and to disease elsewhere. Many sudden lesions, in the region affected in this disease, may cause similar symptoms, but these are distinguished by their onset. Compression of the medulla may also give rise to symptoms of similar distribution, but these are commonly unilateral, or one side suffers before, and more than, the other, and they are often accompanied by a preponderant affection of the muscular part of the spinal accessory, and by great weakness, without wasting, in the limbs. In the 'pseudo-bulbar paralysis,' movements of the tongue are impaired by disease of the cerebral hemispheres. These movements are represented in the lower part of each ascending frontal convolution. When this is diseased on one side, the loss is soon compensated for by the centre in the opposite hemisphere, but a symmetrical bilateral lesion in this situation may cause complete paralysis of the tongue and other parts affected in the disease now described. Such paralysis also is acute in onset.

PROGNOSIS.—The prognosis is most grave. The disease consists in a slow degeneration of the nerve-elements, the effects of which often lead to death in about a year from the onset. Although, in some cases, a temporary arrest may be obtained, and occasionally the malady, after causing a considerable degree of disability, is arrested for several years, it is doubtful whether, in any instance of this form, considerable improvement has occurred. The prospect of prolongation of the state of arrest, if this occurs, depends very much on the amount of care the patient can secure. The older the patient, the less is the chance of arrest.

TREATMENT.—The degenerative tendency

of chronic labio-glosso-laryngeal paralysis is usually beyond the reach of remedies. Therapeutical efforts must be directed to the endeavour to retard it, by securing freedom from any cause that can be traced, especially from depressing emotion, over-work and the like, by attention to hygiene, improving the general health, and by the administration of nervine tonics, quinine, strychnine, arsenic, nitrate of silver, and the like. Strychnine is most useful when given hypodermically, one injection being given daily, in any convenient place. The nitrate of strychnine is the salt for the purpose;  $\frac{1}{100}$  gr. may be gradually increased to  $\frac{1}{10}$ . Electricity may be tried, although too often it is unsuccessful. Faradisation should be applied to the affected muscles, if they still react to it. Erb has recommended the application of the voltaic current through the throat, the positive electrode being placed on the back of the neck, the negative stroked down the side of the pharynx externally, and such a strength being employed as shall produce reflex movements of deglutition; but the use of central galvanisation is difficult to discern in the experience of most observers. Change of air is desirable in the early stage, and rest is imperative. Food must be carefully regulated; easily digestible varieties being reduced to a semi-solid condition. In the later stages, should swallowing be impossible, nourishment must be administered by an œsophageal tube, or, what is better, by a catheter introduced through the mouth or nose. Belladonna or atropine may, to some extent, check the troublesome flow of saliva.

**(B) Sudden Labio - Glosso - Laryngeal Paralysis.**—SYNON. : Acute or Apoplecticiform Bulbar Paralysis.

**DEFINITION.**—Paralysis of similar distribution to that of the chronic form, with or without conspicuous wasting, of sudden onset, and due to a vascular lesion.

**ÆTIOLOGY.**—The causes of this affection are for the most part those which lead to sudden lesions elsewhere in the brain, especially degenerations of vessels, syphilitic disease, and injuries. It is a disease chiefly of late life.

**ANATOMICAL CHARACTERS.**—Little is known of the exact condition in cases of sudden onset which have recovered with persistent labio-glossal paralysis. In cases which have died rapidly, foci of softening in the medulla have been found; and there is reason to believe that such softening, from vascular occlusion, is the most common cause of the condition. Usually there is disease of one of the vertebral arteries, and closure of one or more branches that come off from the diseased part. Sometimes the diseased vertebral is much larger than the other, and its branches have taken the larger share in the blood-supply to the nuclei. Probably a small

hæmorrhage may also give rise to it. The frequent symmetry of the symptoms in the sudden form, and their limitation to the parts which are affected in the chronic disease, show the close relation of the central structures, so that they are all affected by the one lesion. The wasting in some of the cases of this variety is very much slower than that which follows an acute lesion of motor grey matter elsewhere. It is possible that the ascending fibres from the nuclei concerned may pass up close to the middle line, in a situation in which all may be damaged by a single lesion, but the bilateral symmetry of the symptoms may also be due to the large size of the diseased vertebral, as explained above.

**SYMPTOMS.**—The onset is sudden, often with headache and giddiness, rarely with loss of consciousness. The patient suddenly finds a difficulty in swallowing and in articulation, with inability to protrude the tongue. Respiratory disturbances—cough, dyspœna, and hiccough—may be present. There may be convulsions and weakness in the limbs, sometimes with tingling, but very rarely with loss of sensibility. Many cases which present these symptoms die rapidly in the course of a few hours or days. In those which recover, a paralysis of the tongue, lips, throat, and larynx may remain, resembling that which characterises the chronic form, and there may or may not be conspicuous wasting. In many cases, however, the symptoms are incomplete, some part escapes, or the bilateral symmetry is imperfect. This we should expect from the more random character of the lesion. The course of the disease, moreover, is not progressive. The patient may remain in the same condition for a considerable time, and even exhibit marked improvement. But it is not uncommon for several slight attacks to occur at various intervals, as other branches of the artery become occluded.

**DIAGNOSIS.**—The diagnosis of the acute form of labio-glosso-laryngeal paralysis calls for little remark. It must be remembered that the symptoms may deviate from the type more than in the chronic variety, as the lesion sometimes produces irregular effects. It is chiefly liable to be confounded with the impairment of movements of the palate and tongue sometimes left after double hemiplegia, from which the history suffices to distinguish it, the two attacks of hemiplegia usually occurring at different times. The rare symmetrical affection of the surface-centres for the tongue, mentioned above, is also not simultaneous on the two sides, and it is associated with at least transient hemiplegic weakness.

**PROGNOSIS.**—If the patient recovers from the immediate effects of the lesion, the prognosis of the paralysis of the lips, tongue, and other parts is better than in the chronic

form, inasmuch as recovery of slightly damaged structures may lead to a considerable degree of restoration of power. The prognosis is also better if there is any reason to ascribe the mischief to syphilitic disease. Nevertheless, in some acute cases the paralysis remains absolute, although even in these there is not the tendency to increase which is seen in the chronic variety.

**TREATMENT.**—Any causal indication must be carefully sought for in acute labio-glossolaryngeal paralysis, and treated, especially evidence of syphilis. In other conditions the treatment is that for the lesion which is supposed to exist. Electrical treatment of the muscles is of great importance, in order to prevent, as far as possible, secondary degenerations, which are apt to occur before the partially damaged structures have recovered. The remarks regarding diet and feeding in the chronic form are equally applicable to the acute variety.

**(C) Acute Myelitic Bulbar Paralysis.**—SYNON.: Myelitis Bulbi.

In rare cases, symptoms of bulbar paralysis, as above described, come on in the acute (not sudden) manner characteristic of central inflammations, and depend on this process in the grey nuclei which are the seat of degeneration in the chronic form. Such cases, when the lesion is limited to this region, are too rare to make it necessary to do more than mention their occurrence. The process is analogous to spinal polio-myelitis, and the principles of its treatment are the same. It is more common for such an onset of bulbar symptoms to follow those of an ascending inflammation of the cord. They may also occur as the final stage in the mysterious disease, 'acute ascending paralysis,' in which it is probable that a toxic agent in the blood acts on certain parts of the nerve-centres and abolishes their function.

**(D) Pseudo-Bulbar Paralysis.**—The fact has been already mentioned that bilateral lesions of the cerebral hemispheres may cause symptoms closely resembling those produced by disease of the nuclei of the medulla. This effect is due to the fact that the muscles paralysed, being bilateral in use, are represented in both hemispheres, and can be innervated from either; hence compensation by the other side prevents paralysis from a unilateral lesion; but if disease afterwards occurs in the part effecting the compensation, paralysis results such as is caused by disease of the medulla, or rather (since there is no wasting) by degeneration of the fibres from the cortex to the bulbar nuclei, homologous with those of the pyramidal tracts. The lesions causing pseudo-bulbar paralysis may be in the cortical centres, or in the internal capsule, or in the outer parts of the lenticular nuclei, where they probably implicate the fibres from the

centres in the lower part of the 'motor' region related to the lips, tongue, &c. The lesions may be of any kind, but are generally sudden and due to vascular disease. Hemiplegia usually attends the onset, and constitutes a distinguishing feature of these cases.

W. R. GOWERS.

**LACRYMAL APPARATUS, Diseases of** (*δάκρυον*, a tear).

The lacrymal apparatus consists of the gland, with its excretory ducts; and of the puncta, the canaliculi, the lacrymal sac, and the nasal duct, through which superfluous tears are conveyed into the nose. The diseases of this apparatus are almost limited, with the exception of growths affecting the gland itself (*see* ORBIT, Diseases of), to the excessive secretion of tears, and to impediments to their escape into the nose. To excessive secretion, or to impeded outflow, the common term *epiphora* has been applied; but the great majority of cases of epiphora are due to the latter of the two causes.

**Epiphora.**—*Excessive secretion of tears* is described by authors as an affection for which it is not always possible to discover an adequate cause, and it may perhaps be sometimes due to the prolonged operation of emotional influences. In most instances, however, it is associated with some kind or degree of conjunctival irritation, and is to be regarded only as a reflex phenomenon hence arising. It is well known that any temporary or accidental irritation, such as may arise from the intrusion of a foreign body into the conjunctival sac, is apt to be followed by a copious secretion of tears, which assist in dislodging the offender; and irritations of a more chronic kind, produced by congestion or irregularity of the lining membrane of the lids, may have a similar effect.

*Impediments to the escape of the tears*, causing them to collect in the conjunctival sac, or even to flow over the cheek, may depend upon displacement of the puncta, so that these apertures are no longer applied to the conjunctival surface, from which they normally remove superabundant moisture by capillary attraction. Such displacements affect chiefly the punctum of the lower lid; and may be consequent either upon conjunctival swelling, by which the lid is pushed away from the eye, or upon paralysis or weakness of the orbicularis muscle, which allows the lid to fall by the action of gravity. In some cases epiphora will depend upon obliteration or occlusion of the puncta. The former condition is incurable; the latter may be produced by plugs of inspissated mucus, which may be removed by the careful employment of a probe.

But the most ordinary cause of obstruction is stricture of the nasal duct; in which condition the tears are arrested a little below the sac, and the sac consequently becomes over-

distended. In this condition, the sac can be seen and felt as a small lump, situated just beneath the *tendo oculi*. When pressure is made upon this lump, a fluid, consisting of tears mixed with more or less mucus or muco-pus, will regurgitate into the eye, and the lump itself will disappear. The danger in such cases is that the continued distension of the sac will in time excite inflammation of its lining membrane, leading to the formation of pus, and this to an opening upon the cheek, producing what is called a *lacrymal fistula*. Such an opening never heals until the duct is again pervious, and it is liable to undergo periodic attacks of unsightly inflammation.

**TREATMENT.**—In all cases of lacrymal hypersecretion, the first thing to be done is to search under the lids for any concealed foreign body which may be lurking there. If none be detected, examination must be made for conditions likely to be irritating; and they are to be treated, if they exist, by mild astringent or other suitable local applications. There is probably no medicine which can be said to exert any positive effect in diminishing the amount of the lacrymal secretion.

If the displacement of the lid can be cured by treatment addressed to its causes, the tears will usually return to their accustomed channel. If the displacement be incurable, as happens in some cases of paralysis of the *portio dura*, or of chronic ectropion, the patient may often be relieved by slitting open the canaliculus as far as the caruncle, so as to carry back the aperture to the secretion which it is designed to remove.

The treatment of stricture of the nasal duct can often be only palliative. The patient should acquire the habit of emptying the distended sac by finger-pressure many times a day, and of wiping away the fluid; while, to diminish the irritation of the mucous membrane, a drop of any mild astringent lotion may be applied to the inner corner of the conjunctiva two or three times a day, immediately after such pressure has been made. Perhaps the lotion most generally suitable for this purpose is a solution of acetate of lead in distilled water, of a strength not exceeding three grains to the fluid ounce.

When a radical cure is desired, the canaliculus must be slit up, and the patency of the duct restored by the passage of probes through the stricture. If fistula has already formed, or even if the sac is the seat of an abscess, no other plan is available; but for the necessary details the reader is referred to works on ophthalmic surgery.

R. BRUDENELL CARTER.

**LACTATION, Disorders of.**—SYNON.: Fr. *Troubles de la Lactation*; Ger. *Störungen der Milchdrüsen*.—The disorders of lactation are numerous. Sometimes the quantity of the lacteal secretion is excessively

small and quite inadequate for the support of the child. At other times it is so abundant that the milk will flow from one nipple as the infant is sucking the other; and when the child is removed from the breast, the secretion continues from both sides. The term *agalactia* is applied to the former, and *galactorrhœa* to the latter condition.

**1. Agalactia.**—Agalactia signifies either a total suppression of the mammary secretion, or a very scanty supply. It results generally from anæmia and general debility.

The treatment should be directed towards improving the health of the patient as much as possible, by a generous and judicious diet, and tonics, particularly those containing iron. Certain drugs have been employed as galactagogues, and, it has been said, with benefit. The leaves of the castor-oil plant, boiled, have been used as a local application—the liquid for fomentation, and the leaves as a poultice; and a strong decoction of the same plant has been given as a drink. It is doubtful, however, whether such remedies are efficacious. See GALACTAGOGUES.

**2. Galactorrhœa.**—This occurs in two forms. In the one the composition of the milk is normal, but the quantity excessive; in the other form the increase in the bulk of the secretion is due to a preponderance of the watery part of the fluid.

Remedies employed to reduce the amount of the mammary secretion are termed *galactophyga*, and the chief of these are belladonna and iodide of potassium. Belladonna is employed as an outward application, as well as administered internally. The extract rubbed up with glycerine may be spread on lint, and thus applied to the breasts, or the emplastrum belladonnæ may be used. The child should not be put to the breast too frequently. If the excessive secretion continue for any length of time, great emaciation may result; and to this condition the term *mammary diabetes* has been applied. Under such circumstances lactation should be entirely stopped as soon as possible. Strapping the breasts tightly immediately after they have been emptied is of use. Every care must be taken to avoid the formation of a mammary abscess; and if the breasts get hard and knotty a breast pump should be employed to free the tubes.

**3. Depressed Nipples.**—Depressed nipples are generally produced by the pressure of stays. If this condition be observed during pregnancy, periodic attempts should be made to draw the nipples out by means of a glass nipple-shield, to which an india-rubber tube and teat is attached.

**4. Fissures and Excoriations of the Nipples.**—These often lead to abscess, and it is said that they may sometimes end in malignant disease. To avoid the occurrence of these lesions, astringents should be applied to the nipples during pregnancy, in order to harden them. Eau-de-Cologne and water,

brandy and water, or a weak solution of tannin, may be employed for this purpose.

Sometimes an abrasion on the surface forms an ulcer or a crack at some part of the nipple, most frequently at its base, which gives rise to great pain during suckling. The remedies for these cracks are astringent applications, such as tannin, flexible collodion, a weak solution of nitrate of silver or of carbolic acid. Care should be taken to sponge these away before the infant is again put to the breast; and a nipple-shield with an india-rubber teat will be found of great service.

For abscess and other morbid conditions of the mammary gland supervening during lactation, see BREAST, Diseases of; MILK FEVER; and NIPPLE, Diseases of.

CLEMENT GODSON.

**LACTEAL VESSELS** and **GLANDS**, Diseases of.—See LYM-PHATIC SYSTEM, Diseases of; and MESEN-TERIC GLANDS, Diseases of.

**LADYSMITH**, in Natal.—See AFRICA, SOUTH.

**LAGOPHTHALMOS** (*λαγός*, a hare; and *ὀφθαλμός*, the eye).—This term is derived from an old supposition that the hare sleeps with its eyes open; and is applied to a condition in which there is inability to close one or both eyes. Lagophthalmos may be due to paralysis of the orbicularis, in which case it will be attended by falling of the lower lid, and will generally be associated with paralysis of other muscles supplied by the *portio dura* (see FACIAL PARALYSIS); to the contraction of cicatrices; to spasm of the upper eyelid (see THIRD NERVE, Diseases of); or possibly to congenital malformation of the lids. In paralytic lagophthalmos, the treatment is that of facial paralysis. Where there is contraction or deformity, each case must be considered on its own merits, with regard to the possibility of obtaining relief from a surgical operation.

R. BRUDENELL CARTER.

**LANGEN-SCHWALBACH**, in Nassau.—Chalybeate waters. See MINERAL WATERS.

**LARDACEOUS DISEASE** (*lardum*, bacon).—A synonym for albuminoid disease, which is so called from the supposed resemblance of the cut surface of an affected organ to raw bacon. See ALBUMINOID DISEASE.

**LARVALIS** (*larva*, a mask).—A term usually associated with porrigo. The thick incrustation which is sometimes seen covering the face of children affected with eczema, and constituting a hideous mask to the features, is an example of *porrigo larvalis*, or rather *eczema larvale*, as likewise is ordinary milk-crust.

**LARVATED** (*larva*, a mask).—A term applied to certain diseases, when their ordinary characters are masked or concealed; as, for example, typhoid fever. See TYPHOID FEVER.

**LARYNGEAL PHTHISIS**.—A morbid condition of the larynx, of a tubercular nature, and almost invariably associated with pulmonary phthisis. See LARYNX, Diseases of: 8. Tuberculosis; and PHTHISIS.

**LARYNGISMUS STRIDULUS** (*larynx*, the windpipe; *stridor*, a noise).—A form of obstructed breathing, attended with a peculiar stridor or crowing sound during inspiration, and dependent on spasm of the muscles of the glottis. See LARYNX, Diseases of: 6. Spasm.

**LARYNGITIS**.—Inflammation of the larynx. See LARYNX, Diseases of: 2. Inflammation.

**LARYNGOSCOPE**, The (*λάρυγξ*, the larynx; and *σκοπέω*, I look).—DEFINITION.—An instrument for illuminating the interior of the larynx and trachea, and reflecting those parts so as to present their image to the eye of the observer.

DESCRIPTION.—The apparatus for laryngoscopy consists of a small round plane mirror, set on a metal stem and fixed in a wooden handle, for introduction into the throat; and, for concentrating and reflecting the light into the throat, a concave reflector to be worn in front of the forehead, or perforated for wearing in front of the eye of the observer. With this throat-mirror and reflector, any lamp, or even a candle, is available; but brilliant illumination contributes so much to the distinctness of the image that some apparatus to condense the light, such as a bull's-eye lens, is also desirable. The oxyhydrogen or limelight gives the best illumination, but a further development of the electric light may be expected to yield equally good results.

APPLICATION.—In practising laryngoscopy, the first object is to throw a brilliant light into the pharynx.

The patient being placed beside and a little in front of the lamp, the operator seats himself opposite him, and adjusts the reflector which he is wearing, so that when the patient sits upright, with his head inclined slightly back, his mouth widely open, and the tongue put out, the light shall be concentrated on the back of the pharynx and velum palati.

The first object being thus attained, the next is to throw the light into the larynx. While the patient breathes deeply and quietly, his protruded tongue, protected by a napkin or handkerchief to prevent its slipping, is held steadily but gently forward, either by his own hand, or by the disengaged hand of the operator; and the throat-mirror, *previously*

warmed to prevent the condensation of moisture on its surface, supported like a pen, is passed into the patient's throat, and held with its back steadily pressing against and raising the soft palate and uvula, at such an angle that it throws the light into the larynx. This angle will vary according to the position of the patient and the part of the larynx which we wish especially to examine. The inclination of the mirror must then be altered—it must be raised or lowered, brought forward or advanced further in the pharynx, as may be necessary. The operator must observe which part is reflected in the mirror; and in moving it, he is guided by his knowledge of the relations of the various parts of the larynx to each other. Unless the tonsils are enlarged, no part of the fauces or pharynx, except the soft palate and uvula, should be touched; and the observer must be specially careful that the lower edge of the mirror does not come in contact with the back of the pharynx. He must remember also that, while steady pressure can be borne, titillation of any part of the fauces will induce retching. Where the throat is very irritable, a 20 per cent. solution of the hydrochlorate of cocaine brushed over the soft palate and pharynx will greatly facilitate laryngoscopic examination. Enlarged tonsils may present an insuperable obstacle to successful examination; in slighter cases either a very large round mirror which presses the tonsils on one side, or a small ovate mirror, should then be used. More frequently a pendulous epiglottis hanging back over the upper part of the larynx impedes the view; in such a case the patient must utter, or try to utter, in a high falsetto tone, a prolonged 'eh,' or he must force a laugh or a cough, and the mirror must be held lower in the pharynx, in a more vertical position.

For the topical treatment of laryngeal diseases, in addition to the laryngoscope, the practitioner requires a laryngeal probe or sound; brushes or cotton-wool holders on stout metal handles, curved at a suitable angle for applying solutions; an insufflator for the introduction of powders; and a scaring instrument. For the operative treatment of polypus, &c., a set of special forceps, a laryngeal écraseur, special knives, and other instruments are necessary; while, for use by the patient, a spray-producer or atomiser, and a simple inhaler, will be required.

THOMAS JAMES WALKER.

**LARYNX, Diseases of.**—The functions and peculiar anatomical position of the larynx give to its diseases a special importance; and in addition to such objective and subjective symptoms as are common to affections of other organs, we find here modifications of respiration, vocalisation, and deglutition.

The principal diseases and disorders which affect the larynx may be conveniently enumerated and described in the following

order: (1) Disorders of Circulation; (2) Inflammation; (3) Leprosy; (4) Lupus; (5) Nervous Affections; (6) Spasm; (7) Syphilis; (8) Tuberculosis; and (9) Tumours. See also CROUP; DIPHTHERIA; and TRACHEA, Diseases of.

**1. Larynx, Disorders of Circulation of.**—*Anæmia* of the larynx does not exist as a separate disease. Like pallor of the gums, it is described as one of the symptoms of incipient phthisis.

*Congestion* of the larynx requires a passing notice. Hyperæmia of some portion of the mucous lining of the larynx results from over-exertion of the voice, exposure to cold, the action of irritants, or any cause obstructing the circulation through the larynx; and it may be consecutive to laryngitis. It causes more or less persistent hoarseness, and a sense of discomfort in the throat; and it excites what is commonly called a 'tickling cough.' Congestion is clinically inseparable from laryngitis.

**TREATMENT.**—Treatment of congestion of the larynx is unnecessary, beyond resting the voice, if the congestion is merely temporary. If more persistent, astringents should be applied locally, as in cases of chronic laryngitis.

**2. Larynx, Inflammation of.**—The varieties of laryngitis treated of here will be considered under the following headings: (a) **Acute**—including i. *Catarrhal*; ii. *Phlegmonous*. (b) **Chronic**.

(a) i. **Acute Catarrhal Laryngitis.**—**SYNON.**: *Laryngitis Catarrhalis*; *Cynanche Laryngea*; Fr. *Laryngite Aiguë Catarrhale*; Ger. *Kehlkopfentzündung*.

**DEFINITION.**—Acute catarrhal inflammation of the mucous membrane of the larynx.

**ÆTIOLOGY.**—Laryngitis is more common among males than females, and among children than adults. The most frequent exciting cause is exposure to cold, but the disease may often be attributed to sudden changes of temperature, a damp or irritating atmosphere, the inhalation of acid vapours or gases, excessive use of the voice, and to the abuse of alcohol, hot drinks, and spices. It may be due to accidental attempts at swallowing boiling water. The catarrhal process may be propagated from the pharynx or nose. It occurs also as a complication of exanthematous fevers—especially of measles. Sedentary occupations, and anything that depresses the vitality of the body, may predispose to laryngitis.

**ANATOMICAL CHARACTERS.**—Acute laryngitis is characterised by the changes common to catarrhal inflammation of other mucous membranes: hyperæmia, swelling, and increased secretion. Small superficial erosions may occasionally occur, especially at the posterior extremities of the vocal cords, but ulceration does not extend below the epithelial layer.

**SYMPTOMS.**—A mild attack of laryngitis

commences with hoarseness, irritable cough, a sense of tickling and soreness about the larynx, without any febrile disturbance. Subsequently the voice may be completely lost. In severe cases the patient becomes restless and anxious; experiences a feeling of constriction about the rima; complains of sore-throat in swallowing; and points to the larynx as the seat of pain. The breathing is altered, having more or less the characteristics of laryngeal obstruction, namely, the long-drawn hissing inspiration, prolonged expiration of the same character, but with less of the sibilant sound, and diminution or complete abolition of any pause between successive respiratory efforts. The voice also is altered, becoming husky and uncertain, deeper in tone, and croaking in quality. The patient likewise suffers from cough, of the same character as the voice; from the imperfect closure of the vocal cords it wants sharpness, is husky, and sometimes is accompanied by a hollow, clanging sound, constituting what is commonly called the *croupy cough*. The expectoration varies according as the inflammation involves the larynx alone, or extends to the trachea and bronchi. In the former case it is usually clear, thin, tenacious mucus, which is *hawked* rather than coughed up, mixed with the saliva, which is freely secreted but not swallowed. Sometimes the expectoration contains harder pellets, secreted in the ventricles of the larynx; whilst, if the trachea and bronchi are involved, the usual thick expectoration of bronchitis is also coughed up. Deglutition is painful and difficult owing to implication of the aryepiglottic folds and epiglottis; yet the pharynx, on inspection, shows only a little redness, quite inadequate to account for the dysphagia. Accompanying these symptoms there is a rapid pulse, and slight rise of temperature, though the tongue may be moist, and other indications of inflammatory fever slight.

The laryngoscope reveals congestion of the larynx, which varies much in extent and degree. In slight cases the reddening may be confined to special parts, like the epiglottis and vocal cords, without any appreciable swelling of the affected structures. The cords usually exhibit changes, which account sufficiently for the hoarseness. During attempts at phonation they are often seen to be imperfectly approximated, being separated by an elliptical fissure, the result of paresis of their internal tensor muscles, the thyro-arytenoides interni. Less frequently the posterior part of the glottis remains open during phonation, forming a triangular space with the apex directed forwards, a condition depending on paresis of the arytenoides muscle. These pareses of the cords are probably due to inflammatory implication of their neuro-muscular apparatus. Protrusion of the swollen inter-arytenoid fold between the posterior ends of the cords, preventing

their due adduction, is an occasional source of hoarseness. At other times no definite loss of movement can be recognised, and here the hoarseness is to be ascribed to alterations in the tension and vibration of the cords produced by the catarrh. In the severe type the whole larynx is congested, and the aryepiglottic folds, epiglottis, and ventricular bands are swollen so as to hide the vocal cords more or less.

In most instances of the mild form, recovery ensues in a few days.

If the case progress untowardly, the restlessness and anxiety increase; suffocative paroxysms occur and recur with increasing frequency; the patient's whole attention appears concentrated on the effort of breathing; he dreads to speak or swallow, and if obliged to say anything, he takes first a laboured inspiration, and then, with a straining effort, brings out what little voice is left. Gradually, as the aëration of the blood becomes more imperfect, drowsiness comes on; the eyes, staring in the previous stage, are half closed by the drooping lids; the face, bathed in perspiration, becomes livid; and death occurs, probably in a paroxysm of suffocative dyspnoea.

Acute laryngitis occurring in young children, sometimes termed *spasmodic laryngitis* or *false croup*, differs somewhat from the description just given. These differences depend on the small size of the child's larynx, and on the tendency to spasm incidental to this age. Without any previous illness beyond, possibly, a slight cough and hoarseness, the child is suddenly seized at night with a barking cough, noisy stridulous inspiration, and more or less urgent dyspnoea. The suffocative seizure seldom lasts more than a few minutes; but it may recur the same night, or on successive nights, the child being generally free from all symptoms, except slight cough and hoarseness, during the daytime. These attacks are mainly due to spasm of the sphincter muscles of the larynx; but in all probability they are also partly the result of drying of the secretions accumulated during sleep, and consequent narrowing of the glottis. Paroxysmal dyspnoea of the same nature, and due to the same cause, may develop as a complication of the exanthemata, especially measles.

DIAGNOSIS.—Acute laryngitis is apt to be confounded with that form of inflammation which is characterised by the formation of a false membrane in the larynx, namely, croup or laryngeal diphtheria. If practicable, a laryngoscopic examination, showing the absence of false membrane, is the most certain means of diagnosing simple from pseudo-membranous laryngitis; but even where perfect casts of the trachea, bronchi, and primary bronchia are expectorated, the larynx may be absolutely free from false membrane, and simply inflamed. The absence

of diphtheritic membrane in the fauces, of acrid exoriating discharge from the nostrils, and of glandular swellings, as well as the mode of accession of the symptoms, would lead us to regard the case as simple laryngitis; while the presence of any of these symptoms, or the prevalence of an epidemic of diphtheria, would lead us to suspect false membrane. Even after due consideration of these points, and of the character of the voice, cough, &c., in certain cases, we are unable to say whether we have to deal with simple or with pseudo-membranous laryngitis. The acute laryngitis or false croup of children is further distinguished from true croup by the sudden accession of the symptoms at night, and by the progress of the case—true croup or diphtheria increasing in intensity, while, from the first sudden onset of the symptoms, false croup diminishes, unless complicated with lobular pneumonia or other severe disease. From laryngismus stridulus, acute laryngitis is distinguished by the presence of slight pyrexia and other indications of primary inflammatory affection of the larynx and air-passages; by the absence of indications of any other affection of the nervous system, or of a tendency to convulsions; by the usual occurrence of the attack in the night only; by the slighter affection of the breathing; and by the frequent *croupy cough*, which is the prominent symptom, and which is wanting in laryngismus.

**PROGNOSIS.**—In mild attacks of laryngitis all the symptoms exist only in a slight degree, and disappear in a few days under simple treatment. Such is the ordinary course of the false croup of children. In adults laryngitis may occur in any degree between the mildest form and that in which, the symptoms being all most intense, it may prove fatal in a day or two, or even in a few hours. The danger depends in great measure on the amount of swelling present. Although mild laryngitis usually terminates favourably in a few days, it is necessary that every case should be sedulously watched, as at any period dangerous symptoms may set in and prove rapidly fatal. The disease is most deadly in the young. Acute may pass on to chronic laryngitis.

**TREATMENT.**—As soon as the first indications of even slight laryngitis are observed, the patient should be confined to a warm room, at a temperature of 65° F., a kettle being kept boiling to moisten the atmosphere. Talking must be forbidden, in order to ensure as much rest to the larynx as possible. The diet should be chiefly liquid, and no stimulants are required. As a rule, no further treatment is necessary; but, if the cough be troublesome, we may prescribe a saline diaphoretic draught, with small doses of morphine, and 4 or 5 minims of antimonial wine every three or four hours. The cough

and sense of dryness, or soreness of the throat, are generally relieved by the inhalation of simple steam, or by the vapour of compound tincture of benzoin, one drachm being added to a pint of hot water. In every case a regular evacuation of the bowels is to be secured. If swelling of the larynx ensue, the patient must be directed to suck ice constantly. Should the dyspnoea become urgent, tracheotomy must be performed at once. With due care in the performance of the operation and after-treatment of the patient, in cases of simple laryngitis, tracheotomy is almost always successful, but it should not be resorted to unless the case is urgent.

Simple cases of false croup in children are usually relieved by warm poultices to the throat. An emetic may be administered in the comparatively rare instances in which dyspnoea depends on insufficient removal of secretions from the larynx.

A brief allusion may here be made to the method of intubation which has been successfully practised by Dr. O'Dwyer and others for the relief of laryngeal diphtheria. It is possible that further experience may warrant the adoption of this treatment in place of tracheotomy, in certain cases of laryngitis associated with stenosis, whether in the child or adult.

**ii. Phlegmonous Laryngitis.**—**SYNON.:** *Edema Glottidis*; (Edema of the Larynx; Edematous Laryngitis; Fr. *Laryngite Édémateuse*; Ger. *Kehlkopfödem*.)

**DEFINITION.**—Inflammatory exudation into the submucous tissue of the larynx of serous, sero-purulent, or sero-fibrinous fluid.

**ÆTIOLOGY.**—Phlegmonous laryngitis occasionally arises as a primary disease, and is then to be regarded as a symptom of septic infection; thus in rare instances it seems to have been the earliest manifestation of erysipelas.

Mechanical injuries, and the direct action of boiling water, corrosive poisons, and foreign bodies are less uncommon causes. Phlegmonous laryngitis may also develop in the course of specific fevers, such as measles, scarlatina, small-pox, typhoid fever, diphtheria, pyæmia, and in renal disease.

In most cases, however, the laryngeal affection is the result of extension of disease from neighbouring parts, pharyngitis, whether dependent on erysipelas or other causes, being the most common antecedent.

Inflammatory affections of the cervical connective tissue, and perichondritis, or deep ulceration of the larynx, associated with carcinoma, syphilis, or tuberculosis, are also occasional causes of phlegmonous laryngitis.

**ANATOMICAL CHARACTERS.**—The mucous surface presents a dull red or livid colour, and is greatly swollen, owing to infiltration of the submucosa. A certain amount of pus is generally diffused through the exudation,

but it is rare for a circumscribed abscess to form. The process usually attacks first the aryepiglottic folds and the epiglottis, and it may extend thence to the ventricles and other parts of the larynx, the vocal cords themselves being seldom affected. The submucous tissue below the vocal cords, lining the cricoid cartilage, may very occasionally be the sole seat of the disease.

**SYMPTOMS.**—The symptoms indicative of the severe laryngitis exist in an increased degree in this affection, the swelling of the epiglottis interfering especially with deglutition, and that of the aryepiglottic and ventricular bands causing very dangerous dyspnoea. By examination with the finger and, far better, by the laryngoscope, we observe further the physical condition caused by the swelling. The epiglottis is congested, misshapen, rounded like a chestnut, or its two sides swollen so as to resemble two mucous bladders pressed together in the middle line; and, unless this hides the rest of the larynx, the aryepiglottic folds will be recognised as two long rounded swellings, passing from before back, and nearly meeting in the centre, the ventricular bands being visible only when there is little swelling above. The prognosis in such cases is extremely grave.

**TREATMENT.**—The patient should suck small pieces of ice, leeches might be applied over the larynx, and an attempt may be made to reduce the swelling by scarification with a curved laryngeal bistoury, but in most instances tracheotomy is required.

(b) **Chronic Laryngitis.**—**DEFINITION.** Chronic inflammation of the mucous membrane of the larynx.

**ETIOLOGY.**—Chronic laryngitis is often, but not invariably, a sequel of acute laryngitis, and is due to the same causes. Chronic affections of the nose and pharynx frequently co-exist, and seem in some instances to be the starting-point of the malady.

**ANATOMICAL CHARACTERS.**—The mucous membrane of the larynx is hyperæmic, and may be slightly swollen, the vessels being often dilated in patches. The morbid process may extend in rare cases to the submucosa, producing diffuse connective-tissue thickening, or small warty growths, especially on the vocal cords—'chorditis tuberosa' of Türk. Marked enlargement of the veins on the epiglottis is occasionally observed, and is probably the result of the chronic catarrh.

**SYMPTOMS.**—The symptoms of chronic laryngitis are hoarseness or aphonia after slight exertion of the voice; a hacking cough, with but little of the brassy, laryngeal character; either no expectoration, or only a little tenacious mucus; and a sense of dryness and tickling, with slight pain, in the throat.

The laryngoscope shows a varying amount of congestion, either general or local, but the hyperæmia is seldom so pronounced as in

acute laryngitis. The cords are usually affected, presenting a pinkish, slightly thickened appearance.

Mucous secretion is often seen sticking to the inter-arytenoid fold or to the anterior angle of the glottis. In a special variety of the disease, known as 'laryngitis sicca,' the secretion is apt to dry and form adherent scales or crusts, in consequence of some modification of its composition. Epithelial erosions are not uncommon, but ulceration never occurs. Pareses of the vocal cords often ensue, giving rise to changes in the glottis, as described under Acute Laryngitis.

In the rare instances in which thickening invades the subglottic region, stenosis commonly ensues, and obstructive dyspnoea becomes a prominent symptom.

**DIAGNOSIS.**—In cases of chronic disease of the larynx a laryngoscopic examination is indispensable. Without the laryngoscope diagnosis and treatment can be only guess-work; with it, the appearances described above are clearly recognised. Where chronic laryngitis is persistent, the possibility of tuberculosis must be kept in mind, and repeated examination of the lungs and sputum should be made. In elderly or middle-aged patients a localised swelling and congestion of the vocal cords, especially if associated with impaired mobility, should arouse the suspicion of carcinoma.

**PROGNOSIS.**—Chronic catarrhal laryngitis may subside spontaneously, but is usually persistent unless properly treated.

**TREATMENT.**—Any imprudence in diet or hygiene must be corrected, pungent condiments or spices, very hot drinks, alcohol in excess, smoky or dusty rooms, being avoided, and the voice rested as much as possible. About every other day the larynx should be brushed out with an astringent lotion. In mild cases a solution of chloride of zinc, 30 grains to the ounce, will suffice; but if this fail, a more powerful astringent, nitrate of silver 10 to 40 grains to the ounce, should be employed. Such applications should not be continued for more than two or three weeks at a time.

Inhalations of chloride of ammonium, pine oil, and creasote are recommended by some physicians. In some obstinate cases the waters of Ems, Reichenhall, Marienbad, Aix-les-Bains, and other spas are believed to yield good results. Attention must be paid to the general health, and to the state of the digestive organs.

3. **Larynx, Leprosy of.**—**ETIOLOGY.**—The causes are identical with those of leprosy in other situations. Leprosy of the larynx may be associated with a similar affection of the buccal mucous membrane, the tongue, and the palate.

**ANATOMICAL CHARACTERS.**—The lesions at first consist of a nodular thickening of the epiglottis and aryepiglottic folds, which may

be succeeded by very chronic ulceration and cicatrisation. Ultimately the disease may invade other parts, and cause stenosis of the larynx.

**SYMPTOMS, COMPLICATIONS, AND DIAGNOSIS.** The symptoms of laryngeal leprosy are those of chronic inflammation; and, if the nodules ulcerate, the ulceration is very slow, and not of the destructive character of syphilis. As leprosy of the larynx occurs only when the disease of the skin has been long established, its diagnosis is clear. It usually exists for a long time, even years, before it ulcerates; and even after this it is very slow in its progress, and not fatal.

**TREATMENT.**—As in other forms of leprosy, treatment is almost useless. Tracheotomy must be performed if stenosis occur. See **LEPROSY**.

**4. Larynx, Lupus of.**—**ÆTIOLOGY.**—The causes of this disease and of lupus of the skin are identical. It is doubtful whether the larynx is ever primarily affected.

**ANATOMICAL CHARACTERS.**—Small nodules appear on the epiglottis, and subsequently on the aryepiglottic folds and other parts of the larynx, associated sooner or later with thickening and ulceration of the mucous membrane. Cicatrisation of the ulcers is almost an invariable feature of such cases.

**SYMPTOMS, COMPLICATIONS, AND DIAGNOSIS.** Lupus of the larynx may give no definite symptoms, even when pronounced lesions exist, and doubtless on this account this affection of the larynx is often overlooked. In other cases the symptoms are those of chronic inflammation; but there appears to be less tendency to œdema or to those affections of the cartilages which occur in syphilis and tuberculosis. Lupus is distinguished from syphilis by its prevailing nodular character, and by the slow progress of ulceration. From tuberculosis, which it more closely resembles, it differs mainly in its marked tendency to the formation of cicatrices. But in all cases the diagnosis turns principally on the presence of lupus of the skin. It lasts for many years.

**TREATMENT.**—Lupus of the larynx must be treated constitutionally in the same way as other forms of lupus, and locally by caustics or the galvanic cautery. Lactic acid, applied as in cases of laryngeal tuberculosis, promises to be of service in lupus also. See **LUPUS**.

**5. Larynx, Nervous Affections of.**—

**(1) SENSORY DISORDERS OF THE LARYNX.**—*Hyperæsthesia* of the laryngeal mucous membrane is a usual accompaniment of inflammation, acute and chronic, and is a not infrequent symptom of hysteria, hypochondriasis, and neurasthenia. *Anæsthesia* occurs in cases where the superior laryngeal nerve or its centres are affected, and especially as a sequel of diphtheria. It is also an occasional symptom of bulbar paralysis and hysteria,

and is said to accompany epileptic fits. *Hæmiæsthesia* may result from tumours of the base of the skull. *Paræsthesia*, in which the larynx is the seat of various perverted and unaccountable sensations, is not an uncommon manifestation in anæmic, hysterical, and hypochondriacal patients.

**(2) MOTOR DISORDERS OF THE LARYNX.**—

**(a) Spasm** is considered in a separate section. See **6. Larynx, Spasm of**.

**(b) Paralysis** of the larynx is of great variety. It will be best discussed, first, under the head of the *nerves* affected; and, secondly, with respect to the individual *muscles* paralysed.

**i. Paralysis of the Superior Laryngeal Nerve.**—In this rare condition, depending either on diphtheria or bulbar paralysis, the interior of the larynx is quite insensitive to the introduction of a probe; the epiglottis lies motionless against the base of the tongue, owing to paralysis of its depressors—the thyro-epiglottic and aryepiglottic muscles; and the voice is said to be rough and low-pitched, from palsy of the cricothyroid muscles. The glottis is represented by a wavy line, in consequence of paralysis of the external tensors. The immobility of the epiglottis and the anæsthesia are a source of great danger, as food is likely to enter the larynx and lead to pulmonary complications. Food must be administered by the œsophageal tube in such cases. See **LABIO-GLOSSO-LARYNGEAL PARALYSIS**.

**Paralysis of the Recurrent Laryngeal Nerve.**—Complete paralysis is generally due to pressure of an aneurysm of the aorta or other intrathoracic tumour, or of a goitre, on the nerve after its origin from the vagus, but the same effect may follow degenerative or destructive lesions of the motor fibres, in any part of their course from the medulla oblongata down to their peripheral termination. Bilateral paralysis, in the very rare instances in which it has been observed, was the result of carcinoma of the œsophagus, double aneurysm of the aorta and innominate artery, tumours of the base of the brain, extensive pericardial effusion, and, lastly, pressure on the trunk of one pneumogastric nerve, for example, by enlarged bronchial glands. Bilateral paralysis, following unilateral pressure, is regarded by Sir George Johnson, who first observed it, as an instance of reflex paralysis; but the more general belief is that secondary changes are set up in the nuclei of the recurrent laryngeal nerves, as the result of ascending irritation. The symptoms of bilateral paralysis are aphonia, inability to cough, and slight stridor. Slight dyspnoea may be induced by attempts to speak. The vocal cords are seen lying motionless and relaxed, in a position midway between abduction and adduction. In unilateral paralysis, which is the usual form and is more common on the left side, one cord alone occupies the

position just described, but, as the healthy cord crosses over the middle line during phonation to meet its palsied fellow, there is no aphonia, though the voice is generally weak and uncertain.

Hitherto complete paralysis alone has been considered; but paralysis depending on structural disease of the fibres or nuclei of the recurrent laryngeal nerves is far more often incomplete. In these circumstances, as Dr. Semon and Dr. Rosenbach have shown independently, the fibres supplying the abductor muscles are the first to suffer. In course of time the adductors may become affected also, but paralysis of these muscles is seldom complete. This fact is attributed to a greater vulnerability of the abductor nerve-fibres than of those supplying their antagonists, the adductors. Dr. Semon believes that the abductor muscles themselves are less resistant than the adductors. It is impossible to discuss here the experiments and clinical facts on which this important doctrine is based.

ii. *Paralysis of Individual Muscles.*—*Bilateral Abductor Paralysis.*—The cause may be either central—as in tabes dorsalis, bulbar paralysis, syphilitic or other intracranial disease; or peripheral—lesions of both recurrent or pneumogastric nerves, pressure on the trunk of one vagus, or degenerative affections of the abductor muscles, as described under the preceding heading. The symptoms are severe inspiratory dyspnoea and stridor, and weakness or slight hoarseness of the voice. With the laryngoscope the cords are found lying close together, being separated by a mere chink. On phonation they approximate as usual, but during deep inspiration no divergence takes place, and a slight movement of adduction is generally observed. This action is attributed either to the effects of suction, or to a perverted innervation of the unopposed adductors. Bilateral paralysis may cause fatal suffocative attacks, and, if persistent, necessitates the performance of tracheotomy.

Abductor paralysis may be simulated by mechanical fixation from ankylosis or arthritis of the crico-arytenoid joints, or by cicatricial or inflammatory changes in their neighbourhood. Diagnosis is often difficult in such cases, but in the latter condition there is usually some distortion of parts, such as contraction, irregularity, or swelling about the arytenoid cartilages, which is not the case in paralysis.

*Unilateral Abductor Paralysis.*—This condition is generally attributable to peripheral causes, and especially to compression of the left recurrent or vagus nerve within the thorax by an aneurysm or other tumour, as we have just seen in discussing paralysis of the recurrent laryngeal nerve. Palsy of the right abductor is an occasional result of chronic disease of the apex of the right lung.

The affected vocal cord occupies the median position; but if the adductor becomes involved, the cord recedes from the middle line and takes up a situation between that of abduction and adduction. The symptoms of unilateral paralysis are slight hoarseness and dysphonia, but dyspnoea and stridor do not occur except on exertion.

*Paralysis of the Adductors.*—Paralysis of these muscles is nearly always bilateral, and is probably the result of a central nervous affection of a functional nature, occurring in hysterical, neurotic, or anæmic subjects. It is doubtful whether gross nervous disease ever leads to isolated paralysis of the adductors. The patient, generally a female, becomes suddenly aphonic, but the reflex acts of sneezing and cough are still attended with the usual sound. Attempts to speak occasion a slight degree of dyspnoea, due to the escape of breath through the widely open glottis. The laryngoscope shows that the cords are abducted normally, but do not approach one another during efforts to phonate.

*Paralysis of the Internal Tensors.*—The internal tensors or thyro-arytenoidei interni may be paralysed, on one or both sides, as a consequence of laryngitis or of hysteria. According as the affection is bilateral or unilateral, there is aphonia or mere hoarseness. During phonation the vocal cords are separated by an oval or elliptical space, and appear to be very thin and narrow, owing in all probability to deficient tension.

*Paralysis of the External Tensors,* the crico-thyroids, is unknown, apart from the results of paralysis of the superior laryngeal nerve.

*Paralysis of the Arytenoideus.*—This is also commonly a result of hysteria or of laryngeal catarrh, and gives rise to hoarseness or aphonia. During phonation the cords meet in front, but are separated posteriorly by a triangular gap.

**TREATMENT.**—If the paralysed condition depend on serious disease outside the larynx, local treatment is of little avail. When syphilis or aneurysm is suspected, iodide of potassium should be prescribed freely. In cases of adductor paralysis, anæmia and the hysterical state must be suitably treated, and intra- or extra-laryngeal faradisation may be tried, but sudden recovery often occurs without any treatment. See PNEUMOGASTRIC NERVE, Diseases of.

6. **Larynx, Spasm of.**—In the description of other diseases of the larynx, this condition has been referred to as a frequent complication. Spasm causes the urgent symptoms when the larynx is irritated by the lodgment of a foreign body or the like. It may result from pressure on, or disease of, the pneumogastric or recurrent laryngeal nerve; from hysteria; or as a manifestation of a more general affection of the nervous system, in

which case it constitutes the following special malady.

*Laryngismus Stridulus*.—SYNON.: Child-Crowing; Spasmodic Croup; False Croup; Fr. *Spasme de la Glotte*; *Pseudo-croup Nerveux*; Ger. *Kehlkopfkrampf*.

DEFINITION.—Laryngismus stridulus is characterised by short or more prolonged accessions of suffocation; depending on tonic spasm of the adductor muscles of the larynx, and usually of the diaphragm and other respiratory muscles, causing closure of the glottis and a sudden arrest of inspiration; and ending in a shrill crowing sound, as the inspiratory act is resumed and concluded. It is purely a nervous disease; is unaccompanied by any inflammatory affection of the larynx or air-passages; and is often associated with other convulsive affections.

ÆTIOLOGY.—Anything causing excessive reflex irritability, rickets, chronic hydrocephalus and other organic affections of the brain or medulla oblongata, predispose to this convulsive affection. It is sometimes associated with the irritation of teething. A sudden fright, irritation of the larynx by the accidental entrance of food, or some such slight agitation as that caused by a child being tossed in the air, may excite the attack.

SYMPTOMS.—With or without premonitory indications of a tendency to convulsive affection, such as drawing-in of the thumbs and great toes, or clenching of the hands; often during sleep, and with no evident exciting cause, or at any time in the day; a child is suddenly attacked with difficult breathing, inspiration being accompanied by the crowing sound characteristic of laryngeal spasm. This may continue for some time, and then gradually subside. The spasm may be short, or it may be longer and more intense, inspiration being proportionately difficult. It may be complete, and the act of inspiration cease entirely, until, just as death seems imminent, the spasm relaxes, and with a crowing inspiration breathing is re-established. In the worst cases, and sometimes in the first attack, death does actually occur.

DIAGNOSIS.—The diagnosis of this disease from laryngitis is considered under Acute Laryngitis. The symptoms caused by a foreign body lodged in the larynx closely simulate laryngismus stridulus. The nature of the case is decided by its history; and, unless the age of the patient precludes it, by a laryngoscopic examination.

PROGNOSIS.—The milder forms of laryngismus stridulus yield to suitable treatment, and disappear as improvement takes place in the condition inducing the attack. Severer forms, if not fatal in a first, may be so in a subsequent attack. When the spasm depends on some incurable organic change in the nervous system, the case is of course hopeless from the first.

TREATMENT.—Attention to the diet and

general management of the child, regular bathing, and the administration of remedies suitable to correct faults of digestion, are necessary (see RICKETS). The persevering use of bromide of potassium has been found beneficial by the writer (T.J.W.); and chloral hydrate is of undoubted value. For the immediate treatment of the spasmodic attack, prompt immersion in a warm bath, the administration of an emetic, or the use of an anæsthetic vapour or amyl nitrite may be resorted to; and should breathing not be re-established as the spasm ceases, dashing cold water on the face and chest, friction, application of strong ammonia or vinegar to the nostrils, and especially artificial respiration, must be adopted, with the object of restoring the respiratory function. Tracheotomy may be requisite, but the practitioner is, unfortunately, seldom present when the indications for the operation arise.

7. **Larynx, Syphilis of.**—Syphilis affects the larynx differently according to the stage of the disease at which the organ is attacked. Erythematous maculae, raised mucous patches or condylomata, and superficial ulceration, like that seen in the fauces and pharynx, are described as secondary lesions, but are seldom actually observed, as opportunities for making a laryngoscopic examination are not often afforded. Mucous patches, represented by small greyish flat elevations, and congestion, are the commonest manifestations at this period of the disease. These patches generally disappear without leaving any trace of their existence.

The larynx is more seriously affected in advanced stages of syphilis. The lesions, consisting of diffuse infiltration, nodular gummatous deposits, or ulceration, are especially prone to attack the epiglottis and vocal cords, and in a less degree the ventricular bands and posterior wall.

The special proclivity of the epiglottis to syphilitic disease has been attributed to direct infection from the pharynx, it being an undoubted fact that syphilis generally involves the pharynx together with the larynx. Syphilitic ulceration may be superficial or deep. Deep ulcers, which are often of gummatous origin, are sharply cut, and extend rapidly. As a result, extensive destruction of the epiglottis, suppurative perichondritis, and necrosis of the various cartilages, often ensue. External fistulae are occasionally thus produced. Perichondritis at times develops without previous ulceration. Cicatrices are almost an invariable sequel of deep ulceration, and are a great source of danger, owing to the contraction and stenosis which they occasion. Curious puckering of different parts is commonly produced, at times associated with small polypoid excrescences of the mucous membrane. These last, unlike gummata, show no tendency to ulcerate, and may gradually disappear. Adhesion and

webbing of the cords, adhesion of the epiglottis to the walls of the pharynx, fixation of the vocal cords in various positions, sometimes simulating bilateral abductor palsy, and depending then on fibrous changes around the crico-arytenoid articulations,—these are some of the many effects of syphilitic cicatrization.

In *hereditary* syphilis, lesions of the larynx generally occur within the first few months after birth, but occasionally the larynx is not attacked before the age of puberty is reached. The early manifestations of the disease in hereditary cases are represented by congestion and mucous patches; at a later period they are not to be distinguished from those which occur in the advanced stage of acquired syphilis. Deep ulceration in any case may be associated with extensive œdematous swelling, and hæmorrhage from perforation of large vessels is an occasional occurrence.

**SYMPTOMS.**—Secondary syphilitic affections cause hoarseness and sometimes loss of voice, but there is usually neither pain, cough, fever, nor dyspnoea. The more serious tertiary affections may give rise to all the symptoms caused by tubercular ulceration, but pain and dysphagia are rarely experienced except when the epiglottis is extensively ulcerated, and even then these symptoms are less severe than in tubercular cases, and the constitutional symptoms are those of syphilis, as distinguished from consumption.

**DIAGNOSIS.**—Secondary syphilitic affections of the larynx are easily recognised with the laryngoscope, and there are usually other indications of the constitutional taint. The diagnosis of the disease, in its later stages, must be based on the appearance of the lesions above mentioned, combined with manifestations of syphilis in other parts of the body, especially of the skin and pharynx. In the absence of constitutional symptoms, the diagnosis may present great difficulties, especially as regards tuberculosis. This question will be discussed in the next section.

It is sometimes not altogether easy to distinguish syphilitic from carcinomatous ulceration, but in the latter disease the appearance of a growth is more pronounced, ulceration is less rapid, and pain is more often present. Moreover, the vocal cords and ventricular bands are the favourite seats of malignant growths, the epiglottis and other parts being seldom primarily affected.

The good results following the use of iodide of potassium will often assist in arriving at a correct diagnosis, but this test is open to a fallacy, as carcinoma and syphilis may co-exist.

**PROGNOSIS.**—The prognosis in cases of laryngeal syphilis must vary according to its form. In early cases, and where ulceration is superficial, the prognosis is very favourable, but in advanced stages the prospects of the patient are more uncertain, for, although

the progress of ulceration can almost invariably be arrested by treatment, subsequent cicatrization may cause dangerous stenosis. A knowledge of the complications incidental to deep ulceration should induce us always to give a guarded opinion.

**TREATMENT.**—Local treatment is unnecessary for the secondary syphilitic affections of the larynx, and we must trust to mercurial inunction and other constitutional remedies. Syphilitic ulceration of the larynx requires persevering treatment with large doses of iodide of potassium; and where it does not yield to this remedy, appropriately combined with quinine, cod-liver oil, &c., mercury must be employed, the best method being by mercurial inunction or the hypodermic injection of the bichloride. A combination of mercury and iodide of potassium is often useful. The local application of a solution consisting of iodine 10 grs., iodide of potassium 100 grs., glycerine 1 oz., is sometimes of service in the case of syphilitic ulcer, in addition to the above treatment. When the dyspnoea is dangerous, tracheotomy must not be delayed. It will usually prove successful, although the destruction of tissue and cicatrization may be such as to necessitate the permanent wearing of the tube. Operations for the division of cicatricial bands and adhesions seldom give satisfactory results, owing to the tendency of the parts to unite again. Systematic dilatation, by means of Schrötter's hollow vulcanite bougies, may be tried in such cases.

**8. Larynx, Tuberculosis of.**—**SYNON.:** Laryngeal Phthisis; Fr. *Phthisie Laryngée*; Ger. *Kehlkopftuberculose*.

**ÆTIOLOGY.**—Laryngeal tuberculosis is almost invariably secondary to pulmonary phthisis, but in a few instances a necropsy has demonstrated the existence of tuberculous disease in the larynx without any affection of the lung. In the former case the larynx is either directly inoculated with infective sputum derived from the lung, which is the usual method, or the tubercular virus is conveyed to the larynx by the blood. Where the larynx is primarily affected, we may suppose that the infective agent, the tubercle bacillus, is inhaled, and effects a lodgment here without reaching the lung.

**ANATOMICAL CHARACTERS.**—In the larynx, as in other mucous membranes, the tuberculous process may be considered under two heads—infiltration or deposit, and ulceration; but inasmuch as the necrotic element predominates over the fibrous or indurative, the tendency is ultimately towards ulceration. The degree of infiltration varies greatly, now giving rise to massive swelling of parts like the epiglottis and aryepiglottic folds, where the submucous tissue is loosely arranged, at other times merely extending to the sub-epithelial layer. In the latter case, owing to the early development of ulceration, a stage

of infiltration cannot generally be seen to precede loss of substance, though from analogy there is reason to believe that this is invariably the case. Definite tuberculous tumours are occasionally met with.

The parts most prone to tuberculous disease may be thus enumerated, in the order of frequency: the vocal cords and inter-arytenoid fold in about equal proportions, the aryepiglottic folds, epiglottis, and, lastly, the ventricular bands. Extension of the infiltration and ulceration to the deeper parts may lead to suppurative or adhesive perichondritis, especially of the arytenoid cartilages. Suppurative perichondritis causes necrosis, and at times extrusion of the cartilage. Œdema is a frequent complication of extensive tubercular lesions. It is unnecessary in the present article to describe the manifold changes that may be produced in different cases.

**SYMPTOMS.**—The symptoms of this affection are those of an aggravated laryngitis: hoarseness or aphonia, irritable cough, difficulty or pain in swallowing when the epiglottis or aryepiglottic folds are involved, and inspiratory dyspnoea and stridor if stenosis be present. Pain shooting to the ear is occasionally complained of, and is probably referred from the sensory nerve of the larynx, the superior laryngeal, to the auricular branch of the vagus. The constitutional and local symptoms of pulmonary phthisis, which are present in varying degrees, add much to the patient's distress. In early cases the laryngoscope reveals small shallow ulcers or pale granulations on the vocal cords, particularly their posterior ends, fleshy swelling of one or both cords, and prominence of the inter-arytenoid fold, often with an irregular papillary margin. Swelling of one or both sides of the epiglottis, aryepiglottic folds, and ventricular bands, and deep ulceration may be observed later on. The ulcers present either a greyish yellow or pinkish granular base, and minute yellowish nodules are occasionally developed in their neighbourhood. In certain instances the vocal cords are split longitudinally into terrace-like reddish or ulcerated ridges, so that each cord seems to be composed of several individual segments. Small tubercular tumours, of a greyish pink colour, may occasionally be seen springing from the cords, ventricles, ventricular bands, or other parts. When the epiglottis is much swollen or hangs backwards, it may be impossible to obtain a view of the rest of the larynx. A similar difficulty is sometimes experienced when the larynx is coated with a copious viscid secretion. The colour of the larynx varies greatly: generally speaking, a greyish pink or pale yellowish tint prevails, contrasting often with localised patches of congestion in the vicinity of ulcers or infiltrations. But this colour, which is very characteristic, is not invariably met

with, and general congestion may exist. Impaired mobility of the cords is very common: it mostly depends on mechanical fixation of the arytenoid cartilages from surrounding infiltration. At times the vocal cords occupy the median position, simulating bilateral abductor paralysis, and giving rise to severe stenosis. Neuropathic paralysis also occurs. Thus one cord may be motionless in the cadaveric position, midway between that of adduction and abduction. On the right side this condition may occasionally be the result of implication of the recurrent nerve in pleuritic thickening of the apex of the right lung; whereas the left recurrent may be compressed by enlarged bronchial glands as it winds round the arch of the aorta. Imperfect closure of the glottis depending on bilateral paresis of the adductors, as in cases of functional or hysterical paralysis, is a more common appearance. Lastly, in advanced cases of phthisis, aphonia may be unassociated with any defect of movement, the feeble action of the respiratory muscles being insufficient to cause the requisite vibration of the cords. In other instances paralysis is probably of myopathic origin.

**DIAGNOSIS.**—Chronic laryngitis is sometimes one of the earliest symptoms of phthisis, and therefore should always direct attention to the state of the lungs. The diagnosis of tubercular disease rests on the detection of localised infiltration or ulceration of certain parts of the larynx, in association with physical signs of pulmonary tuberculosis. Syphilis is the disease with which it is most likely to be confounded, and the difficulty is increased by the fact that the two affections may co-exist; some authors even assert that syphilitic ulceration predisposes to tuberculosis. Pronounced pallor of the larynx, swelling of the inter-arytenoid fold, with papillary excrescences, fleshy swelling of one cord, chronic ulcers of the cords, and a pale swollen condition of the aryepiglottic folds and epiglottis, are very characteristic of tuberculosis. On the other hand, marked congestion, rapidly extending ulceration of the cords or epiglottis, and cicatricial contraction, would be in favour of syphilis. Nevertheless, in many cases the laryngoscopic appearances are quite inconclusive; and, should examination of the lungs yield a negative or equivocal result, the detection of the tubercle bacillus in the sputum will alone establish the diagnosis. Tuberculosis can seldom be mistaken for a malignant growth, but the diagnosis will be considered under the latter disease.

**PROGNOSIS.**—Laryngeal tuberculosis is rarely cured, though spontaneous cicatrisation undoubtedly occurs in exceptional cases. The progress of the disease can often be temporarily checked, and at times is arrested by suitable treatment. As a rule, however, the prognosis is most unfavourable. Infiltration

of the epiglottis and aryepiglottic folds has a most ominous significance, owing to the interference with deglutition thereby entailed.

**TREATMENT.**—The constitutional treatment is discussed in the article on PHTHISIS; consequently the local treatment alone will be considered here. Lactic acid, introduced by Krause, is the most efficacious remedy at present known. The writer (P. K.) has seen several cases where permanent cicatrization of tubercular ulcers has followed the systematic application of this remedy. The larynx must first be well brushed out with a 20 per cent. solution of cocaine; and then, after an interval of three to five minutes, a 40 per cent. solution of the acid should be thoroughly applied on a small piece of cotton wool, fixed to the screw of a laryngeal holder. The strength of the solution should be increased till saturation is reached, and the treatment should be continued for ten days to a fortnight, the applications being made every other day, or in some cases daily. It is extremely important to paint the larynx thoroughly with cocaine two or three times before using the acid, otherwise the application is very painful. If, notwithstanding this precaution, pain be experienced, a weak spray of cocaine to the larynx may be trusted to give relief. The selection of fit cases is a matter of great moment. Real benefit is only to be expected where localised ulceration is unassociated with much submucous thickening. Where deep infiltration exists, radical treatment is out of the question.

A solution of menthol in olive oil, 20 per cent., painted on the larynx, gives much relief to pain and dysphagia, and is said to promote healing of ulcers; but in the last respect it cannot be compared to lactic acid. Insufflation of a powder consisting of boric acid and iodoform equal parts, with cocaine or morphine  $\frac{1}{2}$  to  $\frac{1}{3}$  of a grain, has also been much praised, but its effects are only palliative.

Painful deglutition can generally be relieved by spraying the throat, a few minutes before food is taken, with a 2 to 5 per cent. solution of cocaine. A lozenge, containing  $\frac{1}{10}$  to  $\frac{1}{6}$  of a grain of cocaine, also gives relief in less severe cases. Insufflation of morphine,  $\frac{1}{2}$  of a grain, with a little starch-powder or sugar of milk, produces less complete analgesia, but the effect lasts rather longer.

In cases where liquids enter the larynx during drinking, as sometimes happens when the epiglottis is destroyed, Dr. Wolfenden recommends that the patient should drink through a tube, lying flat on his face. Thickened drinks can often be taken better than ordinary fluids in such cases. Laryngeal cough may be mitigated by a cocaine spray, the use of menthol or oil of peppermint in an oro-nasal respirator, insufflations of morphine, or by sucking ice.

Tracheotomy is only to be performed when

marked stenosis is produced; but this operation is seldom required, and its effects on the pulmonary disease are very unfavourable, on account of the difficulty of expectoration which is experienced when the action of the glottis is abolished by the introduction of a tracheal cannula.

**9. Larynx, Tumours of.**—(a) **Benign.**  
SYNON.: Polyphi, or Growths of the Larynx; Fr. *Tumeurs, Kystes, et Polypes du Larynx*; Ger. *Kehlkopfpolypen*.

**ÆTIOLOGY.**—The invention and use of the laryngoscope, leading to accurate diagnosis, has established the fact that tumours of the larynx are of much more frequent occurrence than was formerly supposed. They are most common in adult males, but they occur in either sex, and at any age from infancy upwards. Inflammatory attacks, syphilis, and anything leading to habitual congestion of the larynx, are said to favour their development.

**ANATOMICAL CHARACTERS.**—Growths of various kinds occur in the larynx, papillomata being much the commonest. These present the appearance of a sessile warty or mulberry-like growth, attached generally to the anterior part of the vocal cords, though they may spring from almost any part of the larynx. Papillomata vary in size from a millet-seed to a walnut, and may be single or multiple. They sometimes show a tendency to recur when removed. Fibromata, the next commonest form of innocent tumour, may be sessile or pedunculated; their surface is usually smooth or slightly lobulated, but in size and localisation they closely resemble papillomata.

All other benign growths are very rare, and it must suffice to enumerate myxoma, lipoma, enchondroma, adenoma, angioma, and cystic tumour.

**SYMPTOMS.**—Benign tumours may cause little inconvenience; but, owing to the fact that the commoner varieties generally affect the vocal cords, some modification of the voice is almost a constant symptom. A dry spasmodic or croupy cough is sometimes excited. The breathing is but little affected until the growth attains some size, when dyspnoea will set in, at first only on exertion, occasionally spasmodic; as the growth increases, it becomes constant, and at last it may prove fatal if the disease be unrelieved. Tumours attached below the vocal cords are rare; when they exist, and are large enough to interfere with the breathing, expiration is as noisy and difficult as inspiration. These growths are usually painless. They may be so situated as to interfere with swallowing, but this is not usual. In addition to these symptoms, the growth can be seen with the aid of the laryngoscope, and felt with the laryngeal probe or sound, and sometimes with the finger. Occasionally portions of a papilloma are expelled by coughing.

**DIAGNOSIS.**—A certain diagnosis can only be arrived at by examination of the larynx. The laryngoscope will often, but not invariably, enable us to distinguish a papilloma from a fibroma. The diagnosis of a benign from a malignant growth is sometimes no easy matter, though the absence of ulceration, congestion, or infiltration, which characterises an innocent tumour, is rare in malignant disease. The fact that simple growths hardly ever originate from the inter-arytenoid or aryepiglottic folds will help to distinguish these from the polypoid excrescences that often fringe the margin of tubercular or syphilitic ulcers in these regions.

**PROGNOSIS.**—The importance of these growths varies with their situation and rate of increase. A few months have sufficed for the growth of tumours from their origin to their attaining a size sufficient to threaten suffocation; in other cases they may exist for years without giving rise to any symptom beyond dysphonia or aphonia. As soon as a growth causes dyspnoea it has become dangerous. The disease must practically be regarded as incurable, except by operation or other local treatment.

**TREATMENT.**—A small stationary fibroma or other tumour, giving rise to but little inconvenience, requires no treatment. All other tumours must be removed by operation. Removal may be effected by instruments introduced into the larynx from above with the aid of the laryngoscope; or, where the disease is very extensive, an artificial opening into the larynx (thyrotomy or division of both thyroid and cricoid) may be necessary, the growth or growths being removed through this opening. This is the only feasible method of operation in the case of children. If the growth is removed *per vias naturales*, it may be crushed through at its base by a properly constructed *écraseur*; it may be seized and torn off by forceps; or it may be cut off by knives or scissors. The particular operation, and the instruments to be used, must be determined by the circumstances of the case. The use of a cocaine spray renders such manipulations much easier. The statement which has been advanced that the irritation of endo-laryngeal operations may induce the transformation of innocent into malignant growths has been conclusively disproved.

(b) **Malignant.**—Malignant tumours of the larynx may be primary or secondary. Primary growths are far less rare than was formerly thought, carcinoma being much more often met with than sarcoma. Epithelioma is the commonest form of carcinoma, the medullary and scirrhous varieties occurring less frequently. Carcinoma develops as a cauliflower or nodular growth from the vocal cords or ventricular bands, though occasionally other parts are first affected. The tumour, at first localised, tends gradually to

involve the whole of one side or more of the larynx. Owing to the infiltrating nature of the disease, the mobility of the corresponding vocal cord is often interfered with. Ulceration is a common occurrence, and may be followed by œdema, hæmorrhage, perichondritis, and necrosis of the cartilages. Carcinoma sometimes shows little tendency to infect the glands and other parts of the body, and sarcoma is even less liable to spread.

**SYMPTOMS.**—Hoarseness or aphonia is usually the first symptom, but dysphagia and dyspnoea may arise later on. Pain is more pronounced in this than in any other affection of the larynx, and may shoot to the ear. Ulceration is sometimes attended with great foetor of the breath.

**DIAGNOSIS.**—The laryngoscopic appearances are seldom characteristic, often closely resembling those presented by innocent growths, especially papillomata. The diagnostic importance of fixation of the corresponding vocal cord has been pointed out by Dr. Semon. Microscopical evidence of carcinoma or sarcoma may sometimes be detected in small portions of the tumour, removed by the laryngeal forceps, and is then of course conclusive.

The diagnosis of syphilis from malignant disease has been already discussed. Tuberculous lesions seldom present the appearance of a localised growth, and careful examination of the lungs and sputum will generally decide the matter. The age of the patient is of some importance, carcinoma being seldom observed before the age of forty.

**PROGNOSIS.**—The prognosis is unfavourable in all forms of carcinoma, but progress is least rapid in the scirrhous type. Sarcoma of the larynx seems to extend still more slowly.

**TREATMENT.**—Partial resection of the larynx has been successful in a few cases of early localised malignant disease, and is the only radical method that seems at present justifiable. Removal of a cancerous larynx was first practised by Billroth, and has since been performed by several Continental surgeons. The results of the operation have, however, been so unfavourable that it cannot be recommended. The obstruction to respiration may demand the performance of tracheotomy; and if the lower part of the trachea is free from disease, the operation will prolong life. Extirpation of a malignant growth by an endo-laryngeal operation should not be attempted.

Pain and dysphagia must be alleviated by the application of cocaine or morphine, as in cases of tuberculosis.

THOMAS J. WALKER.      PERCY KIDD.

**LATENT** (*lateo*, I lie hid).—This word is applied to cases of certain diseases in which their usual characteristic features are obscured and concealed; for example, *latent*

*pleurisy, latent scarlatina.* Symptoms are also said to be latent when they do not occur under circumstances in which they ought to appear. For instance, *cough* may be *latent* in certain cases of phthisis.

**LAVAGE** (Fr.).—SYNON.: Ger. *Magenspülung*.

**DEFINITION.**—A method of washing out the stomach, introduced by Kussmaul.

**DESCRIPTION.**—In order to perform lavage it is necessary to be provided with the following: (1) A soft flexible gastric catheter or stomach tube, about twenty inches in length; (2) a large-sized glass funnel; (3) about a yard of india-rubber tubing; and (4) some suitable lubricating material.

The glass funnel or receiver is affixed to the one end of the gastric catheter by means of the india-rubber tubing, which may be interrupted at one part by the insertion of a small piece of glass tubing, so that the observer can determine when the irrigation is complete by noticing that the returning fluid is quite clear. The free or distal end of the stomach tube is provided with good-sized lateral oval openings, placed a short distance from its rounded end.

Having lubricated the tube with glycerine, the practitioner introduces it into the pharynx with his right hand, and gently presses it down, whilst the patient, who is preferably in the sitting posture, performs the act of swallowing. The sensibility of the pharynx, if it be excessive, may be subdued by the usual appropriate measures.

To charge the apparatus, it is only necessary to fill the receiver with fluid, and, having filled it, to raise it above the patient's head, and to keep adding fluid until by its regurgitation we know that a sufficient quantity has been used. A syphon action can now be established by depressing the funnel. The stomach is thus emptied; and by repeatedly filling and emptying it as described, it can be thoroughly washed out. The repugnance some patients have to the use of the tube is generally soon overcome, and with a little practice a patient generally acquires the requisite manipulative skill to introduce the syphon tube himself; but it is necessary to encourage him to persevere with its use, which in many cases may necessarily be of long duration.

The fluid which may be employed in lavage may be either lukewarm water; 1 per cent. salt solution; 3 to 5 per cent. solution of bicarbonate of sodium; 3 per cent. solution of either boric acid or borax; or other such familiar substances possessed of anti-septic or antifermentative properties.

In the beginning, lavage may be resorted to once or twice daily, less frequently afterwards as the case improves.

**USES.**—Lavage has been employed for *diagnostic* purposes, as it affords a simple

and highly efficient means of removing the contents of the stomach for chemical investigation in certain gastric disorders.

Lavage is one of the most important among the numerous *therapeutic* measures which are at our command for stimulating the activity of the gastric glands in cases of chronic dyspepsia, especially in those instances which are associated with excessive secretion of mucus.

By its employment we may also increase peristaltic action, strengthen the enfeebled gastric muscular fibres in cases of insufficiency or atony, remove fermenting and stagnant gastric contents, thereby cleansing the gastric mucous membrane. With the improvement in the tone of the walls of the stomach, associated intestinal disorders such as constipation may also be overcome.

Amongst the diseases in which lavage has been successfully employed may be mentioned simple gastric dilatation, due either to perversion of function or to mechanical obstruction; chronic gastritis; and intractable cases of chronic dyspepsia. See STOMACH, Diseases of.

It should be remembered that there are certain possible dangers in connexion with lavage. The chief of these are injury to the œsophagus or to the gastric walls, with hæmorrhage, or perforation. Fatal syncope even has attended its use; but such cases are fortunately of extreme rarity.

JOHN HAROLD.

**LEAD, Poisoning by.**—SYNON.: Plum-bism; Fr. *Intoxication Saturnine*; Ger. *Bleivergiftung*.

Pure metallic lead has probably no injurious action on the system; but owing to the ease with which it oxidises and forms salts, all of which are poisonous, lead-poisoning is of common occurrence amongst all persons whose occupation brings them much in contact with metallic lead and its alloys; among those engaged in industries in which lead-salts are manufactured or largely employed; as well as from contamination, accidental or otherwise, of articles of food, drink, or luxury with lead or its compounds.

*Acute* poisoning with lead is not common, nor are any of the lead-salts actively poisonous. The acetate or 'sugar of lead' is popularly believed to be a virulent poison, but it is by no means so. There are very few fatal cases on record, even from swallowing quantities amounting to an ounce of the acetate, or its equivalent. The symptoms are in the main those of irritant poisoning, the chief difference being that constipation is the rule, and diarrhœa the exception.

The *chronic* form of poisoning by lead is of infinitely more importance and prevalence. The sources of it are extremely numerous and varied, and it is almost impossible to specify all individually. The following are

among the chief, classified according to the primary form of the poison.

**1. Metallic Lead, Poisoning by.**—Lead-poisoning occurs among lead-miners, metallurgists, and workers in lead or its alloys, such as plumbers, solderers, type-founders, compositors, and manufacturers of lead toys (toy soldiers). It has been observed also among fishmongers, from using lead counters; in pot-boys; as the result of packing articles of food or luxury in lead-foil; and from contamination of articles of drink by shot used for cleaning bottles, siphon soda-water bottles, &c.

Under this head may also be included the very frequent contamination of *drinking-water* by lead pipes and cisterns. Pure water, free from gases and excluded from contact with air, does not act upon lead, but in presence of air the lead becomes speedily acted on, and the water contaminated. An oxide of lead is formed, which, being partially soluble in water, allows the action to go on. From its solution the oxide is in great measure precipitated by carbonic acid, which is absorbed. The lead falls as an oxycarbonate, though the presence of carbonic acid in the water keeps a certain quantity in solution. The purer the water the more rapid the action. The presence of certain salts in the water considerably modifies its action on lead. Thus the nitrates, nitrites, and chlorides, by forming soluble compounds with lead, increase the solvent action of the water; and as these salts usually result from sewage-contamination, such water is rendered still more dangerous by passing through lead pipes. Other salts, usually found in spring and river waters, act as protectives by forming insoluble lead-compounds, which, being deposited as a crust in the interior of the pipe or cistern, prevent further action. Of this class are the sulphates, phosphates, and carbonates. Hence waters, unless containing much less than the average proportion of lime-salts, after a time cease to be contaminated to any great extent, though the presence of carbonic acid in the water renders a solution of the crust possible in some measure. Waters so deficient in lime-salts as the Loch Katrine water supplied to Glasgow cannot safely be conveyed in lead pipes. Galvanised iron, earthenware, or slate may in many cases be advantageously substituted for lead, if not possible in all. Lead covers to cisterns are very objectionable, as the water which rises by evaporation condenses and drops back contaminated. The electrolytic action of solders also helps to contaminate water with lead.

**2. Oxides of Lead, Poisoning by.**—The most important oxide of lead in this relation is litharge, or plumbic oxide (massicot), which is largely used in making glass and glazes for earthenware and iron. Those engaged in glass making and grinding, glaz-

ing pottery, &c., suffer. Owing to the solubility of lead-glazes, articles of food if acid may be contaminated, as by the artificial wines made in glazed jars decomposing the oxide by their fermentation. Litharge is also used for hair-dyes, japanning, &c., whence poisoning may result.

The 'red lead,' which is a mixture of lead oxides, has caused poisoning by being used to colour wafers, adulterate snuff, and make putty or cement for tanks, &c.

**3. Lead Salts, Poisoning by.**—Of these the most important in this relation is the carbonate, or 'white lead.' Those engaged in the manufacture, grinding, and packing of white lead are the most frequent sufferers, and also those who largely use it, such as painters, glaziers, plumbers, glazed card manufacturers, lacquerers, lace manufacturers, and those who apply it as a cosmetic (clowns, &c.). The acetate of lead has been used for correcting the acidity of wine and cider, and has been a frequent source of lead-poisoning. Poisoning has also occurred among seamstresses from using silk thread adulterated with it; in dye-works, where it is largely used; from using it as a hair-dye; and from long-continued medicinal administration. Lead colours, chromates, &c., have also caused poisoning, by being used to colour confectionery. (See a series of cases by Marshall, *Med. News*, 1887.) From these and similar sources lead may be introduced into the system by inhalation, ingestion into the stomach, and apparently also by cutaneous absorption.

**SYMPTOMS.**—The affections caused by lead are frequently termed *saturnine*, owing to lead being the symbol of Saturn, the malign planet of the astrologers. They are of a manifold character, and do not always occur in regular order or sequence.

**Saturnine Cachexia.**—As a rule, after long-continued introduction of lead into the system, a saturnine cachexia is developed, characterised by anæmia, an earthy or dull hue of the skin, digestive derangement, dryness of the mouth, frequently a styptic or sweetish astringent taste, coated tongue, and fetid breath. The teeth are discoloured, and frequently appear elongated from retraction of the gums. At the margin of the teeth and the gums a bluish or violet line is developed. This, which is regarded as the specially diagnostic indication of lead-poisoning, was first described by Burton, and is shown to be due to the formation of a lead sulphide in the parts.

**Lead Colic.**—**SYNON.**: *Colica Saturnina*; Fr. *Colique de Plomb*; Ger. *Bleikolik*.—Following the symptoms just described, but sometimes without marked prodromata, a very characteristic affection occurs, namely, lead colic. This is known by many other synonyms, of which the more common are Painter's Colic, Devonshire Colic, Colica

Pictonum, the last being derived from the inhabitants of Poitou, among whom in modern times it was first extensively prevalent owing to adulteration of wine with lead-salts.

Patients affected with lead colic exhibit a cachectic look, earthy hue, blue line on the gums, coated tongue, and fetid breath; and suffer from nausea and, occasionally, vomiting. The bowels are obstinately confined, or scanty hard motions are passed with difficulty. Paroxysms of excruciating pain occur in the abdomen, which feels hard, and is retracted in the region of the umbilicus. The pain is of a truly colicky nature, and is relieved by pressure. The countenance is anxious, and the skin is covered with cold perspiration. The respiration is shallow, and the pulse generally slow and hard, though this is not always the case. The urine is not infrequently almost or entirely suppressed.

**Lead Palsy.**—SYNON.: Fr. *Paralysie Saturnine*; Ger. *Bleilähmung*.—After repeated attacks of lead colic, or it may be after one, and sometimes without antecedent colic, various other affections occur. One of the most common of these is a form of paralysis termed 'lead palsy,' or, from its special features, 'dropped wrist.' The paralysis shows itself more particularly in the extensor muscles of the forearm, or region of distribution of the musculo-spiral nerve; and in consequence, when the arm is raised, the hand drops by its own weight. The paralysis generally commences in the extensor digitorum communis, and gradually extends to the other muscles supplied by the musculo-spiral, with the remarkable exception of the supinator longus. The paralysis does not necessarily confine itself to the forearm, for in advanced cases it may attack other muscles in the arm, the muscles of the leg, and the dorsal muscles; showing itself by preference in the extensor and abductor muscles of the body, and giving rise to a peculiar stooping, tottering gait. Aphonia occasionally results. The paralysed muscles undergo atrophy; while they lose their faradic, and in incurable cases also their galvanic, excitability. See NEURITIS, PERIPHERAL.

*Other phenomena.*—Tendinous swellings of an oval or elongated shape frequently form on the tendons at the back of the wrist, and contrast prominently with the atrophied muscles. Neuralgic pains in the muscles and joints are often complained of. In the more advanced cases various forms of encephalopathies occur. Epileptiform convulsions, with headache and optic neuritis, are common; and psychical affections are not infrequent, in the form of delirium, mania, or melancholia. Apparently in causal relation with lead-poisoning, disease of the kidneys and albuminuria may occur; and gout is frequently seen among those who work in

lead (see BRIGHT'S DISEASE; and GOUT). Abortion occurs to a large extent among women employed at white-lead works; according to Paul, in the proportion of 60 per cent. of those so employed.

The tendency is to recovery, if the cause of the symptoms is removed; but if not, the paralytic and other affections become incurable, and death occurs in a miserable state of cachexia.

**ANATOMICAL CHARACTERS.**—There are no very characteristic appearances of chronic lead-poisoning. Lead is found in almost every organ and tissue in the body, the greatest quantity, according to Heubel, being found in the bones; next in the kidneys, liver, brain, and spinal cord; and to a less extent in the muscles.

Among the appearances which have been described are constriction and apparent thickening of the muscular coat of the large intestine; and an atrophic condition of the intestinal mucous membrane has been found by some authors (Kussmaul and Maier). These authors have also found an increase of the connective tissue, and atrophy of the nervous tissue in the abdominal ganglia of the sympathetic. The paralysed muscles exhibit atrophic degeneration, with hyperplasia of the connective tissue, and disappearance of the striæ; while the nerve-trunks likewise exhibit various stages of inflammation or atrophy. We possess, as yet, no very reliable knowledge of the condition of the nerve-centres. Degeneration has been found in the nerve-cells of the brain, as well as in those of the anterior horns of the spinal cord.

The mode of action of lead in the causation of the various symptoms induced by it is not in all respects satisfactorily determined. The paralytic affections seem, however, to be essentially due to its action on the peripheral nerves, and the special action on extensors and abductors appears to be due to a special proclivity on their part to influences acting on the nervous system in general. The intramuscular nerves, in cases of lead-palsy, have been found in a state of inflammation; and a similar condition has been developed in animals as the result of chronic poisoning. The anterior cornua of the spinal cord, though sometimes affected, have been found normal in cases where the palsy has been well-marked, and the peripheral nerves in a state of inflammation.

**TREATMENT.**—In *acute* lead-poisoning from any cause the stomach must be emptied by the stomach-pump, or by emetics—of which sulphate of zinc is to be preferred. Solutions of the alkaline or earthy sulphates—of which the best is sulphate of magnesium—are indicated, with the view of forming the comparatively insoluble sulphate of lead, and expelling it from the intestines.

As regards *chronic* poisoning by lead, pro-

phylaxis is the most important consideration. The great principles in lead-works are the inculcation of cleanliness; avoiding eating with unwashed hands, or in working clothes, or in workshops; moist grinding; free ventilation; precautions against dust rising, or wearing of flannel respirators where it is unavoidable; and occasional doses of sulphate of magnesium, acidulated with sulphuric acid. Sulphuric-acid lemonade has been recommended as a drink.

Workmen who begin to show signs of lead-poisoning should at once give up the work, and take to some other employment. As regards water-contamination, what has already been said on this subject will suffice to indicate the prophylactic measures.

In the treatment of *lead colic* purgatives are indicated, and opium or belladonna may be necessary to allay the excruciating pain. The sulphate of magnesium is the best purgative. Iodide of potassium is generally given with the object of removing the lead from the system, and is on the whole satisfactory in its results. A combination of this iodide with sulphate of magnesium is very beneficial. Sulphur baths are also recommended.

The local *paralytic* affections require local in addition to the general treatment. Unless the muscles are in an advanced state of atrophy, and give no response to electrical stimulation, good results may be obtained from the use of the galvanic current applied to the muscles and to the musculo-spiral nerve. Faradisation has also been found beneficial, and is recommended by Duchenne, but the preference is to be given to the continuous current. D. FERRIER.

**LEAMINGTON**, in Warwickshire.—Sulphated common salt waters. See MINERAL WATERS.

**LEECHING**.—The local abstraction of blood by means of leeches. See BLOOD, Abstraction of; and DEPLETION.

**LENK**, in Switzerland.—Sulphur waters and climatic health resort. See MINERAL WATERS.

**LENS**, Diseases of.—See CATARACT.

**LENTIGO**.—A synonym for freckle. See FRECKLES.

**LEPOTHRIX** (*λεπίς* or *λέπος*, a scale; and *θρίξ*, a hair).—DEFINITION.—A term applied to a hair in which there is loosening and partial detachment of the overlapping edges of the scales of its cuticle.

Such hairs are usually met with in the axilla, and their peculiar conformation is attributable to the heat and dampness of that region, which causes maceration of the hair, particularly when it is of feeble structure. Sometimes the scales completely surround

the hair; very commonly one side of the shaft is more affected than the rest, and presents the appearance of a fringe; and not infrequently the scales are roughened with earthy and saline crusts deposited by the sweat.

TREATMENT.—The treatment most suitable for this evil is saponaceous ablution, followed by the use of a lotion composed of two to four drachms of oxide of zinc to half a pint of lime-water. ERASMUS WILSON.

**LEPRA**.—See LEPROSY.

**LEPROSY** (*λεπρός*, scaly).—SYNON. : *Lepra*; *Leontiasis* (old); *Elephantiasis Græcorum*; Fr. *Lèpre*; Ger. *Der Aussatz*.

DEFINITION.—A chronic general disease; characterised by structural changes in the skin, mucous membranes, and nerves; leading to the production of new morbid tissue, and giving rise to great disfigurement of the features and deformity of the extremities; and under certain conditions communicable from person to person by inoculation.

NOMENCLATURE.—The large number of names that have been applied to leprosy at different times has rendered the nomenclature difficult and obscure. Without going into the question of the early names, which are very numerous, we find that even the modern nomenclature is confusing. Willan and Bateman, and also Erasmus Wilson, following Hippocrates, have used the term 'lepra' to indicate the disease now universally known as psoriasis; while on the other hand the word 'elephantiasis' has been applied to two distinct diseases, namely, leprosy and the 'Barbadoes leg.' It will be well in future to reserve the word 'lepra' for leprosy, and to allow the other names that have been applied to drop out of use.

HISTORY.—All writers agree in regarding Egypt as the early home of leprosy, and it is highly probable that the Jews carried it with them when they migrated from that country to Palestine. The Biblical accounts of the disease, though highly interesting, are of no scientific value, as the writers evidently included more than one malady under the name which is translated 'leprosy.' In support of this view, it may be mentioned that the recovery of some of those afflicted with the disease is evidently assumed as highly probable, and provision is made for their re-admission into Jewish society. It is also quite evident that the leprosy of Naaman and Gehazi, mentioned in the Book of Kings, was a form of leucoderma, still very common in the East, especially in India, and known as 'white leprosy.' The earlier Greek medical writers give no clear account of leprosy; therefore we may assume that they were but little acquainted with it. Aretæus, however, who lived in the first century, gives an admirable description of it, which it would be difficult to improve upon in the present day.

During the Middle Ages leprosy spread continuously from Eastern to Western Europe, and it is generally believed that the Crusades contributed in no small degree to this result. It is, however, certain that the disease existed in Western Europe prior to the first Crusade. During the twelfth and thirteenth centuries leprosy increased to a terrible extent, especially in England, Italy, and France. Vellay, in his history of the latter country, says that Louis VIII. promulgated a code of laws in 1226 for the regulation of leper-hospitals in France, and that the number of these hospitals was computed at that time to be not less than two thousand. At a later period the number had increased, so that there was scarcely a town in the country unprovided with a leper-house. The late Sir James Simpson collected records from the *Monasticon Anglicanum* and other sources of no less than ninety-five religious hospitals for lepers, besides innumerable smaller pest-houses, nearly all of which were founded in Great Britain during the twelfth, thirteenth, and fourteenth centuries. In addition to these, there were at least fourteen houses of the first order in Ireland. The total number of similar institutions in Europe was estimated by Matthew Paris at nineteen thousand, but this probably included houses of the smaller class. It is interesting to note that no new leper-hospitals were built in England after the fifteenth century. The last one of any note was founded at Highgate in 1472, but long before that time many of the old leper-houses had fallen into disuse, and their revenues had become diverted to other purposes. On the Continent, Hensler has clearly demonstrated that towards the end of the fifteenth century scarcely a trace of tuberculated leprosy could be found in any of the more civilised parts of Europe. The disease has, however, continued to exist up to the present day in a few isolated spots, especially the west coast of Norway about Bergen, Iceland, and in some parts of Spain. There can be little doubt that the isolation of lepers during the twelfth, thirteenth, and fourteenth centuries contributed largely to the almost complete extinction of the disease in Europe in the following century.

**GEOGRAPHICAL DISTRIBUTION.**—There is no disease that has a wider geographical distribution than leprosy. It is found both in the northern and southern hemispheres, and in almost every latitude from the poles to the equator. It is, however, far more common in the tropical than in the temperate regions.

In Europe, leprosy has almost disappeared as an endemic disease. It is nevertheless met with in Western Norway, Iceland, Lapland, and the Russian shores of the Baltic, in certain parts of Spain and Portugal, and along the coasts of Provence and Nice; but in all these places it is confined to a small portion of the population, and, with the exception

perhaps of Spain, is generally dying out. It is also sparsely distributed in Greece and European Turkey and most of the islands of the Mediterranean. In addition to these districts, in which the disease may be said to be still endemic, we meet with a considerable number of imported cases scattered throughout Europe. This fact especially holds good with regard to England, and is obviously the result of her possession of India, Burmah, and the West Indian Islands.

In temperate North America leprosy is rare; it has, however, long been established amongst the French population of the Canadian province of New Brunswick, and it exists also at Cape Breton Island off Nova Scotia. Imported cases are, of course, occasionally met with just as they are in Europe.

In tropical America, both North and South, including the West Indies and the Bermudas, the disease is very common. Its presence in the Sandwich Islands is of comparatively recent date, and it is believed to have been introduced by the Chinese. Prior to the year 1848 it was unknown, yet by 1866 the number of lepers was reported to be 280, and by 1882 it had further increased to about 4,000. An asylum for lepers has been established in the island of Molokai, where there is now a settlement containing upwards of 1,000 sufferers. This extraordinary increase of leprosy in the Sandwich Islands since 1848 seems to point to some mode of propagation of the disease more rapid than inoculation.

With regard to Africa, it has already been stated that Egypt was the birthplace of leprosy, and it exists there to the present day; indeed, there is scarcely any part of Africa that is entirely free from the disease. It is, however, especially rife on the west coast, and is also met with in the Azores, Madeira, and the Canaries. On the east side it is very prevalent in Madagascar and the coast opposite. In Cape Colony it is met with not only among the native populations, but has also spread among the Dutch settlers. The writer has himself had under his care two Boers from the Transvaal suffering from it.

Of all quarters of the globe Asia contains the largest number of lepers. The disease is found in Syria, Persia, Turkey, and Arabia, and is especially common in Russia on the western shores of the Caspian. Its prevalence in India, Burmah, and the islands of the Indian Ocean is notorious; while Southern China may be regarded as a centre from which it has spread to many other parts of the world.

The history of leprosy in Australia is equally remarkable with that of the Sandwich Islands. Thirty years ago it was unknown among the European settlers, but has apparently been introduced chiefly, if not entirely, by the Chinese, from whom it has now spread to the Europeans. The writer has met with

three cases of the kind in employers of Chinese labour.

Taking a general view of the distribution of leprosy throughout the world, two questions especially suggest themselves: (1) Is it to any considerable extent on the increase? and (2) Is it capable of being transferred from one country to another by the exportation of lepers? With regard to the first of these questions, it seems probable that in most civilised countries the disease is on the decrease, but that here and there centres may be found where the reverse of this is the case. As to the second question, there can be no doubt whatever that the introduction of lepers amongst a new population leads to a dissemination of the malady.

**ETIOLOGY.**—During the Middle Ages the wildest notions were entertained as to the causes of leprosy. Some held that it was produced by an excess of animal food, while others believed that a vegetable diet had the chief share in its production. The combination, however, of milk and fish seems to have been considered as especially favourable to the development of the disease. Bernhard Gordon says: ‘Comedere lac et pisces in eadem mensa inducit lepram’ (*Lilium Medicinæ*). This curious and unaccountable belief, that such a terrible disease as leprosy could be produced by eating fish, has continued even down to the present time. It may be stated positively that neither climate, race, soil, nor food can by any possibility originate leprosy *de novo*, but that each and all may contribute to its perpetuation in those countries where it already exists, and that general improvement in hygiene will contribute not a little to the stamping out of the disease. Dismissing, then, these causes as at most only contributory to its perpetuation, it is necessary to consider two very important questions: (1) Is it hereditary? and (2) Is it contagious?

**Hereditary.**—Five-and-twenty years ago the hereditary nature of leprosy was generally considered one of the best-established facts with regard to the malady. Nevertheless, even at that time such able and distinguished men as Virchow and Kaposi pointed out that it could not be *hereditary* in the strict sense of the word—that it was never, for example, inherited in the same way as syphilis is; and that if the word ‘hereditary’ was applied at all, it could only be used in the sense of *predisposition*. The belief in the hereditary nature of leprosy is easily explained, and originated in the fact that it was often met with in different generations of the same family living together; and as its inoculability was overlooked, there seemed no other explanation for the facts observed. At the present time these facts can be easily accounted for on the supposition that it is inoculable, and hence a belief in its hereditary nature has almost entirely died out.

**Contagion.**—There is no disease about which so much has been written in former times as leprosy, and there is no disease the nature and history of which have been more thoroughly investigated at the present day. The only problem about which until lately some doubt remained was as to its contagious nature, and that problem is now being rapidly solved, if indeed the solution has not been already completed. The evidence in favour of contagion is of two kinds—(1) indirect or inferential; and (2) direct. The former is based on the fact that in recent times the disease has apparently been imported into new countries, formerly quite free, by the introduction of lepers; this is notably the case with regard to the Sandwich Islands and Australia. The more direct evidence is that of individual cases of inoculation; and, of the two kinds of evidence, the latter is of course the more satisfactory. About a quarter of a century ago the conclusions arrived at by a leprosy committee of the Royal College of Physicians, after a most careful investigation of the subject, were as follows: ‘The all but unanimous conviction of the most experienced observers in most parts of the world is quite opposed to the belief that leprosy is contagious, or communicable by proximity or contact with the diseased. The evidence derived from the experience of the attendants in leper asylums is especially conclusive upon this point. The few instances that have been reported in a contrary sense either rest on imperfect observation, or they are recorded with so little attention to the necessary details as not to affect the above conclusion. That leprosy is rarely if ever transmissible by sexual intercourse when one of the parties has no tendency whatever to the disease, is the opinion of the great majority of the respondents who have had the largest opportunities of observation.’

Those writers who have commented unfavourably on the above conclusion of the College of Physicians appear to have lost sight of the fact that the question of inoculability is hardly dealt with, the satisfactory evidence before the College at the time on that point being almost *nil*. All that is stated is that the disease is not contagious or communicable by proximity or contact with the diseased, and this statement in the ordinary acceptation of the words holds good at the present day. During the last five-and-twenty years much stronger evidence has been adduced in favour of the conclusion that the disease is communicable from person to person by inoculation. The following cases alone are sufficient to satisfy the minds of all unprejudiced persons on this point. In 1877, Dr. Hawtreys Benson published the following case: In 1872 he had shown to the Medical Society of Dublin a man who had contracted leprosy in the

West Indies, where he had lived twenty-two years. After remaining a certain time in the hospital he returned to his Irish home, and died after about a year and a half. During this latter period his brother slept in the same bed with him and wore his clothes. His brother, who had never left Ireland, except forty-six years before, when he had passed some time in England, became a leper and was shown in 1877 to the Medical Society of Dublin. There had been no other cases of leprosy known in the family. In the same year the writer published the following case: J. L., aged twenty, a native of Guernsey, was admitted into Middlesex Hospital in an advanced stage of tuberculated leprosy on July 12, 1877. His father, a native of Birmingham, was a soldier who had served in India, and died aged about fifty-five; his mother was a native of the island. His father when in India cohabited with a coloured woman, and died in Guernsey from a disease the description of which exactly corresponded with tuberculated leprosy. He had sores on his fingers and toes, enlargement of the nose and ears, and discoloration of the skin of the face. J. L. at the time that he was admitted into the hospital had suffered for five years from the disease, which had become fully developed before he left his native island; he had several brothers and sisters older than himself, all quite healthy. This case was considered at the time by those interested in the subject as strong evidence in favour of the inoculability of the disease. Dr. Gairdner in 1887 published in the *British Medical Journal* a very interesting account of the apparent transmission of the leprosy virus by vaccination. The history was briefly as follows: An English medical man living in a tropical island where leprosy is endemic, vaccinated his own son from a native child who turned out to be leprosy; from his own son he vaccinated another boy, the son of a Scotch sea-captain trading from Scotland to the island in question. Both his own son and the son of the sea-captain subsequently developed leprosy during childhood. It is a fair inference, but not of course a certainty, as the disease is endemic in the island referred to, that the leprosy was transmitted by inoculation.

The conclusions to be derived from the well-known experiment by Dr. Arning in Hawaii of inoculating a criminal with leprosy virus, whose sentence of death was commuted on the condition that he should submit to the operation, were somewhat vitiated by the surroundings and previous history of the individual. It is true that he developed leprosy after, and probably, but not necessarily, in consequence of the inoculation.

The present leprosy committee of the College of Physicians, having these and many other cases before them, thought it

right to slightly modify the conclusions at which their predecessors had arrived; and they pointed out that, though leprosy is not contagious in the common sense of that term, yet that it is probably so in a minor degree, and they compare it in this respect with syphilis.

Taking all points into consideration, it can hardly be doubted that this conclusion is the right one, and that leprosy, at all events in its later stages, when open sores are present, may become a source of danger to others. On the other hand, in the earlier stages of the disease, it seems highly probable that it is not contagious even in a minor degree.

The oft-quoted history of the Sandwich Islands, and also the accounts from certain districts in Spain, where the disease has spread in modern times with great rapidity over a limited area, are facts not easily explained on the assumption that the disease is propagated only by inoculation, the time occupied being apparently too short. To meet this difficulty the writer suggested in his *Goulstonian Lectures* in 1872 the hypothesis that the leprosy poison (the bacillus was not then discovered) might be conveyed, like that of typhoid fever, through water or uncooked food: for example, a leper with open sores might bathe or wash his clothes in a tank used by others for drinking purposes, and thus the germs of the disease would be conveyed to others. This hypothesis involves, however, the supposition that the leprosy germs can exist and multiply apart from the human body in the same way that those of typhoid fever and cholera do, and at present there is no evidence to show that this is the case.

ANATOMICAL CHARACTERS.—A general description of the more obvious visible changes produced in the organism by leprosy will be presently given. These changes are for the most part confined to the skin, mucous membranes, lymphatic glands, cutaneous nerves, and other superficial structures, which all become gradually but irregularly infiltrated and thickened by the new leprosy tissue, the deeper structures of the body remaining unaffected. Dr. Thin says: 'If a vertical section is made from a leprome of the skin, and examined under the microscope, the chief morbid change found is that large clusters of cells have taken the place of bundles of connective tissue which are broken down. Very often, if the leprome is not advanced towards the ulcerative stage, a thin band of sound, unbroken connective tissue is observed between the rete mucosum and the affected part of the corium, which may be found the seat of a dense infiltration of cells. The cells are very similar to the so-called "granulation cells" observed in lupus and in syphilis. In the centre of a group of such cells in a lupus tissue one or more sections of enlarged blood-vessels filled

with granular material may be discovered—the so-called “giant cells” of authors. “Giant cells” are referred to by several authors in connexion with leprosy, but in a very large number of sections which have been examined by us we have not observed the characteristic appearances described by ourselves and others in lupus vulgaris. The cells in leprosy vary in size from that of an ordinary white blood-corpuscle to double and even four times that size and more. If specially stained for the purpose, it is seen that these cells contain leprosy bacilli, the smallest of them containing a few, the largest of them masses of bacilli matted together.’

The leprosy bacillus was first discovered by Hansen. It bears a very close resemblance to the tubercle bacillus, so close, indeed, that it is exceedingly difficult to distinguish the one from the other. It has even been suggested that the leprosy and the tubercle bacillus are identical; if this, however, were the case, the conclusion would be unavoidable that the bacillus has very little to do with either the one disease or the other, a conclusion which would not be accepted for a moment by bacteriologists. The bacilli are found in great abundance in the discharge from open leprosy sores.

**SYMPTOMS.**—Three varieties of leprosy have usually been described by authors—(1) *tuberculated*; (2) *anæsthetic* or ‘mutilating;’ and (3) *macular* leprosy. There is, however, no essential difference between these three forms, the last being simply an early stage of the other two, while tuberculated leprosy differs from mutilating leprosy chiefly, if not entirely, in respect of the tissues involved.

The general symptoms of the disease belong to all its forms, but they vary much in severity in different cases. The invasion of the disease is usually slow and insidious; in exceptional cases, however, the onset is acute, and the symptoms develop with great rapidity. The malady is rarely met with in infants under the age of two or three years; and is equally common in males and females. The period of incubation is very uncertain, and may generally be reckoned by years rather than by months or days. The early symptoms of the malady consist of general constitutional disturbance, debility, mental depression, loss of appetite, a sense of chilliness, and slight recurrent febrile attacks. All these symptoms may subside for a time, but sooner or later they return; associated with them we meet with slight alterations in the pigmentation of the skin, and temporarily impaired sensibility over limited areas. Occasionally the development of scattered blebs resembling those of pemphigus is met with as an early feature, especially when anæsthesia is present. Blebs also appear sometimes at a later stage.

*Tuberculated leprosy* is the most severe form of the disease. It begins with the usual symptoms just indicated; after these have lasted with more or less intermission for months or years, the first characteristic changes in the skin appear. These consist of spots resembling some form of erythema, which develop during one of the febrile attacks. They are of a dull brown or reddish brown colour, tender to the touch, often slightly swollen, and varying in size from half-a-crown to that of the palm of the hand, or larger; they partly disappear on pressure, showing their hyperæmic character. These spots are always roughly symmetrical, and are most frequently seen on the extensor surfaces of the extremities, but sometimes on the face and trunk. After a short time their hyperæmic nature disappears, leaving a patch of skin discoloured, and perhaps a little thickened. In dark races, portions of these patches are often paler than the normal skin, giving a somewhat piebald appearance; but in Europeans the skin is for the most part darker than normal, the idea that lepers are usually white being a common popular error. Some of the spots after a time entirely disappear, while others lead more or less to slight permanent changes. Associated with these changes in the skin we often find the superficial nerves affected, so that patches of partially anæsthetic skin are produced. These are for the most part only temporary, the skin sooner or later resuming its normal sensibility.

All the above-mentioned symptoms may reappear and subside over and over again without much permanent alteration in the skin, except perhaps a little discoloration; but ere long we notice a slight permanent change in the face, which is not easily mistaken. The skin a little below the eyes looks rather swollen and puckered, the nose appears somewhat thickened, the patient at the same time complaining of not being able to breathe quite freely through it, and the tone of voice is a little altered in consequence; he speaks, as we say, through the nose. As a further and later change, characteristic tubercles develop in the skin, especially on the face and hands; these swellings are tender on pressure, and produce much thickening of the tissues and consequent alteration of the features. The forehead becomes tuberculated and its wrinkles deepened, the eyebrows somewhat overhanging, which gives a peculiar heavy and leonine expression to the countenance; the hair of the eyebrows is quickly lost; the nose becomes tumid with nodules; the cheeks are irregularly thickened and puckered; the lips hard, swollen, and sometimes everted; the chin is nodulated; and the ears, greatly enlarged, stand out stiffly from the side of the head; the whole appearance is hideous and revolting. One striking effect of the deepening of the furrows and wrinkles

of the face is to make young people look prematurely old. Coincidentally with these changes in the face, the dorsal aspect of the hands and feet may be similarly affected: the skin becomes brown, and the fingers, greatly enlarged, stand stiffly apart; the nails become dull, dry, and fissured. Some of the tubercles of leprous tissue shrink, while others ulcerate and leave open sores, very difficult to heal. Sooner or later the mucous membrane of the mouth, tongue, and throat becomes altered and thickened, and the voice assumes a hoarse whisper, which is very characteristic of the disease. The eyes also suffer; the cornea becomes opaque, and a partial or complete loss of sight is the consequence. The invasion of the cornea by leprous infiltrations constitutes one of the most common and hopeless features of the malady. Together with these visible changes in the skin and mucous membrane we find alterations occurring in the nerves, and leading to the formation of patches of permanently anæsthetic skin, which vary very much in size, and are met with chiefly on the forearms, hands and feet, and legs below the knee, more rarely on the trunk. In fact, in ordinary cases, the new-growth and structural changes are confined to the face, ears, hands, feet, forearms, and legs, the mucous membrane of the nose, mouth, and throat, and the nerves of the extremities. The ulnar nerve is particularly liable to be affected, and a nodulous swelling of its trunk may be easily felt just above the point where it crosses the elbow-joint. Gradually, though not uniformly, these symptoms increase, the health becomes greatly enfeebled, the temperature is often slightly below normal, the vital powers are exhausted. Sooner or later, some internal complication arises, and the unhappy victim is carried off after years of suffering, by some disease of the lungs or kidneys.

*Anæsthetic or mutilating leprosy* differs in no essential from the tuberculated form, and for the most part we meet with what are called mixed cases—that is, tuberculated leprosy in which the nerve-lesion forms a marked feature, the most important point of distinction being that when the nerve-lesion is considerable, a destruction or mutilation of the fingers and toes is more liable to occur. The distal phalanges are especially apt to be affected and the bones destroyed, so that we sometimes see the nail transferred from the distal to the second, or even to the proximal phalanx, the intervening bones being lost. In other cases, great atrophy and stiffening of the fingers occur. The final falling off of the fingers and toes is for the most part unattended with pain. In other respects, mutilating runs much the same course as tuberculated leprosy, though it is often more protracted.

**DIAGNOSIS.**—Fully developed lepra is distinguished by such characteristic symptoms

that it cannot be easily mistaken for any other disease. This is not, however, the case in an earlier stage, when the symptoms are but slightly marked. The skin may present a perfectly healthy appearance, and the evidence of disease may be limited to some slight anæsthesia of the extremities. Occasionally an eruption of blebs has been mistaken for the onset of pemphigus. Leprosy is, however, far more often mistaken for syphilis than for any other disease. This is not to be wondered at, as the two have an undoubted resemblance, and the differential diagnosis is rendered more difficult when the patient has previously suffered from syphilis. The loss of sensibility in the skin, which is so common in the former disease, will serve as a valuable aid in the differential diagnosis. Later on, the tubercles of lepra may be distinguished from those of syphilis by their much slower development, and their less tendency to soften.

Leucoderma, in which the skin assumes a somewhat piebald appearance, has often been mistaken for an early stage of lepra. This mistake is more liable to occur amongst the dark Oriental races than amongst Europeans.

Lastly, the writer has known this serious disease mistaken for some harmless form of erythema.

**PROGNOSIS AND TREATMENT.**—The writer has had more or less under his care, during the last twenty years, about forty cases of leprosy, of which some have been watched for many years. The number is absolutely small, though relatively large for a country in which the disease is not endemic. Out of this number, in two only has the disease apparently died out. One of the two cases referred to, when last heard of, had remained free from any return of the disease for fourteen years; the other for a somewhat shorter period. Both of the cases had persistently taken chaalmoogra oil—a fact which may be taken for what it is worth; one remained in England, and the other in a healthy part of India. The writer has used chaalmoogra oil internally in a considerable number of cases, and with apparent benefit in most, if not all. The oil appears to be more easily borne by lepers than by other people; but, in order to produce any marked effect, the treatment must be continued for years. He is strongly impressed with the belief that the application of external remedies produces no permanent good result, although it may temporarily relieve some of the symptoms. Of the many other drugs that he has tried, Gurjun oil and quinine are the only ones that have apparently done good, the latter probably only as a suitable tonic. From the comparative rarity of leprosy in this country, it is very difficult to obtain a sufficient number of cases on which to base our conclusions as to the effect of treatment, and therefore the results of any experiments in the use of

drugs must be received with caution. There is another fact which must be taken into consideration, namely, that leprosy is liable—at least in healthy climates—to long periods of comparative rest or subsidence, quite apart from any special treatment. It is, in short, one of the features of the disease that it does not progress uniformly. Sometimes it appears for a time as if cured, and then breaks out again without any apparent exciting cause. It is very necessary to bear these facts in mind, because those who are unacquainted with the peculiarities of the disease are apt to attribute to remedies what really is the natural course of the malady, under the influence of a mild and healthy climate.

In the later stages of the disease, it is well to bear in mind that those who dress the open sores of lepers should be careful not to allow the discharge to get into any cuts or wounds in their hands. Scrupulous cleanliness and disinfecting dressings are also advisable.

With regard to the segregation of lepers in those countries where the disease is endemic, it is highly desirable that leper-hospitals should be provided, and the lepers encouraged as much as possible to enter them. This would not only be an advantage to the lepers themselves, but of even greater benefit to the community at large.

ROBERT LIVEING.

**LEPTOMENINGITIS** (*λεπτός*, delicate or thin; and *meningitis*).—A term signifying inflammation of the pia mater. By its use, in association with arachnitis and pachymeningitis, we are enabled accurately to indicate the precise seat of inflammation involving the meninges of the brain or spinal cord. There is a practical convenience, warranted by pathological facts, in retaining the term 'arachnitis,' although anatomists are not now disposed, as they were formerly, to believe in the existence of an arachnoid membrane, distinct externally from the dura mater, and internally from the pia mater. See MENINGES, Diseases of.

**LEPTOTHRIX** (*λεπτός*, delicate or slender; and *θρίξ*, a filament or hair).—*Leptothrix buccalis* is a name assigned by Robin to certain vegetable parasites or minute filaments, which can be recognised, by means of the microscope, amongst the epithelial scales of the tongue or other parts of the mouth; and especially between the teeth, or in the hollows of decayed teeth. They occur in healthy persons, as well as in the sick, and have in this situation really no pathological signification.

Dermatologists also employ the term 'leptothrix' to indicate a morbid thinness and weakness of the hair. See LEPTOTHRIX.

**LESION** (*lædo*, I hurt).—This word originally signified a hurt or an injury; but its use is now extended to comprehend all organic changes of a morbid character, affecting an organ or tissue.

**LETHARGY** (*λήθη*, oblivion; and *ἀργία*, idleness).—A disorder of consciousness, which consists of prolonged and profound sleep, from which the patient may be momentarily aroused, but into which he falls off again immediately. One form of it corresponds with the final stage of hypnotism. See CONSCIOUSNESS, Disorders of; and TRANCE.

**LEUCE** (*λευκός*, white).—This term has been applied to blotches in the skin of a white colour; and hence it has alternately been confounded with *lepra alphoides*, with vitiligo, and with the leucodermic blotches of leprosy. It, however, seems more than probable that the pathological condition intended to be signified by this word is a circumscribed scleriosis, namely, that which we at present term *morphæa alba*. See SCLERODERMA.

**LEUCIN** (*λευκός*, white).—SYNON.: Fr. *Leucine*; Ger. *Leucin*.

Leucin or amido-caproic acid is a decomposition-product of the complex proteid molecules, when these are acted upon by strong acids or alkalis, or when they are submitted to the prolonged action of the pancreatic ferment trypsin. Its chemical composition is represented by the formula  $C_6H_{13}NO_2$ , thus showing it to be a compound of ammonia with a fatty acid. It is a product of intestinal digestion, but does not occur normally in the fæces or urine.

As the internal administration of leucin increases the amount of urea excreted, physiologists now regard it as an antecedent of urea in the decomposition of proteids; the transformation in all probability occurring in the liver.

**CHARACTERS.**—Pure leucin is soluble in water, slightly in alcohol, insoluble in ether.

*Microscopically*, leucin, as it occurs in the urine, appears as oil-like, highly refracting, laminated, crystalline, globular masses, ob-

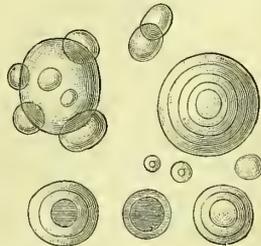


FIG. 66.—Crystals of Leucin. × 200.

scurely radiating; but, when pure, leucin forms white, glistening, flat crystals (fig. 66). The microscopic test is not a thoroughly reliable one, hence we must have recourse to the following method: Free the urine of

albuminous substances, and precipitate it with basic lead acetate; filter, and pass sulphuretted hydrogen gas through the filtrate to remove the excess of lead; filter, and evaporate the filtrate to dryness. Extract the residue with boiling alcohol; filter, and evaporate the filtrate to a syrupy consistence, when, if present, leucin will crystallise out.

*Chemically*, leucin is to be detected by the following characteristic reaction: A little urine mixed with nitric acid is carefully evaporated to dryness on a platinum foil; a nearly imperceptible colourless residue is left. If caustic soda is added to this residue, and heat applied, the leucin will be dissolved, and according to its degree of purity the solution thus formed will be either colourless or of a yellowish-brown colour; and on concentrating the fluid by heat on platinum foil, an oily drop is formed, which rolls about on the platinum foil.

**PATHOLOGICAL SIGNIFICANCE.**—Leucin is a constituent of many organs of the body, more especially of the glandular tissues. It is found in the liver, kidneys, pancreas, the thymus, thyroid, salivary, and lymphatic glands, and in the brain. Leucin and tyrosin have been detected in the urine only in cases of acute yellow atrophy of the liver, and in the atrophied hepatic cells in the same disease (see LIVER, Atrophy of, Acute Yellow). Tyrosin is invariably found associated with leucin. See TYROSIN.

JOHN HAROLD.

**LEUCOCYTHÆMIA** (λευκός, white; κύτος, a cell; and αίμα, blood).—**SYNON.**: *Leukæmia* (Virchow); *Splenopathia leucocythæmia* (Huss); *Fr. Leucocythémie*; *Diathèse lymphogène à forme leucémique* (Jaccoud); *Ger. Leucocythämie*.

**DEFINITION.**—A chronic disease, in which there is a considerable and permanent increase in the number of the pale blood-corpuscles; usually associated with enlargement of the spleen, sometimes also with that of the lymphatic glands, and with disease of the medulla of bone.

The term 'leucocythæmia,' proposed by the late Dr. Hughes Bennett, is a convenient and significant designation, the essential feature of the disease being the excessive proportion of leucocytes in the blood. The shorter word 'leukæmia,' proposed by Virchow, and much used, is less obviously accurate, since the blood (as Parkes urged), although appreciably paler than normal, is not white.

Excess of leucocytes in the blood, slight or transient, is known as 'leucocytosis,' and is met with in many morbid states. Permanent excess, sometimes considerable, but rarely very great, also occurs, associated with a primary enlargement of the lymphatic glands—'lymphatic leucocythæmia.' These cases differ in many important respects from the cases of leucocythæmia associated with

primary enlargement of the spleen; so that it is most convenient to describe them in connexion with lymphadenoma, and to consider here only cases of splenic leucocythæmia. See LYMPHADENOMA.

**HISTORY.**—Pallor of the blood, as if pus were mixed with it, was noted by Bichat in the beginning of this century; and the combination of this appearance with enlargement of the spleen was observed by Velpeau in 1827. The dependence of this alteration in the blood on an excess of pale corpuscles was described by Donné in 1844, and interpreted by him as due to imperfect transformation of white into red corpuscles. In 1845, two cases of the disease were published together, the one by Dr. Craigie, the other by Dr. Hughes Bennett; and to the latter appears to belong the credit of recognising the salient features of the affection as a distinct malady. A month later, however, Virchow published another case, independently and admirably worked out. In all these cases the change in the blood was only recognised after death. It was first observed during life in 1846, by Dr. H. W. Fuller, and subsequently by Dr. Walshe. In Germany the first case was diagnosed during life, by Vogel, in 1848. Since then numerous cases and descriptions of the disease have been published, of which the more important are those of Virchow, Hughes Bennett, Vidal, Huss, Ehrlich, and Mosler.

**ÆTIOLOGY.**—In only a small proportion of cases of leucocythæmia can any causation be traced. Race, as such, seems to be without influence. Heredity has only been traced, as a history of splenic disease in ancestors or collaterals, in one or two isolated instances. The disease is twice as frequent in men as in women. It may occur at all ages. It is very rare under the age of ten, and the numbers gradually rise, taking both sexes, to the decade between thirty and forty, when nearly one-third of the total occur (46 out of 154 cases). After forty they fall in each decennial period. In females, however, the maximum is reached in the period between forty and fifty; and of eleven cases over sixty collected by Dr. Gowers, only one was in a woman. Position in life appears to exercise no influence on the occurrence of the disease. Depressing influences, inanition, over-exertion, and especially depressing mental emotion, are antecedents which have been occasionally noted. Sexual processes, in women, appear to have a distinct influence. The disease has been seen to be most frequent, in them, during the climacteric decade, and practically to cease when the menstrual epoch is over. In some cases the disease has commenced during pregnancy; in a larger number it has succeeded parturition. Injury to the spleen seemed, in one or two recorded cases, to be the cause of the disease. Small-pox, typhoid fever, acute rheumatism,

pneumonia, and syphilis have been supposed to be causes of the affection, but the ætiological relation is doubtful. Of all antecedent conditions, intermittent fever is incomparably the most frequent. In one-fourth of the total number of cases (150) analysed by Dr. Gowers, there was a history either of ague or of residence in an ague district. The interval between the malarial affections and the disease varied from a few months to thirty years. The fact that, in many cases, a long period elapsed, and that the attacks of ague, in some instances, were trifling, makes it probable that, in the cases in which the patients had merely lived in an ague district, the malarial influence, which did not cause ague, led to morbid changes which eventuated in the leucocythæmia. One patient under Dr. Gowers's care, a middle-aged woman, had lived in a malarial district only during the first few years of her life, but, shortly before her birth, her mother had suffered from an attack of ague.

**ANATOMICAL CHARACTERS.**—*Blood.*—The blood, as seen after death or during life, is paler than normal, and may even be greyish-red in colour. In extreme cases coagulation is imperfect; a grunous chocolate-brown mass results. After defibrination three layers form—red corpuscles, pale corpuscles, and serum. Under the microscope the pale corpuscles are seen to be in great excess; instead of two or three per field, as in

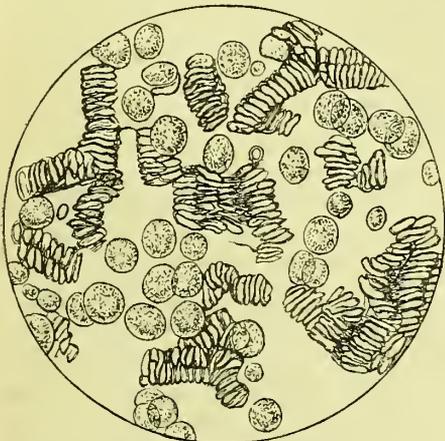


FIG. 67.—Blood in Leucocythæmia.  $\times 600$ .

the normal, there may be several hundreds. Enumeration (*see* HÆMACYTOTEMETER) shows that not only are the white corpuscles increased, but the red are lessened out of proportion to the increase in the white, so that the total number of corpuscles is always diminished. Instead of the normal 5,000,000 per cubic millimeter, there may be only 2,500,000 or even 1,150,000 (50 and 23 per cent. of the normal). The proportion between the white and red varies, being 1-20, 1-10,

1-5, 1-2, or 1-1; or the white may be the more numerous. The apparent is greater than the real excess of white, in consequence of the closer contact of the red. It was proposed by Magnus Huss to regard as leucocythæmia only those cases in which the proportion is greater than 1 to 20, and the rule has been largely followed; but it must be remembered that in commencing cases the proportion may be smaller than this. The greatest change hitherto recorded was a reduction of the red from 5,000,000 to 470,000 per cubic millimeter (9 per cent. of the normal), and an increase of the white to 680,000 per cubic millimeter instead of 15,000, the normal average. The pale corpuscles may be of normal size; but usually some are large, and often many are smaller than normal (the *globulins* of Donné), especially when the lymphatic glands are affected. Reagents bring into view one to four nuclei. Some of the corpuscles present obvious fatty degeneration. The red corpuscles are either normal in appearance, or present great variety in shape and size (*poikilocytosis*). They are sometimes unduly pale. Nucleated coloured corpuscles have been seen in a few cases, believed to be intermediate forms between the white and red corpuscles. The amount of hæmoglobin is less than that which corresponds to the number of red corpuscles present. The specific gravity of the blood is lessened from 1,055, the mean in health, to an average of 1,042; the change being due to an increased proportion of water, from 790 parts per 1,000 in health to 840 in leucocythæmia. The fat and fibrin are increased, and the latter sometimes presents a peculiar granular appearance. The iron is lessened. Abnormal constituents have also been found in the blood, such as the albumin of Reichardt, mucin, a substance analogous to glutin, hypoxanthin, lactic and formic acids. Minute octahedral crystals have been found in the blood and in many organs after death, about  $\cdot 016$  mm. in length. Their nature is uncertain. Since they have been found only after death, it is conjectured that the substance of which they consist is held dissolved during life. They are not peculiar to this disease.

*Organs.*—The *spleen* is always enlarged, sometimes extremely. Its weight varies from twice to fifty times the normal—1 lb. to 18 lbs. The average of 72 cases analysed was about 6 lbs. The average length is nearly 12 inches. The enlargement is commonly uniform; the shape of the organ being preserved. The surface is smooth, but often presents traces of local peritonitis, in the form of yellowish opaque patches. It is frequently adherent to the diaphragm, omentum, abdominal wall, intestines, or liver. Its consistence is usually increased, rarely diminished. The cut surface is smooth, and yields comparatively little blood; it is

brownish-red, or even brownish-yellow, mottled with paler lines, due to thickened trabeculæ. The Malpighian follicles are not usually conspicuous. In cases which begin with enlargement of the lymphatic glands, and which are really cases of primary lymphadenoma, the Malpighian follicles may be so enlarged as to constitute small growths. There is rarely an area of distinct softening. Wedge-shaped, yellowish-white, caseous portions are frequent, and are evidently infarcts; when recent they are deep red. The histological change in the organ is an overgrowth of the splenic pulp; the trabecular tissue is increased; and so also is the retiform tissue of nucleated fibres and cells, among which the lymphoid corpuscles lie. The Malpighian bodies may not be discoverable with the microscope, or they may be found to have undergone fatty or lardaceous degeneration. The infarct-like masses present the splenic tissue-elements in a state of fatty degeneration. In many cases the crystals, already described, have been found in the spleen in great abundance. This organ has been found by analysis to contain gluten, glycocoll, hypoxanthin, xanthin, leucim, and tyrosin.

The *lymphatic glands*, some or many, are enlarged in one-third of the cases of primarily splenic leucocythæmia (51 out of 157 cases). The order in which the several groups are affected is, beginning with the most frequent, the mesenteric, cervical, inguinal, axillary, retro-peritoneal, thoracic, portal, and iliac. In only five cases was the change universal. The enlargement is not considerable, the individual glands rarely attaining the size of a walnut. They are usually smooth, soft, grey, or reddish-white on section; rarely caseating or suppurating; sometimes presenting hæmorrhagic extravasations. Their minute structure differs little from the normal. Lymphoid corpuscles lie in an adenoid reticulum, which is rarely increased, as it is in lymphadenoma.

With respect to the *alimentary canal*, the gums are occasionally swollen and ulcerated, the swelling being due to infiltration of the gum with leucocytes, or to an actual lymphoid growth. The tonsils and follicles of the tongue are sometimes enlarged by lymphoid growth; and there may be a similar change, usually slight, in the lymphoid tissue of the wall of the stomach, and much more considerable in the solitary and agminated glands of the small intestine. These growths frequently ulcerate. Similar changes are also found in the large intestine. The *peritoneum* sometimes presents similar growths. The *liver* is enlarged in at least two-thirds of the cases, varying in weight from 5 lbs. to 14 lbs. In some cases of slight enlargement no structural change beyond congestion has been found; frequently, however, there are disseminated lymphoid growths, minute, greyish-white, commonly interlobular in position,

often surrounding branches of the portal vein. The capillaries are always distended with pale corpuscles. Fatty degeneration of the liver-cells is also common. The *Kidneys* are abnormal in at least one-half of the cases. They may be simply pale, from anæmia; pale and enlarged, from granular degeneration of the cells and distension of the capillaries with leucocytes; or they may present minute growths similar to those in the liver, and situated between the tubuli of the cortex, especially near the glomeruli. Marked fatty degeneration of the kidney has also been found. The *suprarenal bodies* have been found diseased in several cases, and in one or two there was bronzing of the skin. The *thymus* and *thyroid* glands have also been found enlarged. The *heart*, as in other cases of intense anæmia, may present granular and fatty degeneration. Extravasations of blood have been found beneath the endo- and pericardium. The capillaries of the heart are often distended with leucocytes. Pericardial effusion is common. The *lungs* may present simply distension of capillaries, or hæmorrhagic infarcts, or actual growths, similar in structure to those found elsewhere. These commence at the bronchi and infiltrate adjacent tissues. Rarely they may break down and form cavities. Pleural effusion is very common, and lymphoid growths have been found on the membrane. In the *brain*, hæmorrhages, usually multiple, constitute the most frequent change. Minute growths in the membranes, distension of the meningeal vessels with pale blood, and their obstruction by masses of leucocytes (Bastian), have also been recorded. The *skin* is, in rare cases, the seat of growths. The *bones* have been found diseased in many cases (Ranvier, Neumann, &c.) The marrow is grey or reddish-grey, diffuent, presenting lymphoid cells and blood-corpuscles. Sometimes cells intermediate between white and red corpuscles have been found in it. The vessels are fewer than normal. The change may be found in all the bones, most marked in those which possess most spongy tissue, as the ribs and vertebræ, but also considerable in the long bones. Externally the bones may be normal or enlarged. The compact substance may be reduced in thickness, and even perforated (Mosler). The *retina* is frequently diseased. Hæmorrhages may be found, especially in the nerve-fibre layers, and adjacent to them the retinal elements may be degenerated. The capillary vessels are filled with pale corpuscles, and actual lymphoid growths have also been found.

**SYMPTOMS.**—Of the early symptoms of leucocythæmia, the most frequent are those due to the splenic enlargement, namely, abdominal fulness, pain, or an actual tumour. Next in frequency is weakness. Hæmorrhage, especially epistaxis, often occurs early, but rarely before other indications of ill-health.

The change in the blood causes pallor of skin and mucous membranes, shortness of breath, and all the indications of anæmia. But in many cases the face retains a good colour for some time in spite of marked excess of white corpuscles. The altered characters of the blood, readily observable during life, have been already described. The temperature is frequently, but not invariably, raised. It is commonly higher in the evening than in the morning; the evening rise usually reaching 101°–104° F. The morning fall may be considerable or slight. Sometimes periods of considerable pyrexia alternate with others in which there is little fever. The cases in which there is most pyrexia are usually, but not always, those of most rapid course. The enlargement of the spleen presents the typical characters of a splenic tumour. It may occupy the whole left half of the abdomen, extend even beyond the middle line, descend into the iliac fossa, and even into the pelvis, so as to be perceptible by vaginal examination (Spencer Wells). It may vary in size from time to time. Friction may sometimes, and a *bruit de souffle* rarely, be heard over it. It usually causes an unpleasant sense of distension, is often tender, and sometimes is the seat of spontaneous pain. By its pressure it may raise the heart, cause considerable dyspnoea, and interfere greatly with the functions of the stomach. The distension of the abdominal wall may cause lines, similar to those of pregnancy, to appear over the spleen, and their position is sometimes distinctly determined by the course of vessels. The enlargement of the glands is rarely sufficient to give rise to other symptoms than the obtrusive evidence of their presence. The glands thus present, in this disease, a marked contrast to their condition in lymphadenoma. They often lessen in size before death. The alteration in the bones is usually unattended by symptoms. Rarely they become distinctly enlarged and tender. The action of the heart is disturbed by both the anæmia and the displacement. The pulse is frequent; and palpitation is common. The circulation is impeded; effusion of serum into the cellular tissue of the body is almost invariable in the later stages of the disease. Slight ascites is frequent, and great effusion is sometimes due to the pressure of enlarged glands on the portal vein. Hydrothorax is also common. The most striking circulatory symptom is hæmorrhage, which occurs in a large proportion of cases, most frequently from the nose, and less frequently from the bowels, stomach, lungs, uterus, into the skin, brain, joints, cellular tissue, or peritoneum. The hæmorrhagic tendency is so great that slight injuries may give rise to serious loss of blood; the extraction of a tooth, or the puncture for paracentesis, has thus led to death; while most cases in which excision of the spleen

has been attempted have been fatal from the same cause. The respiration is usually interfered with, partly from the splenic enlargement, and partly from the anæmia; that due to the former is increased by the recumbent posture. The anæmic dyspnoea may be unnoticed when the patient is at rest, although any considerable movement causes an agony of breathlessness. The dyspnoea is also increased by changes in the lung, bronchial catarrh, and the frequent pleural effusion in the later stages. Cough is frequent, and may be an early symptom of the disease. The change in the gums, already described, may lead to ulceration—the ‘leukæmic stomatitis’ of Mosler; but it is more rare in this disease than in lymphadenoma. The gastric functions are chiefly interfered with by the pressure of the spleen, which causes dyspepsia, and often vomiting. Diarrhœa is extremely common, and may be accompanied by hæmorrhage. The enlargement of the liver may be recognised during life, but rarely gives rise to subjective symptoms. Jaundice only results from compression of the bile-ducts by enlarged glands. A yellowish tint of skin is, it may be remarked, frequent, apart from true jaundice—the ‘icterus lienalis’; it probably results from the anæmia, the altered blood being unable to destroy the bile-pigment absorbed into it from the intestine. The urine varies in amount, but is usually strongly acid, and of high specific gravity. The amount of urea is unaltered, that of uric acid is increased. Hypoxanthin, lactic acid, and formic acid have been found in it. Albumen is rare, apart from structural changes in the kidneys. Menstruation is usually arrested. The functions of the nervous system are disturbed by the altered blood: languor, tinnitus, and vertigo are frequent; and slight mental failure, delirium, and coma are occasionally met with. The graver symptoms are probably due to capillary obstruction by masses of leucocytes, or to small hæmorrhages. Besides the noises in the ears, deafness is common, especially towards the end. In the fundus oculi changes may usually be seen with the ophthalmoscope. The pallor of the blood in the retinal and choroidal vessels is conspicuous. The retinal veins become very broad, and are often tortuous. Extravasations of blood are almost invariable at some period, usually striated, sometimes rounded. Yellowish or white spots are sometimes seen, due to the collections of lymphoid cells already described. Occasionally the retina is irregularly thickened, so that the vessels present conspicuous antero-posterior curves. These changes constitute the ‘leukæmic retinitis’ of Liebreich. The extent to which sight is interfered with depends on the degree to which the neighbourhood of the macula lutea is involved. The skin is strikingly pale, or sometimes, as already

stated, greenish yellow. Occasionally a peculiar dark pigmentation is present. Sweating is common.

**COMPLICATIONS.**—The most frequent complications of leucocythæmia are pleural effusion, œdema of the lungs, lobar pneumonia, bronchial catarrh, pericardial effusion, dilatation of the heart, venous thrombosis, cerebral hæmorrhage, and fatty degenerations. The more rare complications are cirrhosis of the liver; parenchymatous degeneration of the kidneys, giving rise to the symptoms of sub-acute Bright's disease; renal calculi; persistent or intermittent erection of the penis, the former probably due to thrombosis in the corpora cavernosa; œdema of depending parts; ascites; furuncles; and erysipelas. The various hæmorrhages are rather to be regarded as symptoms of the disease than as complications.

**DURATION.**—Cases of splenic leucocythæmia vary in duration from six months to seven years. One or two recorded cases ran their course in less than six months. The average duration of sixty-three cases was two years. The actual duration of the affection is probably longer than this, because the disease has often reached a considerable degree before the symptoms become troublesome.

**CAUSES OF DEATH.**—The most common causes of death in leucocythæmia are loss of blood, asthenia, diarrhœa, cerebral hæmorrhage, pneumonia, and pleurisy. In asthenia the actual end is often due to cardiac failure. The hæmorrhage most frequently fatal is from the nose, the next most frequent from the bowels.

**PATHOLOGY.**—The pathology of leucocythæmia is still involved in obscurity. We are imperfectly acquainted with the normal life-history of the blood-corpuses. For a full discussion of the facts which have been ascertained, and the theories built upon them, the reader is referred to Dr. Gowers's article on the disease in Reynolds's *System of Medicine*, vol. v. It is only possible here to give a brief outline of the pathology of the disease which these facts and theories suggest. Recent researches make it probable that the red corpuscles arise from a transformation of the smaller lymphoid cells—*globulins* of Donné, hæmatoblasts of Hayem; and that this transformation takes place to a large extent in the splenic pulp and in the marrow of bones, tissues which have many histological characters in common. If these tissues are diseased, the transformation may not take place, and the unchanged lymphoid cells may develop into the ordinary leucocytes, which are either retained in these tissues, increasing their bulk and changing their structure still further, or pass into the blood. Both results probably occur. Hence we assume a primary change of the splenic pulp, which is increased further by the retained leucocytes. Where the hæmatoblasts arise is still un-

certain. They are probably in part developed in the splenic pulp and marrow of bones, from pre-existing cells, and from the protoplasmic trabeculæ of the tissues (Klein); probably in part they come from the true lymphatic structures, the glands, Malpighian follicles of the spleen, &c. Primary disease of these lymphatic structures constitutes lymphadenoma; and the splenic pulp may be normal, and the leucocytes are only in slight excess, or are not more numerous than they should be. In true splenic leucocythæmia, the glands and Malpighian follicles are not primarily diseased, but they may suffer secondarily, when lymphatic growths arise in organs; and this secondary affection is in part the result of the accumulation of leucocytes. That the enlargement of the spleen is not, as has been thought, merely the result of the accumulation in it of pale corpuscles from primarily diseased blood, is shown by the increased consistence of the organ, and by the fact that the splenic tumour precedes the change in the blood. There is some reason to believe that, as the spleen alone may be diseased, so, in some rare cases, the marrow of bones may alone be diseased, and may give rise to a primary 'myelogenic leucocythæmia,' but this is not yet proved. That the disease may be primary in both the marrow and the spleen is highly probable from recorded facts. It is certain, however, that the marrow is, in most cases, not affected primarily, and may be unaffected throughout, or may suffer secondarily, as do the glands. The same is true of the collections of lymphatic tissue elsewhere in the body. The cases in which the lymphatic glands enlarge early—'lymphaticosplenic leucocythæmia'—are, for the most part, if not entirely, cases of composite nature. The spleen presents a double change—growth in the follicles, such as are associated with the glandular growths in lymphadenoma (Hodgkin's disease), and increase in the splenic pulp, as in pure splenic leucocythæmia. In such cases there may be a large increase in the pale corpuscles of the blood.

**DIAGNOSIS.**—The diagnosis of leucocythæmia rests on the existence of enlargement of the spleen, and a considerable excess of leucocytes in the blood. In all cases of splenic tumour the blood should be examined: if the proportion of white corpuscles to the red is greater than 1 to 20, the case is certainly one of leucocythæmia. But if the proportion is less than this, leucocythæmia cannot with certainty be excluded, because it is probable that, in all cases, the splenic tumour precedes the leucocytal excess, and the latter may be in process of development. To ascertain the actual state of the blood, it is always desirable to enumerate the corpuscles with the hæmacytometer. Repeated examination, to ascertain that the proportion of pale corpuscles is not increasing, is necessary before impending leucocythæmia

can be excluded. In cases in which the lymphatic glands enlarge early, the question arises whether the case is one of splenic leucocythæmia, or of Hodgkin's disease. In the latter, as just stated, the enlargement of the spleen depends, not on an increase of the splenic pulp, but on overgrowth of the Malpighian follicles; the splenic enlargement is less than in leucocythæmia, and is less uniform. In the composite cases alluded to above, in which, with enlargement of the glands and splenic follicles (lymphadenoma), there exists also overgrowth of the splenic pulp, and a considerable leucocytal excess in the blood, the two morbid processes are conjoined, and the affection may be termed lymphadenosplenic leucocythæmia. These cases are distinguished from the simple splenic affection by the early enlargement and firmness of the glands. In simple splenic leucocythæmia the affection of the glands is usually late, and rarely considerable. The diagnosis of the disease from conditions in which a considerable excess of pale corpuscles exists, without enlargement of the spleen, is usually easy, because such excess is transient, soon passes away, and is not associated with a splenic tumour.

**PROGNOSIS.**—The prognosis of a disease which depends on a primary affection of the blood-forming organs is necessarily most grave. No means of arresting the progress of the developed disease has yet been discovered. The immediate prognosis is less serious in proportion as the evidence of organic changes in the blood-forming organs is slight, and in proportion to the early stage of the disease. Neither age, sex, nor causation affords prognostic information. The greater the number of white corpuscles and the deficiency of red, as ascertained by counting, the worse the prognosis. The size of the spleen, alone, affords little information. Hæmorrhages are of grave augury, but epistaxis least so.

**TREATMENT.**—The knowledge of the causes of leucocythæmia, slight though it is, suggests important prophylactic measures—the prevention of ague, and the careful treatment of all who have been exposed to malarial influences. Splenic tumours resulting from such exposure should be systematically treated; the subjects of them should exercise great care to avoid exposure to cold, injuries, and all causes of portal congestion. These precautions are especially necessary in women at the menstrual periods; and if such women bear children, their state during pregnancy and after parturition should be carefully supervised, and lactation prohibited. Whether there is simple anæmia or leucocythæmia, every effort should be made to reduce the size of the splenic tumour, by quinine, cold affusion, ergotin, and especially by voltaic electricity, a most powerful agent. By obtaining contraction of the spleen, expelling

retained leucocytes, and perhaps stimulating directly its functional action, we render its condition less abnormal. In a case of anæmia splenica no remedies improved the blood-state till the spleen was galvanised, when the red corpuscles at once began to increase. Remedies which do good in ordinary anæmia have slight influence in this disease. Iron is almost useless; cod-liver oil, however, has seemed to do some good. Arsenic has been largely tried, but without benefit in pronounced cases. Its undoubted value in lymphadenoma suggests its further trial in early cases; it should be given in the largest doses that can be borne. Mosler advises injection of Fowler's solution into the spleen; provided that the organ is firm, dense, and close to the abdominal wall, that there is no hæmorrhagic tendency, and no high degree of cachexia. Phosphorus has been recommended, but in almost every case of pronounced leucocythæmia it has been powerless for good. Nevertheless, its influence in improving the blood-state in lymphadenoma warrants further trial in the early stage of the disease. Iodides, bromides, and mercury are useless. Temporary improvement has been obtained by the inhalation of oxygen to the extent of 30 litres daily (Kirnberger). Change of air may slightly improve the patient's state, but has no influence on the disease. Transfusion has been tried, but the results are not encouraging. Excision of the spleen has been attempted. The operation has been performed with success in cases of anæmia splenica, but in actual leucocythæmia it has been invariably fatal—in most cases from uncontrollable loss of blood, the result of the hæmorrhagic tendency. Further trial of it, in such cases, does not seem to be justifiable. In early cases, where there is no considerable excess of pale corpuscles, and the red are not reduced below 60 per cent. of the normal, it might be successful; but it is questionable whether, in these cases, an ultimate bad issue without interference is sufficiently certain to justify the performance of so grave an operation. Special symptoms may require treatment. Hæmorrhage must be checked by the usual methods, and crystals of perchloride of iron may be applied to accessible places (Jenner). For vomiting, a posture which will relieve the stomach from pressure, and counter-irritation, are useful. Aperients should be employed with caution; and under no circumstances should the yellow tint of the skin lead to the use of mercurials. For the œdema, digitalis and other diuretics are best. For the splenic pain, counter-irritation, sedative liniments, and hypodermic injections of morphine may be used. In proportion to the anæmia, physical rest is important, that the diminished supply of oxygen may not be rendered inadequate for the need of the tissues, by muscular exertion.

W. R. GOWERS.      FREDERICK TAYLOR.

**LEUCOCYTOSIS** (λευκός, white; and κύτος, a cell).—A condition of the blood, in which the white corpuscles are appreciably but moderately increased. See BLOOD, Morbid Conditions of; and LEUCOCYTHÆMIA.

**LEUCODERMA** (λευκός, white; and δέρμα, the skin).—White or achromatous integument. See PIGMENTARY DISEASES OF THE SKIN; and LEUCOPATHIA.

**LEUCOMA** (λευκός, white).—A white opacity of the cornea, generally referable to inflammation or ulceration of that structure. See EYE, AND ITS APPENDAGES, Diseases of.

**LEUCOMAINES** (λεύκωμα, whiteness). A term applied to a class of alkaloids resulting from the normal metabolism of the animal proteid tissues without the intervention of any bacterial agency. To the class of alkaloids resulting from the putrefaction of tissue the term *ptomaines* is given. See PTOMAINES.

**LEUCOPATHIA** (λευκός, white; and πάθος, a disease).—SYNON.: Albinism, Achromia, Leucoderma, Leuce, Leucasmus.

This disease is sometimes general, but frequently partial; in the latter form constituting *cutis variegata* and 'piebald skin.' The whiteness is referable to absence of pigment, which may be simply due to an arrest of function of the rete mucosum, or to an organic alteration of the integument. See PIGMENTARY SKIN-DISEASES.

**LEUCO - PHLEGMATIC TEMPERAMENT.**—See TEMPERAMENT.

**LEUCOPLAKIA BUCCALIS ET LINGUALIS.**—See TONGUE, Diseases of.

**LEUCORRHŒA** (λευκός, white; and ῥέω, I flow).—SYNON.: Fr. *Leucorrhée*; Ger. *Weisser Fluss*; Lat. *Fluor Albus*; Pop. 'The Whites'; 'White Discharge.'

**DEFINITION.**—A non-hæmorrhagic discharge, of pale colour, escaping from the female genital fissure.

**ÆTIOLOGY.**—Leucorrhœa is a symptom rather than a distinct disease; and may result from any of the morbid processes that lead to hypersecretion from the genital mucous surfaces, or from the glands opening upon them, whether the mucous membranes be injured or entire. It is, however, a source of much discomfort and deterioration of health, and so demands special treatment.

**SYMPTOMS.**—Leucorrhœa presents several distinct varieties according to the seat of its cause; and the symptoms of each variety require separate consideration.

1. **Vulvar Leucorrhœa.**—In this variety a glairy viscid secretion is found bathing the

apposed surfaces of the pudenda, stiffening into a crust on the surface of the labia majora or on the insides of the thighs, and sometimes glueing the lips more or less firmly together at their margins. It is usually derived from the muciparous glands covering the internal surfaces of the labia majora and the nymphæ; but in cases of special eruptions and general vulvitis it may come from the vestibular surface; and in still rarer cases it is poured out from the glands of Bartholin. Vulvar leucorrhœa is met with at any period of life, but is most common in the young, *infantile leucorrhœa* almost always being of this variety. In cases of gonorrhœal infection in the female, the vulva is usually the seat of a profuse discharge which is apt to become purulent, but it is rarely confined to this situation, spreading both into the urethra, and upwards into the higher spheres of the genital mucosa.

2. **Vaginal Leucorrhœa.**—The discharge in cases of vaginal leucorrhœa is most frequently white in appearance, of acid reaction, and due to a secretion from the general surface of the vaginal mucous membrane. Its whiteness is found, on microscopic examination, to be owing to the presence of quantities of scaly epithelial cells, many of which are crowded with fatty particles, whilst others have been quite dissolved in consequence of the fatty degeneration. Sometimes the discharge has a more yellowish tint, and then it is found to contain quantities of pus-cells among the epithelial scales. In the former group of cases we have to do with a simple catarrhal condition of the vaginal mucosa: in the latter there are red granulation-like spots scattered over the membrane, which has here lost its epithelial covering. Vaginal leucorrhœa is a complaint to which women are specially liable during their reproductive life. The catarrhal form is extremely common in young married females; whilst the other form occurs rather about the menopause, or, if occurring earlier, is complicated with some of the other varieties of leucorrhœa. Apart from specific causes, it may be brought on by sexual excesses; by the presence of a foreign body, such as a pessary; by a displaced uterus; by a chill; or by any condition that interferes with the circulation in the pelvis. In a large proportion of cases it is secondary to the next variety of leucorrhœa.

3. **Cervical Leucorrhœa.**—The discharge that comes from the canal of the cervix uteri is transparent, like unboiled white of egg, very tenacious, and of alkaline reaction. It may still present these characters as it escapes from the pudenda; but it generally becomes somewhat clouded as it passes through the vaginal canal, and gets acted upon by the acid secretion from the vaginal walls. Independently of this change in the vagina, it is sometimes found already

more or less opaque as it lies within the cervical canal, and may be seen of a yellowish or greenish or reddish tint in various cases. The clear cervical leucorrhœa is seen under the microscope to be made up of a viscid magma, having entangled in it large numbers of columnar epithelial cells, which have a tendency to arrange themselves in rows. These are easily seen to be the ciliated epithelial cells that cover the normal mucous membrane, but deprived for the most part of their cilia. They are accompanied by smaller rounded cells like mucous corpuscles or wandering cells, partly derived from the interior of the crypts, and partly shed from the general surface from which the epithelium has been removed. In almost all cases some of the epithelial cells and mucous corpuscles are charged with fatty particles, and surrounded with granules, resulting from the breaking down of some of their number. The more turbid the fluid, the more the cells are found to have undergone such degeneration; and where the discharge is profuse, fluid, and of yellowish colour, it has more the characters of a purulent fluid in which the relatively few cylindrical cells are changed in form, becoming oval or rounded, and nearly all reduced to a compound granular mass. The more deeply tinted discharges owe their discoloration to the admixture of blood, the red corpuscles of which can easily be recognised. Apart from the leucorrhœas of specific origin, this is the commonest of all the varieties. It may be found in females of any age, but specially affects women during their reproductive history, and more especially those who have been mothers. We can understand the special liability of the cervix to catarrhal affections, when we remember that all intra-uterine discharges pass through and may irritate it; that it is exposed to damage during the transit of the fœtus in parturition; that vaginal affections easily pass into it by continuity of structure; and that it may readily be injured by foreign bodies in the vaginal canal, or even by fretting of its orifice against the vaginal wall in cases of displacement or excessive mobility.

4. **Intra-uterine Leucorrhœa.**—Here also the discharge is transparent, like white of egg, and alkaline in its reaction, but it is more fluid than the secretion from the cervical canal, and may escape as a clear liquid from the genital fissure. In cases of long standing, more particularly where there exists some organic disease in the uterine parietes, the fluid becomes turbid, purulent, and more frequently than in any other variety of leucorrhœa tinged with blood, even alternating with irregular discharges of blood. Under the microscope we see many cylindrical epithelial cells, not infrequently ciliated; along with groups of smaller cells, partly cylindrical, partly rounded, that have been discharged from the uterine follicles; all

embedded in a mucous fluid. Where the discharge is more turbid, the epithelial cells are seen to be undergoing fatty degeneration, and to be accompanied with wandering cells, pus-globules, and crowds of free fatty particles. This uterine leucorrhœa may be found at any period of life, but as an independent affection it is found almost exclusively in virgins or young married women, or in women who are ceasing, or have ceased, to menstruate. In the last-named class of cases the cervix is often atrophied, and its orifices narrowed; and the intra-uterine secretion may accumulate for a time, and be expelled with some degree of suffering. Most frequently it is found associated with cervical leucorrhœa, the endo-cervical affection having passed up to the endometrium, or, more rarely, *vice versâ*. Perhaps the most frequent form of it is found in women who are subject to a leucorrhœal discharge before or after the menstrual periods; and in the cases of amenorrhœa where a pale discharge escapes at the usual menstrual periods, this has its source in the interior of the uterus proper.

5. **Tubal Leucorrhœa.**—Doubtless some small portion of the fluid that escapes in certain cases of leucorrhœa is furnished by the Fallopian tubes; but despite the elaborate attempts of Hennig and others to establish a distinction between it and the other varieties, it remains rather as a subject of pathological interest than of clinical importance, and need not occupy us further here.

DIAGNOSIS.—The statements of a patient in regard to a leucorrhœal discharge cannot be relied on in establishing a diagnosis as to its source. If it be white and flaky we may judge that it is vaginal; if more transparent, and escaping in half-coagulated flocculi, we may conclude that it is cervical; whilst a clear and more continuous and fluid discharge would be more justly referred to the uterus proper. But it is never safe to trust merely to the appearance of the discharge as it escapes from the vulva, for it may have become modified as it lay in or traversed some part of the canal, or may be compounded of fluids derived from different surfaces. The seat of the discharge must therefore be exposed. In the vulvar variety it suffices to separate the labia and occasionally to expose the navicular fossa and the orifices of the Bartholinian ducts, by passing the finger into the anus. The vaginal form of leucorrhœa requires for its detection the use of a speculum, duck-bill or tubular; and the cervical, one of these or a bi-valve speculum. For the diagnosis of intra-uterine leucorrhœa it is sometimes helpful to remove some of the fluid for microscopic examination by means of a fine syringe. Unless a clear history of infection can be obtained, it is almost impossible to establish a distinction between a gonorrhœal discharge and the

simpler catarrhal leucorrhœa. In the former there is a very notable tendency to spread through all the contiguous mucous surfaces, though the vulva may be predominantly affected. In children suffering from the infectious discharge, traces of the injuries that are usually inflicted at the period of infection should be sought for.

**TREATMENT.**—In instituting our treatment of leucorrhœa it is of the first importance to have in view the *constitutional* condition of the patient; to use means to counteract any diathetic tendency—tuberculous, strumous, or syphilitic; and to raise as far as possible the general standard of the patient's health, by the administration of tonics and the enforcement of a suitable diet and regimen. It is partly in this way that a change of residence is often useful; and in making a change, it is well for the patient to go to some of the spas, such as Eins or Kissingen, the waters of which are helpful in reducing congestions and catarrhs of the pelvic viscera. In young women of relaxed habit of body, it may be enough to prescribe quinine and iron or arsenic, and the daily use of a cold sponge bath; and in infantile leucorrhœa, cod-liver oil and iron should be administered.

In the great majority of cases of leucorrhœa some kind of *local* treatment becomes an absolute necessity. Sometimes it is enough to pay strict attention to cleanliness, washing the pudendal surfaces with a soft sponge, or syringing the vaginal canal with tepid water; and even when astringent applications are to be made, the surfaces should first be subjected to a detergent stream of water. Where there is marked congestion of the uterus it is best to make the injections with hot water, and to keep the stream passing through the vagina for at least five minutes at a time; the immediate relaxation of the blood-vessels and hyperæmia being followed by contraction of their walls, which favours the cessation of the discharge. The astringents most serviceable for checking *vulvar* and *vaginal* leucorrhœas are alum, aluminated iron, acetate of lead, sulphate of copper, sulphate of zinc, borax, and infusions of oak-bark, matico, and other vegetables charged with tannin. They are best applied in the form of an injection with a Higginson's syringe, having a vaginal nozzle attached to it; or of a douche through a long india-rubber tube, with a stop-cock for regulating the flow fitted close to the vaginal nozzle, and the other extremity opening into a wide receptacle, or fitted to a filler into which the fluid is poured. Where there is a difficulty in using the injection, and where it is desirable to keep up a more prolonged application of the medicament, it may be introduced into the vagina in the form of pessaries made with cacao-butter or with gelatine. Topical applications to the canal of the cervix and cavity of the uterus ought always to be made through the speculum, and without

such applications it is a hopeless task to undertake the cure of *cervical* leucorrhœa. Here, more concentrated or more powerful astringents or escharotics become necessary. Nitrate of silver in the form of a stick of caustic is easily applied, but its repeated application may lead to mischief. Zinc-alum, dried sulphate of zinc, sulphate of copper, perchloride of iron, or tannin may be introduced, in the form of rods or arrows made with starch and gum. If a uterine sound or stilette be dipped in water and a thin film of cotton wadding wrapped round the point to the length of about two inches, the adherent mucus can be cleared away, and the same or another sound mounted with wadding can be charged with fuming nitric acid, the acid nitrate of mercury, strong carbolic acid, a solution of perchloride of iron, tincture of iodine, or iodised phenol, and carried through the speculum along the cervical canal. In *intra-uterine* leucorrhœa it becomes necessary to carry the application right up in the same way to the interior of the uterus. It is usually best to begin with one of the stronger liquids, apply it a few days after a menstrual period, and follow it up with applications of iodine. So long as the stilette or sound with the dry wadding passes easily through the os internum, it is usually necessary to continue from time to time the intra-uterine application.

ALEXANDER RUSSELL SIMPSON.

**LEUKÆMIA.**—See LEUCOCYTHÆMIA.

**LEUTERBAD (LOCITE),** in Switzerland.—Thermal earthy waters. See MINERAL WATERS.

**LEVICO,** in the Trientino, Austria. Arsenical sulphate of iron waters. See MINERAL WATERS.

**LICE,** Diseases Due to.—See PEDICULUS.

**LICHEN** (λεῖχην, an eruption).—The somewhat loose definition given by the older writers to the word 'papule' caused them to include under the head of '*lichen*,' diseases, or rather conditions of the skin, some of which belong to other categories. Thus Willan's list included seven species: (1) *Lichen simplex*; (2) *L. pilaris*; (3) *L. circumscriptus*; (4) *L. agrilus*; (5) *L. lividus*; (6) *L. tropicus*; and (7) *L. urticatus*.

1. **Lichen Simplex.**—This condition is merely a temporary lichenous condition—in short, the papular stage preceding the vesicular stage of eczema.

2. **Lichen Pilaris.**—SYNON.: *Keratosis Pilaris*; *Pityriasis Pilaris*.—Lichen pilaris is an affection which is differently classed by authors; it is essentially a chronic condition, characterised by a heaping up of epidermis in horny papules round the hair-follicles, giving the part affected the feel of a rasp.

The papules are of the colour of the surrounding skin, which is generally ichthyotic or xerodermic; in fact, the condition keratosis pilaris is part of the general malformation of skin belonging to ichthyosis. The outer surfaces of the extremities and the backs of the hands and phalanges are the seats of election.

**3. Lichen Circumscriptus.**—SYNON.: *L. Annulatus*, *Serpiginosus*; *Seborrhœa Corporis*.

**DEFINITION.**—A serpiginous, ringed, papular eruption, occurring on the trunk, and accompanied by seborrhœa.

**DESCRIPTION.**—The sternal and interscapular regions are the seats of election, from which the eruption may spread to the epigastric or lumbar regions. The eruption is characterised by numerous circles and segments of circles (looking much like tinea). The margins of the circles are made up of minuted papules; the space enclosed is somewhat buff-coloured, and desquamating minute scales; the eruption has a greasy appearance, and is greasy to the touch. It is often associated with the wearing of flannel. The pathology is obscure, but some think its essential nature is that of a seborrhœa.

**TREATMENT.**—Any parasiticide ointment will remove it, such as creasote ten minims to one ounce of lard, rubbed in every night.

**4. Lichen Agrius.**—This, like lichen simplex, is apparently but a temporary stage of eczema.

**5. Lichen Lividus.**—*Lichen lividus* is a purpuric condition, in which the purpuric spots appear as papules round hair-follicles. See PURPURA.

**6. Lichen Tropicus.**—SYNON.: Prickly Heat.—This is a sudoral eruption due to inflammation of the sweat-glands. See SUDORIPAROUS GLANDS, Disorders of.

**7. Lichen Urticatus.**—*Lichen urticatus* is an affection which is now classed as a variety of urticaria. It is common in children. See URTICARIA.

In accordance with the teaching of Hebra, modern dermatologists are disposed to restrict the term 'lichen' to papular affections in which the papules retain their character as papules, and do not undergo any further evolution. There is another condition imposed, namely, that the papule must be inflammatory. The group 'Lichen' is thus narrowed down to two newly described diseases, *Lichen ruber* and *Lichen scrofulosorum*.

**8. Lichen Ruber.**—SYNON.: *Lichen Planus*.

**DEFINITION.**—A chronic eruption, more or less extensive, of flat and angular, or acuminate inflammatory papules, in colour either red or bluish-red.

**ETIOLOGY.**—The disease may be due to nervous exhaustion, consequent on anxiety or any lowering influence, disorders of the stomach and intestines or uterus; in some

cases possibly to chill. It generally attacks persons towards the middle period of life—twenty to fifty; but it may occur in the young.

**SYMPTOMS.**—*L. ruber* or *L. ruber planus* (so named in contradistinction to the second variety, *L. ruber acuminatus*) commences as flat millet- or pea-sized papules, red or violet-red in colour. The lesions are discrete at first, but soon aggregate in patches or lines; if closely observed, a central depression (umbilication) is noticeable in the papules. As the patches enlarge, the appearance of individual papules becomes lost; the skin is thickened, raised above the surface, coarsely furrowed, and covered with minute goldbeater-skin-like scales. The seats of election are the wrists and forearms (flexor surfaces). No region is exempted. The eruption is often symmetrical, and it may be restricted to a certain nerve-distribution. Itching is almost a constant symptom, and may be intense. The disease may last for years, and ultimately become generalised. A single attack is the rule, but it may recur. The mucous membranes may be affected.

*Lichen ruber acuminatus* is the form originally described by Hebra as *lichen ruber*. It is more frequently general, and attended with severe symptoms, shivering, general aching, and profuse perspiration (Crocker). The papules are smaller and more conical than in the preceding variety; the whole process is more acute. The two forms of eruption may be present at the same time.

**PATHOLOGY.**—In the plane variety the process appears to be inflammatory, associated with the sweat-ducts at first, with subsequent thickening of the rete. In the acuminate variety the hair-follicles are first affected (Crocker).

**DIAGNOSIS.**—When the papules are well marked (and in most cases a few are to be observed), they are distinctive of the disease. It is when the patches enlarge, and the disease becomes generalised, that it may be confounded with chronic eczema or universal psoriasis.

**PROGNOSIS.**—This is only grave in generalised cases, in which the patient may drift into marasmus.

**TREATMENT.**—The general health must be dealt with, especially in cases of nervous exhaustion, by means of iron, quinine, or cod-liver oil. Itching must be combated by rest in bed, and the application of antipruritics. Arsenic should be tried and persevered with in all cases where it can be borne; the two conditions in which it is contra-indicated being dyspepsia with constipation, and acute disease, in which it often makes the skin worse. External treatment is of great assistance. Soothing applications, if the eruption is acute, such as lead and calamine lotions; mild tar lotions if they can be tolerated—*Liquoris carbonis detergentis* ℥ii. to ℥vi. of

water; oleum cadinum; or even inunctions with soft soap and pix liquida, if the patches are very chronic, and do not resent such treatment after trial.

**9. Lichen Scrofulosorum.**—Lichen scrofulosorum is another affection first described by Hebra. It is extremely rare in this country, although it undoubtedly exists, especially amongst the poorer classes. It is characterised by pale papules, approaching the colour of the skin; these tend to range themselves in circular patches, not circles, generally on the trunk rather than the extremities. The papules are scaly, and after a time disappear, leaving small yellowish pigmented spots. Here and there a papule inflames, and becomes acne-like. There is little itching. The disease occurs in scrofulous subjects. When occurring in adults it might be mistaken for the small papular syphilide. Cod-liver oil is the remedy internally, and also externally by inunction.

ALFRED SANGSTER.

**LIEBENSTEIN**, in Saxe Meininger.—Chalybeate waters. See MINERAL WATERS.

**LIENTERIC** (λείος, smooth; and *ἐντέρον*, the intestine).—A form of diarrhœa in which the stools contain much undigested food, in consequence of its having passed rapidly along the alimentary canal. See DIARRHŒA; and STOOLS.

**LIFE ASSURANCE.**—The great extension of life assurance of late years has placed the solution of important questions involving large financial operations within the range of the daily work of the medical practitioner. It is important, therefore, that his duties and the nature of the knowledge required of him should be defined. We shall dwell briefly on the nature of life assurance, and point out the rules which should guide the medical adviser of a life office in forming an opinion. We shall then consider the principal agents which modify the duration of human life, and their bearing on the questions submitted to us. Life assurance is a contract of indemnity. The amount of the indemnity to be paid on death is fixed by each individual according to his means or wishes, and the company engages to pay the sum when life fails or under other conditions, in consideration of an annual or other payment or premium so long as the contract lasts.

Irrespective altogether of health, the risk undertaken by the company is greater or less according to the variation in two factors: (1) the age of the life to be assured; (2) the duration of the term of assurance. The premium required for an assurance on a life aged forty, is greater than that for a life aged twenty; and again the premium for an assurance over the years of life from forty to

sixty is greater than that for an assurance from forty to fifty only. As the premium is calculated on the assumption that the proposer has a chance of surviving to the extreme limit of life, it is obvious that the company ought to insure at the ordinary rate of premium such lives only as have the best chance of attaining to old age, or, as they are called, *first-class lives*. Hence lives coming under any of the following classes must be discarded from the first-class standard:—

(1) Lives not possessed of sufficient vital power to afford them the possibility of attaining old age. This will include lives which have shown a tendency to certain diseases which will develop later on in life, as gout, rheumatism, &c.

(2) Lives whose family history discloses a tendency to a breaking down short of longevity.

(3) Lives whose place of residence or occupation has a more than usual effect on life, or involves peculiar hazards.

Generally, lives which fall short of the highest standard in respect of one out of these three conditions may often be accepted, but if they fail of the highest level in more than one condition they must be excluded. The ideal, therefore, which the medical examiner should set before himself as constituting a first-class life, is the exclusion of these three conditions.

The tables of *expectation of life* put before medical examiners, which are deduced from the Institute of Actuaries' H<sup>m</sup> tables, and are based on the mortality of healthy males whose lives had been assured with twenty British life offices, are often misunderstood. Expectation of life indicates the average number of years which is lived by all persons of a common age, from that age up to the extremity of life. The total number of years that will in the aggregate be lived by a group of persons of the same age, divided by the number in the group, gives the 'expectation.'

The medical examiner is for the time being the retained adviser of the office which employs him. Any medical man may decline to act for a company, but, if he has accepted a fee, he is bound to consider their interests as of primary importance, and his relations with his patient as secondary. He should not, therefore, allow himself to be swayed by motives of personal friendship, or because he is or has been the ordinary attendant of the proposer. Generally speaking, it is not judicious to employ the ordinary medical attendant of a proposer to report for an office, but it is often necessary to obtain his evidence confidentially, and, unless he declines to act, he is bound not to withhold any information he may have regarding the health, habits, or family history of the life. Every examiner should be aware that his report and opinion are necessarily submitted to the

physician of the company, and thus a greater accuracy in the use of medical terms will often be observed.

The examiner will have an interview, always private, with the proposer, who should not be accompanied by anyone. The wife may ask that her husband or friend shall be present, but this is not advisable. Persons of either sex are always more frank when alone with their doctor. There may indeed be ailments kept secret even from a wife or husband, or former events in life bearing on health, which would not be revealed in the presence of a third party. It is scarcely necessary to advise that the utmost courtesy and quietness of manner should be observed, and the proposer placed at his ease as much as possible. A deterrent inquisitive manner is apt to give rise to prevarications or concealment in the replies given.

An inquiry into the functions of the body is often resented when put abruptly, but the writer has never failed to obtain all necessary information by a quiet questioning. He is in favour, however, of direct questions, plainly spoken out, especially with women; but many questions of the kind are to be avoided.

Regarding temperance, it is generally useless to put the question directly to a proposer. His estimate of what constitutes excess may not be yours. His opinion of his own habits may be comparative as regards his neighbour, and he may be a dram-drinker (the worst form of excess) but never 'get drunk'! It is well to approach the subject with inquiry as to his hours, habits, neighbourhood, and society, and thus elicit both his favourite drink, and how often it is taken.

The proposer being placed in a good light, the examiner will notice his general build, corpulence or otherwise. The face and hands will especially occupy him. The face may indicate intemperance, sensual habits, or nervous excitability. It should be neither pale nor flushed, free from blotches, and the nose not unduly red. The drunkard's nose, protuberant, granulated and rosy at the end, is often associated with a blear-eyed state of the eyelids. There are some forms of skin-affection which simulate it, but the practised eye will discriminate the chronic effects of alcohol which, by impairing the elasticity of the capillaries, has given rise to the nose here indicated. A flushed state of the male may lead us to suspect phthisis; while redness of the cheeks composed of permanently dilated blood-vessels, together with a velvety integument exuding sebaceous matters, always caused Brinton to suspect kidney-disease.

The hand affords evidence of various kinds, and its form, colour, and nails should always be regarded. We may thus detect gouty thickening; or, from the clubbed state of the fingers and incurvated nails, be enabled to

detect chronic phthisis, or old pleuritic or pericardial disease. The tongue should be examined, not only as regards its being furred, fissured, moist or dry, but also for ulcers or growths, which, if present, should always be examined by pressure of the fingers. The throat may also give evidence of present or old syphilitic ulceration. The gums may present the blue line of lead-poisoning, or the dark-red line of phthisis. The gait, nervousness of manner, twitching of muscles, inability to walk straight, peculiarities of the eyeballs and facial muscles, or other symptoms, will betray some deep-seated affection of the nerve-centres, and the patellar reflex should be examined if any suspicions arise. Tremblings or shakiness of handwriting may indicate alcoholism, or abuse of tea or of smoking.

The teeth may be deficient or carious, and their condition is often a good test of general health. The notched teeth of syphilis should always be looked for. Finally, the whole aspect of a man should be noted, as conveying that he is robust, hearty, and vigorous, or the reverse, well developed in muscle of chest and limbs, or puny and weak, with loose tissues and flabby muscles.

The chief duties of the medical examiner are to ascertain with precision:—

(1) The *family history* of proposer; (2) his *past history*; (3) his *present state* of health; and (4) his *habits*.

1. The *family history* is to include not only his parents, brothers and sisters, and grandparents, but the health and ages at death both of them and their descendants collateral to proposer—that is, of uncles, aunts, and first-cousins. These facts need not all be recorded in the report paper supplied by the office, but their elucidation often enables the examiner to form an opinion as to the eligibility of a life where any taint, as consumption, gout, or cancer, exists in one generation.

2. The *past history* of a proposer will occupy us with much care. He is bound to tell us whether he has ever had any serious ailment; but, as memory is fallacious, the direct questions which are found in every form of medical report issued by the offices are to be put. It will be convenient then to ask in detail whether he has had symptoms of diseases affecting the chest, head, and abdomen, or any disorder of a general kind, as fevers; and young men should be plainly asked about syphilis.

Inquiry should be made as to former residence in an unhealthy climate; also whether proposer has had any medical attendant, and, if so, for what.

3. The *present state* of health is then to be ascertained, and a careful examination made of chest and abdomen. We cannot insist too strongly on the necessity for *uncovering the chest*. Without this, percussion

cannot be practised, the expansion, the movements, and alterations in the walls, caused by congenital malformation or by former disease, as pleurisy and phthisis, must escape notice. The heart-sounds cannot be estimated through the dress; and we have known many instances in which morbid conditions were overlooked because the proposer was so examined. There are heart-murmurs and lung-sounds which can be simulated or disguised by a well-starched shirt. For the examination of the *abdomen* the waistband should be unloosed, and palpation and percussion practised, by which the existence of enlargement of the liver or spleen, or of tumours, may be ascertained, and in doubtful cases the reclining posture should be adopted.

4. By the *habits* of the proposer we understand his general mode of living, and his opportunities for exercise, but above all his temperance or excess in the use of stimulants. The writer advises that the average quantity of stimulants taken daily should be written down from the reply of proposer on the document which he has to sign. The testimony of friends is always asked on this point, but the replies are generally fallacious, and often consist in a 'Yes' or 'No' to the queries sent to them. In proposals important as to amount, a written statement as to the friends' estimate of the habits of a proposer should be obtained in the form of a letter, and not on a printed form.

Certain other points may now be briefly discussed.

5. *Age*.—Before considering the diseases which shorten life, some general considerations about age are worthy of note. Different ages predispose to particular diseases. Respiratory diseases prevail most from puberty to twenty-five, and this is the age when phthisis is most fatal. It has been reckoned that at forty half the danger from this disease is over, and three-fourths at fifty years of age; and the rule seems a fair one. An heredity to the development of phthisis at a somewhat advanced age—say after forty—may, however, exist, and prove an actual danger to life. The liability to certain diseases, as gout, apoplexy, degeneration of organs and blood-vessels, and also urinary affections, increases after forty, and it is at and after middle life that the temptation to excess in diet and stimulants, with a diminished desire for exercise, leads the way to slow organic alterations. Persons who attain to great age are generally spare, and have in almost all instances been frugal in their habits, and active in mind and body. The decay of muscle and of organs is precipitated by want of use, and the due exercise of all faculties of mind or body is conducive to longevity. It is good to compare the apparent with the actual age of a proposer. As a general rule, when he is really older than he looks, his expectation of life surpasses the average, and when he has aged

beyond his years the risk is thereby so far impaired.

6. *Occupations*.—The life which is protected from the vicissitudes of fortune by a fair provision for daily wants, which has occupation for both mind and body, without undue strain or the necessity for hurry, and which has daily exercise in open air, combined with a moderate amount of sedentary work—is undoubtedly the best risk. The clergy are perhaps the best lives of all callings, and the statistics of the clerical offices bear out this statement. Teachers, heads of schools, lawyers, and physicians probably come next. The 'business' class, which includes merchants, stockbrokers, bankers, manufacturers, directors of companies, &c., is fairly healthy, and stands among the first on the list, but the examiner will do well to remember the anxieties incident to all these callings.

There are certain classes exposed to manifold dangers, for which special rates are demanded by all offices. Thus the publican is charged 1*l.* per cent. extra for occupation, a sum which appears to be scarcely sufficient.

7. *Heredity*.—Heredity plays an important part in the life-history of every individual, as there can be no doubt that physical, mental, and moral characteristics are often transmitted from ancestors. The whole constitution may not be so communicated; and parents may be mere transmitters along a chain, their offspring representing an earlier ancestor. The examiner will therefore inquire both about parents and grandparents. In tracing the effects of transmitted disease we should find how far others of the same generation, as brothers, sisters, and cousins, may have been affected by it. We have thus a gauge of the intensity of the transmission. Longevity, which implies the perfection of the whole animal system, is no doubt hereditary in some families. If both parents have been the subject of the same disease, the heredity is intensified in the offspring. A disposition for zymotic diseases is marked in certain families. The heredity to insanity is intensified by successive inheritance; to syphilis is lost in the repetition of inheritance. Phthisis and cancer become intensified by inheritance, and appear earlier in each generation. Double inheritance (both parents) induces earlier and a more rapid form of disease. Heredity appears to influence the sexes equally. *Forms* of disease run in families: in some the consumptive taint is shown in acute tuberculosis, in others the most chronic form of fibroid phthisis prevails. The *mother's* transmitting influence seems greater than the *father's*, and it is common to all hereditary diseases for the mother to transmit to sons and the father to daughters. *Family phthisis* appears at an earlier age as we descend the genealogical tree; thus,

the grandfather may have it at sixty, his sons at forty, and his grandsons in early life.

*Cancer* is hereditary in one-third to one-seventh of the cases. It is most prevalent between forty and sixty. The encephaloid form is met with earlier; schirrus from forty to fifty; epithelial cancer later. The risks of hereditary cancer therefore increase with age after a certain point, while those from phthisis diminish. The rule must be to reject the issue of two cancerous parents. The heredity of *rheumatism* and *gout* is incontestable. Hereditary gout appears early, often at eighteen to twenty; acquired gout appears about forty. If both parents, or even one, have had several acute attacks of rheumatism, there is great likelihood of transmission to the child. *Diabetes* is allied to gout and rheumatism, and comes after the latter in frequency of heredity. *Heart-disease* claims the same heredity as gout and rheumatism; and while the acute form in the parent is apt to be represented in the child, it is not to be forgotten also that such parents commonly transmit a tendency to slow thickenings of the valves to their children, who may never suffer from the acute form. The many instances of valvular lesions met with in practice where the individual has had no acute attacks, may often be thus accounted for. If one parent have had gout or rheumatism severely, the proposer being in perfect health, the heredity may be overlooked; but if both parents have had these affections the life should be declined, or a large addition made to the premium.

It has been found that *asthma* was hereditary in fourteen cases of thirty-five, and in seven it was paternal and direct. The most frequent period of development was in youth, and next in old age. If both parents were asthmatic, an addition should be made to the premium. *Albuminuria* may be regarded as only transmissible when the result of gout or alcoholism in the parent.

*Intemperance* must be considered as hereditary in a high degree. It runs in families, who not only inherit the nervous constitution which drunken parents transmit, but that peculiar temperament which flies to stimulants as a resource on any emergency of life. The *craving* for drink, a want which alcohol can alone supply, as it stops for a time the waste of nerve-power, is often derived from the excesses of drunken parents.

Still further, the children of drunkards inherit various neuroses, such as hysteria, epilepsy, mania, ataxy, and different forms of paralysis; and a child begotten during drunkenness early falls a victim to either phthisis, or some deep-seated disease of the nervous system. Intemperance is perhaps the most formidable enemy to the safe assurance of life, and even ranks before phthisis in its deadly effects on the system. Organic

ailments are by it originated, and organic weakness crystallised into disease. The degenerations of age are anticipated and precipitated by alcohol, and the dram-drinker is sure to have a shortened life. The man who carries his drink well and is 'never drunk' is in the greatest danger. Small doses of stimulants throughout the day, ended by a somewhat larger one at night, leave the system always charged with alcohol, and the excretory organs are continuously under its influence. The most searching inquiry should therefore be made as to the quantity taken each day, and the frequency of the dose. The medical examiner must be thrown on his own tact to discover from certain well-known indications whether the proposer is a drinker. Among these are the flushed face and nose, the tremulous tongue or hand, the relaxed skin, the eye, and the manner over-rapid and nervous, or subdued and sullen. Of all classes of the intemperate the habitual dram-drinker is the worst. The occasional drunkard is not in so great a danger, and no doubt is often reclaimed; but the writer cannot recommend the acceptance of any of those for life assurance, as no addition to the premium would compensate the certain loss to the company.

*Diseases of the nervous system* are eminently heritable, as epilepsy, general paralysis, and mania. Of 321 epileptics, one-third had epileptic parents. The children of such become diseased early, but it may be considered that after forty the liability is exhausted.

*Insanity* is commonly hereditary: about one in three or four have had a parent insane. The issue of one insane parent might be accepted if the age be 35 or 40, and the proposer free from any nervous affection.

**PERSONAL EXAMINATION.**—The medical examiner should ascertain the condition of the chief organs of the body, especially the heart and great vessels, the lungs, and the kidneys.

*Heart.*—The size of the heart and the strength or weakness of its impulse should be ascertained.

*Intermission* of heart-beat and pulse, if unaccompanied by other irregularities of rhythm, may not be of grave importance. If it be only a loss of beat once in twenty or thirty it may be regarded as harmless, or referable only to an alteration in the innervation of the heart. It may not be always present, may be indeed absent for days or weeks, or be suspended during a feverish attack, or present only during some trivial alteration of digestion.

*Irregularity* of heart-action is of different import. The heart will repeat a number of rapid beats, then pause, and lose one or several successively. The sounds are confused in character and run into one another,

the impulse is diminished, and the interval lost or irregular. Feebleness is the predominant character. In this condition, degeneration of heart-muscle has already commenced.

A *tumbling* but forcible and struggling heart-beat is not to be confounded with the above. It is a common result of pericardial adhesions and hypertrophy. The danger to life would probably arise from the consecutive events of hypertrophy, dilatation of the heart's chambers, and valvular incompetence.

*Murmurs* may be audible over any of the valvular regions of the heart, and accompany either the first or second sound or both.

The *characters* of a murmur—its softness, flowing, harsh or rasping sound—are *per se* no indications of its value or pathological import: a harsh or a loud murmur is often of a less serious nature than one more distant and less pronounced, as the power of the ventricle behind the obstruction is generally the cause of its loudness, and a moderate degree of hypertrophy is a compensating agent. If there be obstruction at an orifice, all symptoms of heart-disorder may be held in abeyance for years if only there is power to drive the blood through the obstructed valve. It is only when the heart fails to do so that the serious symptoms of dilatation are initiated; or if the valve permit of regurgitation, and the muscular walls of the cavity into which the blood is returned be efficient in contractile power, the dilatation is opposed and all its concomitant symptoms are absent or delayed. *Ephemeral* murmurs often give rise to doubts as to the safety of assurance—such as occur in the left sub-clavian region, when firmly pressed by the stethoscope, or in certain positions of the person examined, or in that portion of heart which is covered by the lung. They may be heard on deep inspiration only, and are temporary in character. They are not to be regarded as prejudicial to life. The *anæmic* murmur, soft, systolic, and blowing, is heard over the first part of the aorta or pulmonary artery. It is occasionally heard over the mitral region. It is associated with a lowered condition of general health due to anxiety, over-work, or loss of sleep; and if these conditions are altered, the individual may recover from all his symptoms and lose the physical signs. But it is not to be forgotten that fatty degeneration of the walls of the heart has been found in cases of prolonged anæmia. The insurance rule is to defer such cases for a month or two.

The lesions most important to consider are those of the aortic and mitral valves.

*Aortic* murmurs may be single or double. The single murmur is probably obstructive, the valve being thickened and warty, and the coats of the vessel diseased.

In *aortic regurgitation*, recognised by a diastolic murmur, the greatest degree of ventricular hypertrophy occurs, and the heart attains an enormous size in compensatory efforts to maintain the systemic circulation against gravity, the open valves permitting the return of a large portion of the blood into the ventricle. The carotids are seen throbbing, and the radials present the well-known character caused by sudden recession of the blood-current called the water-hammer pulse. This form of disease is among the most fatal of heart-affections, and is entirely un-insurable, the elements of durability being wanting.

*Mitral murmurs* are the most common effects of rheumatic endocarditis, which results in the thickening of the valve, and stenosis, or permanent imperfection, permitting regurgitation into the ventricle. The recognition of these cases is easy, but their value for the purposes of life assurance depends not on the character or situation of the murmur, but on the actual conditions, which we shall now consider.

It is now a well-ascertained fact that persons in great number having a marked mitral murmur may live to advanced age without presenting any symptoms, and therefore without the effects of heart-disease recognised as injurious to life; and of this the best medical authorities have recorded numerous instances. The first downward step in the fatal event of mitral disease is when the cavities of the heart begin to yield to the pressure of the regurgitated blood, and dilatation follows. With dilatation a thinning of the muscular walls begins, and degeneration of fibre follows as a necessary sequence. With lessened power of the ventricle, engorgement of the right side of the heart occurs, the return current is blocked in the auricle, which in its turn dilates, the lungs become congested, the return of systemic venous blood is retarded, and the phenomena of congested lung, shown by œdema and hæmorrhage, and of congested venous and capillary systems, evidenced by dropsy and effusions, are certain to follow. The series of events is mechanical; and it may be stated that, so long as the muscular integrity of the heart's walls is maintained, the subject of the affection may enjoy fair health with ordinary precautions. The dangers to such a life would probably arise after middle life, at the period when degenerations of structure commonly commence. The rule of assurance companies has till lately been to reject all such lives as present any form of mitral disease; but if a case present with murmur only, without great enlargement of the heart, if the ventricular contraction be strong and the murmur well pronounced, and if, in addition, it can be shown that such condition must have prevailed for a long time—say, from the date of one attack of

rheumatic fever years previously, if the proposer has his other organs healthy and does not exceed thirty-five years of age, then it is safe to accept such a life with a considerable addition to the premium, or by securing that all the premiums should be paid up in a short term of years—say, eight or ten.

It remains to say of heart cases that the most dangerous are those indicating failure of muscular power, and that the aortic cases are more perilous than the mitral. The presence of albumen in the urine should always be looked for in connexion with disease of the heart.

*Pulse.*—The frequency of the healthy pulse varies with the constitution of the nervous system, and the posture, sitting or standing, of the individual. Its healthy beat might average 60 to 70; but a large range should be allowed for temperament and emotional causes, the very fact of its being examined giving rise to acceleration. The physician will therefore give time for its subsidence, or make a fresh examination on another day. A sustained frequent pulse may indicate fever, phthisis, or exophthalmic goitre. The fulness or incompressibility of a pulse, and a character of wiriness under the fingers, should be noticed. The latter may indicate sclerotic changes in the vessels, when the condition of the temporals and other arteries should also be examined.

*Chest.*—A careful examination of the lungs should be made by palpation, percussion, and auscultation, the dress being removed, and measurements taken by a tape at the level of the sixth rib. These vary in different individuals, and from 33 to 38 inches have been given by different observers as the average in adult males. The difference between extreme inspiration and expiration should not be less than two inches at the level of the nipple. It may be taken that the *mobility* of the chest is of more importance than its size. Attention should be paid to any undue flattening of the chest-walls, such as may have been caused by phthisis or old effusions. Should a spirometer be available, it will be found that a healthy man of average build can expel from 200 to 250 cubic inches of air, which represent his *vital capacity*, leaving still behind a certain amount of residual air which cannot be removed by any expiratory effort; and every inch in stature above five feet should add eight cubic inches to the vital capacity.

*Phthisis.*—The great prevalence of this disease renders its study from an insurance point of view of the highest value. According to the Registrar-General, one death in every eight is due to this cause. We need not dwell here on the methods of recognising existing phthisis by its physical signs; but if these latter are undeniably present the life cannot be considered insurable, unless for very short periods, and even then on terms

which would be rarely accepted by a proposer. However, as every condition of health has its insurance price if we could appreciate its possible future with anything like accuracy, it is well to remember that certain forms of phthisis are necessarily very chronic. Of these, fibroid phthisis and a well-defined single cavity in one lung are illustrations; but, if it were possible to accept them for insurance purposes, their chronicity should have been already shown by a long previous history, and the actual state of proposer be without either fever or much wasting.

*Hæmoptysis.*—There is no question more frequently before the medical examiner than the influence of blood-spitting on the value of life. We have met with the following varieties:—

(a) After a run or strain in athletics, as rowing or lifting weights, a mouthful of fluid blood has been brought up, a few clots some hours, or next day, after; and then a total cessation of the symptom occurs, without any subsequent disorder of health.

(b) The patient having had cold or cough for a week or two, a 'few streaks' of blood mixed with phlegm have been brought up, or a spoonful of red blood in the morning on waking, and the symptom has recurred after an interval of days or weeks.

(c) Without acknowledged disorder of health, or cough, a robust or, it may be, a spare person may bring up a mouthful of fluid blood, dark, or it may be in clots, for a day or two, when it ceases, without any symptom of illness, but may return, and probably will return, some time after, as weeks or months.

(d) A copious blood-spitting, a cupful or pint, fluid at first, then clotted, possibly repeated in a day or two, with or without any manifest disorder of health.

Such are the most common descriptions of blood-spitting which are given us orally, or reach us as medical reports from examiners. As we are dealing with reports only, we do not mention the symptoms, such as bodily temperature, which should guide us if we had the opportunity of seeing the patient at the time.

(a) is the usual story of hæmoptysis from strain or injury, and may occur without any subsequent ill-health. It is common among the young and robust, and is a simple overflow resulting from high pressure. Should an interval of months have elapsed since the occurrence, and the health remain good, and the absence of any physical signs of lung or heart-disease be verified by examination, such lives may be accepted, with a small addition to the premium, especially if the applicant have passed thirty years of age. Of course an abstinence from over-exertion of any kind should be insisted on.

(b) is the characteristic of commencing phthisis, and, as grave disorders of general

health, as well as physical signs of lung-disease, will be manifest, the life is plainly not insurable.

(c) is probably of cardiac origin, or due to hæmophilia. That from heart-lesion is met with in two very distinct forms. The more severe is commonly due to mitral disease, and occurs late in the history of disorders dependent on that cause; and venous congestions, with evidence of dilated heart-chambers, will have generally been in existence long before a hæmorrhage into the lung takes place. As this case does not often present for life assurance we need not further consider it.

The milder form is of not infrequent occurrence. A person commonly under thirty-five will have brought up blood from the mouth on various occasions, fluid, but more commonly dark or clotted, and have but little disturbance of health. There is commonly no cough, nor any expectoration, and only slight dyspnoea may be complained of. The most careful examination will detect nothing wrong in the lungs, and the subjects have commonly large and well-made chests. There is no rise of temperature at or after the attack, and the nutrition of the body is perfect. The writer has examined very many such cases, and in all the only physical sign was a slight click with the systole at the apex or lower end of the sternum. He has watched many of them over a series of years, and found no more serious symptom than here stated. There is little doubt that they are chiefly mitral imperfection, and, as they may eventuate in serious disease, are not eligible for ordinary life assurance. It might, however, be possible to accept some of them for very short periods at an advanced premium. See HÆMOPYSIS.

*Emphysema.*—Proposers often present the physical signs of emphysema, especially at the bases of the lungs. The proposer may or may not be the subject of fits of asthma, but the breath is always short on exertion. The co-existence of bronchitis shown by *râles* of the dry, sonorous, or mucous character, most marked with expiration, is often found, and eventually few advanced cases remain unassociated with heart-failure. Cases of this kind, excepting those which present heart-symptoms, may be accepted with an addition to the premium, the amount of which will vary with the mildness or severity and the rarity or frequency of the attacks, and the degree of loss of elasticity in the lungs. The liabilities increase with age, and the premiums should be paid up before the proposer is much beyond middle life. A short occasional attack of asthma, without the characteristic physical signs, may not materially vitiate a life.

A liability to *bronchial attacks* manifests a delicacy of the air-passages, and is often observed in persons of a phthisical, gouty, or syphilitic family. Chronic bronchitis in the

elderly is commonly found in emphysematous cases, and the gradual supervention of changes in the air-vesicles and chambers of the heart renders such lives precarious. They are, however, occasionally accepted for a short term, and with a considerable addition to the premium.

*Pleural effusions*, if existing, would prevent the acceptance of a life, but neither a contracted side from absorption of a former effusion, nor the marks of paracentesis for the evacuation of the fluid, if the recovery has been complete, and the lung has descended to nearly its former level, should forbid acceptance if the general health be good.

*Digestive tract.*—The tongue and fauces should in all cases be inspected. The salivary glands should be free from hardness or enlargements. The fauces should be examined for marks of former ulcerations. The nostrils should be free from obstruction, and the voice not nasal. The stomach should be examined by palpation and percussion, as stricture of the pylorus, or dilatation, may be present, and if found would justify rejection of the life. The *liver* should not come below the ribs, and a breadth of about two fingers should represent the normal dulness from the lower ribs upwards. Inequalities on the surface of the liver may mean cancer or cirrhosis or hydatids, and their presence would justify exclusion. Mesenteric tumours, fibroid, scrofulous, or malignant, are not admissible. Hardness in the right iliac fossa, especially with a history of former typhlitis, is also inadmissible. Any of the following affections, if known to have existed, should be regarded with suspicion: Hæmatemesis, gastralgia with vomiting, hepatic colic, hydatid cysts, obstruction of the bowels, typhlitis or perityphlitis, and ascites. There are cases of *hæmatemesis*, generally in the female, or the result of strain or direct injury, which might be accepted, but only after years of perfect health have elapsed. *Jaundice* of former occurrence, if the attacks have been slight and not traceable to liver-disease, would not forbid acceptance. *Biliary colic*, if the attacks have been severe or recent, would prohibit acceptance; but a former attack, with subsequent years of good health, need not imply rejection.

*Dysentery* which may have occurred in a hot climate, from which the proposer has been absent for some years, with good present health, need not disqualify, but a considerable extra premium will be required if the proposer returns to a hot climate.

All disorders of the *rectum*, except hæmorrhoids of a simple kind, are serious. Cancer, abscess, or the syphilomata, may be suspected if symptoms of pain or obstruction can be elicited. *Hæmorrhoids* need not disqualify unless they be of a severe form, or have recurred after operation, or presently call for surgical interference.

*Genito-urinary organs.*—Disorders of these organs are among the most important which affect the value of lives. They are often very insidious, as persons in a state of disease may present the appearance of perfect health. It is notorious that a man may have glycosuria or albuminuria, and neither know it himself nor exhibit in his appearance any evidence of the disorder. It is therefore imperatively necessary that the urine should be tested chemically in every case. This rule was formerly not adhered to, and many offices suffered in consequence; but it has now become a general practice, which has in some instances to be supplemented by a microscopical examination.

*Hæmaturia* of recent occurrence, and *stone in the bladder* would forbid acceptance. *Dysuria* from stricture or enlarged prostate are unfavourable conditions, but there are mild cases of stricture which may be accepted with an addition.

*Syphilis.*—Syphilis affects the duration of life considerably, and is to be regarded not only from the length of time which has elapsed since the last attack and its cure, but from the nature and degree of the symptoms. The late Berkeley Hill believed that the curable cases get well in two years; the incurable may last for an indefinite time. For two years a person may expect a return of the eruption. Tertiary affections generally appear about five years after the primary affection. Mr. Arthur Cooper endorses this view. The assurance rules may be thus stated: During the actual existence of any syphilitic disorder the proposal should be postponed. A man who has had syphilis in only the ordinary cutaneous and superficial form, and has been properly treated for it, may be accepted after a year has elapsed without symptoms. If he have had syphilis in the secondary forms, he should only be accepted if two years have elapsed since the latest symptom, and then with a small addition to the premium. Those who have had visceral syphilis affecting the brain, cord, liver, lungs, kidneys, &c., should be rejected.

*Albuminuria* is found in several chronic affections of the kidney, and in acute nephritis. It is also present in several acute inflammatory affections, with which we are not now concerned. Its presence should always be looked for in the urine. It is usually more copious after food and exercise, and its appearance in the urine is often intermittent, being absent at times for hours or even days. Acute albuminuria from exposure to cold and wet, and that which follows scarlatina, may disappear without leaving any organic change in the kidney, but in certain cases it recurs. If, however, some years have elapsed since its occurrence, and the proposer have enjoyed perfect health, the case may be accepted. In chronic cases, it

is most commonly the result of some form of Bright's disease. There is, however, no doubt that albuminuria occurs in many persons without any other symptoms of disordered health, and may so continue for years. According to Professor Grainger Stewart, the proportion of cases of albuminous urine found in presumably healthy persons is 30 per cent. He examined 407 cases. In all, the maximum quantity was found after breakfast. Dr. Saundby and other observers have reported cases of albuminuria which had lasted twenty years without disorder of health. Sir George Johnson considers, however, that its presence in the urine is always pathological; that nearly all cases pass through the stage of intermission, which may last for long periods; and advises that no one with a trace of albumen in his urine should be accepted at the ordinary rate. We should advise the following rules for practice:—

*Albuminuria*, formerly existing as a result of acute nephritis or scarlatina, with an interval of perfect health for years, need not disqualify a proposer. Cases of albuminuria presently existing, and known to have existed for years, in persons of otherwise perfect health and good family history, may be assured for a short period—say of five years—with an addition to the premium, and may come up for examination again at the end of that period. Cases of albuminuria with any of the symptoms of *chronic Bright's disease*, with any cardiac, gouty, or rheumatic complication, or any suspicion of intemperate habits, should be declined. For the tests for albumen, see ALBUMINURIA.

*Casts* in the urine, whether epithelial, hyaline, waxy, colloidal, or amyloid, if present, would tend to confirm the diagnosis of organic disease of the kidneys.

*Glycosuria.*—Sugar may be present in the urine without diabetes, of which all the symptoms may be wanting. Glycosuria is often a temporary condition, and may disappear leaving no ill effects; but it is always to be regarded with suspicion, and its presence alone should forbid the acceptance of a life. It is common in obese persons, gross feeders, and in the gouty habit; and its subjects may have no suspicion of its existence, and feel in perfect health. The urine should therefore be always examined for sugar. Diabetes is a permanent, but glycosuria may be a temporary condition, and the mortality from it increases with age up to sixty or seventy years. Males are twice as liable as females. It is difficult to measure its duration, as dietetic measures can much retard its progress; but the prognosis, on the whole, is bad, as the liability to cerebral affections, to phthisis, carbuncle, and other fatal complications is very great. Its subjects, therefore, must be excluded from life assurance. Glycosuria will forbid the acceptance of a proposer while it exists; but if there be no gout,

and if all traces of sugar have disappeared for years, a calculated addition might be made to the premium. In glycosuria the quantity of urea is generally increased. A high specific gravity, even above 1030, is no evidence of sugar in the urine, unless a chemical examination have proved its existence. For the tests for dextrose, or grape sugar, see **DIABETES MELLITUS**.

The excess of *phosphates* in the urine is generally temporary, and often due to anxiety and waste of nerve-power. It is recognised by a cloudiness on boiling, which is entirely cleared by the addition of a drop or two of nitric or acetic acid. It forms no obstacle to the acceptance of a life.

Disease of the *testicle*, tubercular, strumous, or malignant, disqualifies for assurance. *Hydrocele* and *varicocele* may *per se* be regarded as harmless.

*Female organs*.—Metritis, *pelvic* inflammation, or ovarian disease, are doubtful cases for acceptance. Women who have had repeated abortions, puerperal hæmorrhages, eclampsia, or a necessity for obstetrical operations, are not eligible unless the menopause has been favourably passed. During a *first pregnancy*, it is well to defer assurance till after a safe confinement. In 3,722 *primiparæ* Mathews Duncan found one death in every 74; and in 12,671 *multiparæ* there was one death in every 123. Lactation is no bar to acceptance. The average duration of life in women is longer than in men, and by latest returns is shown to be increasingly so.

*Nervous system*.—Diseases of this system, as cerebral softening, locomotor ataxy, hemiplegia, paraplegia, aphasia, disqualify a proposer. Local paralysis of the motor muscles of the eyeball, and ptosis, unless congenital, would lead to suspicion of brain-disease, and would not be acceptable. *Paralysis of the facial nerve* (Bell's), when purely local, and free from complications with disease of the ear and disorder of the fifth or other nerve, need not disqualify. *Paralysis* from lead-poisoning is always serious, and the liability to diabetes and deep disorders of the nervous system should not be forgotten. A recognition of the blue line on the gums would lead us to decline the life. *Neuralgia* of branches of the fifth nerve may not disqualify unless they be associated with cerebral symptoms, or be the result of syphilis or exostosis. *Vertigo* of a severe kind, associated with symptoms of *Mènière's* disease, would disqualify; and even its more transient forms, if often repeated or associated with gout, may be a bar to acceptance. *Trembling of the hands* may arise from the abuse of tea, tobacco, or alcohol. If from the latter cause, we may expect the tongue to be also tremulous. Although it may be temporary, its presence should cause grave suspicion as to habits.

The more severe forms of *epilepsy* must forbid acceptance, unless ten or fifteen years

have elapsed since the last attack, with an interval of perfect health, and even then an addition should be made to the premium. There is a mild form appearing in early life, which can generally be traced to such causes as overwork or too close application to study, and where the heredity is absent; if years have elapsed without its recurrence, and the proposer has passed thirty and has engaged in the active work of life without symptoms of nervous disorder, and is strictly temperate, the life may be accepted with an addition of five to seven years.

*Height and weight*.—The relative proportion of height and weight is important in estimating the probabilities of future disease in a proposer. In estimating over-fatness, if the chest and shoulders be large and the abdomen moderate in size, the condition is less important than if the abdomen were large and pendulous. In stout persons, therefore, the circumference of chest, abdomen, and limbs should be given, and flabbiness or firmness of tissue is to be considered, while habits as regards exercise, food, and stimulants should be carefully inquired after. The *dangers* of obesity are weakness of heart or ultimate fatty degeneration of that organ. In a large proportion of these cases, fatty degeneration has affected the liver, and atheromatous alterations take place in the coats of the arteries, which lead to their ultimate rupture and consequent hæmorrhages. Obese persons are also worse subjects for fevers and accidents than are the spare and muscular, their power of recovery being lowered from feebleness of circulation. For these reasons the very obese are scarcely subjects for life assurance, although occasionally, if their habits be moderate, they may be accepted with an addition. On the other hand, the existence of excessive *leanness*, which may be the result of phthisis, diabetes, or various other wasting disorders, demands a careful examination. Most companies have tables of relative heights and weights, but a wide margin must be left for individual variations. One is appended which the writer has found sufficiently accurate.

Height in inches	Weight	Height in inches	Weight
	st. lbs.		st. lbs.
63·0	8 13	70·0	12 4
63·5	9 2	70·5	12 8
64·0	9 5	71·0	12 11
64·5	9 8	71·5	13 1
65·0	9 11	72·0	13 5
65·5	10 1	72·5	13 9
66·0	10 4	73·0	13 13
66·5	10 7	73·5	14 3
67·0	10 10	74·0	14 7
67·5	10 14	74·5	14 11
68·0	11 3	75·0	15 1
68·5	11 7	75·5	15 5
69·0	11 10	76·0	15 9
69·5	12 0		

**Claims.**—On the death of an assured life a claim is sent in to the office by the legal owners of the policy. This is accompanied by certificates from the medical attendant, and from the registrar of the district in which death occurred. The practitioner who has to certify the cause of death cannot be too distinct in his statement, and in avoiding the use of terms which express only the symptoms and not the disease which proved fatal. Thus *dropsy* is common to heart, liver, kidney, and other diseases, and should not be employed; and for the same reason *hæmorrhage* and *diarrhœa*, which are common to many affections, should not be used. *Gastritis*, when certified as a cause of death, should always excite suspicion. The writer has known it used to conceal irritant poisoning and intemperance. A sudden attack of persistent vomiting with diarrhœa, leading within a week or two to a fatal issue, should not be received without careful inquiries as to the assumed cause of death. *Syncope*, which may represent the final stage of many diseases, should not be used. *Childbirth* is often used to cover other affections, such as phthisis, and should be limited to incidents proving fatal at or soon after a confinement.

We have also occasionally to consider whether the death of a proposer has been due to diseases which must have existed at the time when the life was accepted, and therefore whether the statements made in the proposal as to the previous state of health and habits of a proposer were strictly true. If not true, the claim could not be maintained. There is here scope for all the acumen of the medical adviser of an office.

It is believed that the practising physician will find in this article sufficient data to guide him in the selection or rejection of most lives offered for assurance. It is not possible to lay down specific rules for the amount of additions to the premium which it will occasionally become the duty of the medical examiner to advise. He may either rate a life at so many years older, or adopt some of the other methods by which the company may be secured against loss—as paying up all premiums within a given number of years, or deducting a portion of the sum to be paid by the company if death occur within a certain time, or making the sum payable at a certain age (endowment). Although on all these questions of finance the medical adviser can only recommend, his opinion will be highly useful to the company, with whom the ultimate decision will rest.

JAMES E. POLLOCK.

**LIGHTNING, Effects of.**—SYNON.: Fr. *Les Effets de la Foudre*; Ger. *Blitzschlag*.

The effects produced by lightning differ only in degree from those produced by the discharge of static electricity, generated in

the laboratory. With a Leyden jar of sufficient size a small animal may be killed, and in larger animals the effects of shock and local injury may be produced. By lightning a person may be killed outright, and a *post-mortem* examination may reveal no lesion whatever. The mode of death in these cases seems to be by the shock to the brain and nervous system generally. Effects not distinguishable from ordinary concussion of the brain may be observed, and the person struck may remain insensible, with slow respiration, scarcely perceptible pulse, and dilated pupils, for periods varying from a few minutes to more than an hour. This may be followed by complete recovery; or there may remain paralysis of the limbs, usually the lower, or occasionally derangements of the special senses—blindness, a metallic taste in the mouth, noises in the ears, and an odour in the nose. The brain may be more or less permanently affected, and we read of delirium, mania, and loss of memory as results of the lightning-stroke.

Various objective phenomena have also been observed. The electricity on its way through the body may produce a number of mechanical effects. Wounds like those produced by a blunt stabbing instrument may mark the points of entry and of exit; bones have even been broken, the membrana tympani has been ruptured, and internal viscera have suffered in a similar way. Patches of erythema, urticaria, superficial ecchymoses, and scorplings of the surface having a curious tree-like and branched arrangement, have all been described; and this last phenomenon has apparently given rise to the assertion that delineations of trees standing in the neighbourhood of the accident have been traced photographically on the body of the victim. Lightning is apt to be attracted by any metal worn about the body. Watch-chains are frequently broken and fused, and by the intense heating of these metallic conductors the clothing has been set on fire. Watches have been broken and partially fused, and have forcibly burst through the pockets in which they were contained. Steel articles, such as pocket-knives, have been rendered magnetic. The clothing is sometimes burnt and torn to a great extent, and strong boots have been found burst open, or thrown off the feet to a distance, or nails in the soles have been driven out of them.

The remote effects of lightning are due to the mechanical injuries produced by it. Permanent paralyses may result from injury to the nerves; and inflammatory action may be set up by the injury inflicted on internal or external parts. One case is recorded in which the whole of the hair on the head and body, as well as the nails of both hands, came off after a lightning-stroke. It has been asserted that *rigor mortis* does not occur in persons killed by lightning, and that the blood

remains fluid for a very long time after death, but neither of these facts has been substantiated.

**TREATMENT.**—The treatment of those who have been struck by lightning consists in first rousing and keeping up the respiration and circulation. The cold douche is often of great value; and this, combined with friction of the limbs, warmth to the extremities, and the administration of stimulants, either by the mouth or in the form of enemata, would seem to be the measures best calculated to restore the suspended animation. Secondly, special injuries must be subsequently treated according to their nature.

G. V. POORE.

**LINEÆ ATROPHICÆ** (Lat. Atrophic lines).—A form of scleroderma. See **SCLERODERMA**.

**LIPIK (LIPPIK)**, in Slavonia.—Thermal muriated saline waters, with iodine. See **MINERAL WATERS**.

**LIPOMA** (λίπος, fat).—A fatty tumour. See **TUMOURS**.

**LIPSPRINGE and INSELBAD**, in Germany.—Earthy waters. See **MINERAL WATERS**.

**LISBON**, West Coast of Portugal.—Warm, moist climate, with very variable temperature. Mean temperature in winter, 54° F. Prevailing winds, N.E.—S.E. in spring; S.W. rainy. See **CLIMATE**, Treatment of Disease by.

**LISDOONVARNA**, in Ireland.—Sulphur waters. See **MINERAL WATERS**.

**LITHÆMIA** (λίθος, a stone; and αίμα, the blood).—A condition of the blood in which it contains excess of uric acid. See **GOUT**.

**LITHIASIS. LITHIC ACID DIATHESIS** (λίθος, a stone).—See **GOUT**; and **URIC ACID DIATHESIS** and **URIC ACID CALCULUS**.

**LITHONTRIPTICS** (λίθος, a stone; and τριψίς, friction).—**SYNON.**: Fr. *Lithontriptiques*; Ger. *Steinauflösende Mittel*.

**DEFINITION.**—Lithontriptics are therapeutic measures used for the purpose of dissolving calculi in the urinary tract.

**ENUMERATION.**—The chief lithontriptics are: Water, Potassium, Lithium, Borax, Phosphate of Sodium, Soap, Lime-water, Nitric Acid, Phosphoric Acid, Hydrochloric Acid, Sulphuric Acid, Piperazin, and Mineral Waters, such as those of Contrexéville and Wildungen.

**ACTION.**—Lithontriptics dissolve stones in various ways. Some of them possess a simple solvent action, as in the case of water. Others unite with the calculi so as to form a more soluble compound, as in the

case of the union of potassium or lithium with the uric acid of a calculus, producing urate of potassium or lithium, which is more soluble than uric acid itself. In the case of phosphatic calculi, diluted nitric acid combines with the bases of which they are composed, to form a more soluble compound.

**USES.**—Lithontriptics may be employed for the purpose of dissolving calculi either in the kidney or in the bladder. They may either be taken internally, so as to act upon the calculi through the medium of the urine; or be injected directly into the bladder. This latter treatment can only be adopted in the case of a vesical calculus, and is inapplicable in the case of a renal calculus. The most useful of all lithontriptics is water, and especially distilled water. When this is taken in large quantities, the urine becomes very dilute, and small calculi may be partially dissolved, so as to be reduced in size and ejected through the natural passages. If the calculus is composed of uric acid, potassium or lithium is the best remedy for internal administration, the urates of these bases being more soluble than the urate of sodium. Piperazin is a solvent of uric acid, twelve times as powerful as lithium carbonate. It has been used as a lithontriptic both internally and as a local injection into the bladder. In the case of phosphatic calculi, acid remedies are employed instead of alkaline; but it is exceedingly difficult to render the urine acid by means of acids given by the mouth, unless they are administered in quantities likely to derange the digestion. In place of mineral acids, benzoic acid and benzoate of ammonium have been employed, as benzoic acid passes out of the body in the form of hippuric acid, giving an acid reaction to the urine. On account of this difficulty, acids have been directly injected into the bladder, in order to act directly upon the stone; for which purpose nitric acid, largely diluted, is the one which has been most generally employed. This procedure, however, is now rarely had recourse to, as it is much easier to crush the stone by mechanical means. The two most important springs for the treatment of renal or vesical calculi are Contrexéville and Wildungen. The waters of both of these places contain carbonate of iron, which appears to aid their action.

T. LAUDER BRUNTON.

**LITHURIA** (λίθος, a stone; and ούρον, the urine).—A condition in which a deposit of uric acid or urates takes place in the urine. See **LIVER**, Functional Diseases of; **URIC ACID DIATHESIS** and **URIC ACID CALCULUS**; and **URINE**, Morbid Conditions of.

**LIVER, Diseases of.**—**SYNON.**: Fr. *Maladies du Foie*; Ger. *Krankheiten der Leber*.

The liver is an organ which has always occupied a prominent place, both with the

profession and the public, as being the seat of important diseases, as well as the origin and source of numerous symptoms and ailments. Not only is it concerned in the formation of the bile; but it has also a peculiar glycogenic function; and is now generally believed to take an important part in metabolic changes, with the production of urea or uric acid, as well as in the destruction of red blood-corpuscles, peptones, and ptomaines. Moreover, the organ has a special portal circulation, by means of which the blood returning from the stomach, intestines, pancreas, and spleen is distributed throughout its substance, and thence conveyed to the inferior vena cava. Hence, hepatic affections, by interfering more or less with the physiological functions or anatomical arrangements of the organ, may give rise to diverse phenomena, not only of a local character, but also associated with the general system, or affecting some remote part.

**SUMMARY OF DISEASES.**—The individual affections of the liver are treated of separately, in alphabetical order, but it may be well to indicate beforehand their general nature. The first great division is that into *functional* and *organic*. *Functional* hepatic disorders are commonly regarded as of peculiar significance, and of great importance. The main *organic* diseases of the liver, in which there is some more or less obvious anatomical change, may be summarised thus: 1. *Hyperæmia* or *congestion*, either active or mechanical. 2. *Hæmorrhage* into the organ, or so-called *apoplexy*. 3. *Acute inflammation*, usually terminating in *abscess*, rarely in actual *gangrene*. 4. *Chronic inflammation*, ending in the condition termed *cirrhosis*. 5. *Hypertrophy*. 6. *Atrophy*, either *acute yellow*—which is a very fatal disease; or *chronic*, the latter being of different kinds. 7. *Biliary accumulation* and *pigmentation*. 8. *Malpositions* and *malformations*. 9. *Infiltrations*, including *fatty* and *albuminoid* disease. 10. *New-growths*, especially *hydatids*, *syphilitic formations*, *carcinoma*, and *sarcoma*. Tubercle is occasionally found in the liver; and rarely lymphatic formations, simple cysts, erectile tumours, or other new-growths.

**ÆTIOLOGY AND PATHOLOGY.**—Taking a general survey of the causes which originate hepatic diseases, and of the circumstances under which they arise, the most important may be indicated as follows: 1. An affection of the liver may be merely a local manifestation of some constitutional or general malady, as in the case of cancer, tubercle, syphilis, or albuminoid disease. The liver is an organ very liable to suffer from albuminoid change. 2. Some local injury or irritation may originate hepatic disease, either from without, as a blow or stab; or from within, as sometimes happens in the case of biliary calculi. 3. Certain animal parasites entering the

body are prone to lodge in the liver. This applies especially to *hydatids*, originating from the *Tænia echinococcus*. 4. From the intimate connexion of the liver with the alimentary canal, especially through the portal circulation, hepatic disorders are very liable to arise from improper diet, as well as from digestive derangements affecting the stomach and bowels, and as a consequence of constipation. 5. Abuse of alcohol, and especially indulgence in ardent spirits, occupies an important position in the ætiology of disorders and certain diseases of the liver. Undue use of hot condiments is also regarded as a factor of some consequence. 6. Long-continued exposure to a high temperature in tropical climates is a powerful cause of hepatic derangement and disease, particularly if accompanied with too free indulgence in alcoholic stimulants. 7. Diseases of the liver may arise by extension from neighbouring structures; or by the convection of morbid materials from more or less distant parts. The latter may be best illustrated by *pyæmia*; and secondary affections of this kind are believed to be particularly frequent in the liver when the morbid products are conveyed directly from the alimentary tube, especially in connexion with ulceration, dysentery, or certain operations. 8. Obstruction to the circulation, due to some forms of cardiac disease, is an important cause of some hepatic affections. 9. Disorders of the liver are often attributed to various hygienic errors, exposure to cold, and other causes, but how far this conclusion is justified in particular cases is a matter of doubt.

**CLINICAL SIGNS.**—So far as the actual diseases of the liver are concerned, the clinical phenomena to be looked for and studied lie within a limited range, and may be grouped under the following heads: 1. *Morbid sensations*, referred to the hepatic region, or to the shoulder, such as pain of various kinds, tenderness, sense of weight, throbbing. 2. *Symptoms due to interference with the biliary functions*, particularly jaundice and its accompanying phenomena. 3. *Symptoms resulting from more or less obstruction to the portal circulation*. These include digestive disorders, due to congestion or catarrh of the mucous membrane lining the stomach and intestines; hæmorrhage from this membrane in some cases, leading to hæmatemesis and melæna; ascites, which is a most important symptom; enlargement of the spleen; hæmorrhoids; congestion of the womb in women, it is said; and, in certain conditions, dilatation of the veins of the abdominal wall. 4. *Symptoms produced by the pressure upon, or other interference with, neighbouring structures, of an enlarged liver*. Thus, the organ not uncommonly extends upwards, pushing up and limiting the movements of the diaphragm, and pressing upon the lung, hence causing dyspnoea. Or it may

compress vessels and other structures; or in some cases it even interferes with the heart's movements. 5. *Physical signs.* These indicate enlargement or contraction of the liver; changes in situation or shape; changes in physical characters; and rarely pulsation. 6. *General symptoms.* These may be more or less independent of the hepatic disease, this being merely a part of a general malady; or the liver-affection may give rise to pyrexia, wasting, and other symptoms affecting the system at large. The absence of any such phenomena is important in the diagnosis of certain affections of the liver. Hepatic derangements are supposed to originate many general and remote symptoms, not obviously connected with this organ; and some authorities attach much importance to these disorders in the development of lithæmia and gout. See GOUT.

The individual diseases of the liver will now be discussed in alphabetical order.

FREDERICK T. ROBERTS.

**LIVER, Abscess of.**—SYNON.: Hepatic Abscess; Fr. *Abcès du Foie*; Ger. *Leberabscess*.

Abscesses of the liver are divisible into two classes: (1) tropical abscess; (2) other forms of abscess, including those resulting from the following morbid states: pyæmic conditions, especially such as arise from typhlitis and surgical operations or affections about the rectum; lobular hepatitis, as described by Sir Richard Quain (*Trans. Path. Soc. of Lond.* vol. iv. 1853); suppurating hydatid cysts and other parasites; forms of traumatism, especially blows on the liver; gall-stones; and pylophlebitis. The latter class fall to be considered under their appropriate heads. Here it may be remarked that, although their ætiology and pathology are widely different from those of tropical abscess, their diagnosis and treatment are practically the same. Tropical abscess, as its name implies, usually originates in tropical or sub-tropical climates; the other forms are not so restricted.

**Tropical Abscess of the Liver.**—ÆTIOLOGY.—The association of abscess of the liver with dysentery has long been recognised, and the more the subject is investigated the more constant is this association found to be. So much is this the case that some recent writers incline to regard the terms 'tropical abscess' and 'dysenteric abscess' of the liver as synonymous. Without going quite this length, it may safely be asserted that in 75 per cent. of cases of hepatic abscess a history of some degree of dysentery—not necessarily of a very urgent character—can be elicited. It must be recognised, however, that in this association the order of events is not always the same: usually the dysentery is antecedent to the hepatitis; sometimes the diseases are co-

incident; more rarely the hepatitis precedes the dysentery; and in not a few cases the two conditions alternate. The advocates of the ætiological identity of the two diseases suggest that in a proportion of cases in which dysentery is not a feature either before, during, or after the occurrence of abscess, the pathological drama has not been played out. They argue that, just as dysentery may exist without hepatitis, hepatitis may exist without dysentery. Whatever may be the exact relationship between these diseases—a relationship succinctly expressed by the term 'hepatic dysentery,' devised by the clinical acumen of Annesley long before there was much theorising in the modern sense—it is certainly a very intimate one, and one which should always be borne in mind in the diagnosis of hepatic abscess, as well as in the prognosis of dysentery; and it should also be borne in mind that the abscess may not develop till months after the dysentery has been recovered from, and the patient perhaps has left the endemic area.

The most potent circumstance in predisposing to hepatic abscess is residence in a hot climate, more especially if the heat is combined with humidity and malaria. There are exceptions, but, speaking generally, liability to this form of abscess increases *pari passu* with these conditions. Intemperance in eating and drinking; lazy, luxurious habits; bad food; hardship and exposure, such as are incident to a soldier's life in time of war; hot, close, and overcrowded barracks; and unhygienic conditions in general have a marked though secondarily predisposing influence. Men are more subject to it than women, adults than youths; children seem to enjoy almost an absolute immunity. Race has undoubtedly some influence, but how it operates—whether in consequence of peculiarities of habit, acclimatisation, or specific racial idiosyncrasies—has not been adequately determined. It is certain that liver-abscess is much more common in Europeans frequenting hot climates than in the natives.

Among the circumstances which determine the formation of abscess in persons predisposed, either by dysenteric hepatitis, or by those subtle and as yet unformulated physiopathological changes in the liver-tissue associated with life in the tropics, are, above all, chills of the surface such as arise from rapid alternations of heat and cold, from sitting in draughts in wet clothes, from bathing in cold water, or sleeping without adequate clothing. A blow on the liver, a bout of drinking, or a surfeit, is often the starting-point of the disease.

**ANATOMICAL CHARACTERS AND PATHOLOGY.**—Tropical abscess of the liver may be single or multiple, and it may vary in size from a minute collection of pus to an enormous sac containing many pounds of fluid, and occupying a large extent of the

liver. When a section of a liver in an incipient state of suppuration is made, one or more circular patches, greyish-yellow or otherwise altered in colour, and surrounded by an area of congestion, are revealed. Abscess is formed by the breaking down of one of these patches, liquefaction commencing in the centre. Frequently two or more neighbouring patches coalesce. When the abscess has attained any size its walls are found to be very irregular, their inner surface being coated usually with a yellowish, tenacious purulent material lying on a thin zone of more or less infiltrated liver-tissue—the abscess sac—which in its turn is surrounded by a hyperæmic zone; beyond this the liver-tissue may be fairly healthy. Around and, so to speak, in the walls of the principal focus of suppuration a number of minute abscesses may form, and it is by the bursting of these into the main cavity, and the necrosis of pieces of intervening tissue, as well as by the slower process of purulent and molecular disintegration of its walls, that the cavity enlarges. The contents vary considerably: usually they are of a chocolate-brown colour, and have a thick, gummy consistence tending to become gelatinous on cooling. The odour too is peculiar. Not infrequently lumps of necrosed liver-tissue or clots of blood are found floating in the fluid, which may also be streaked with blood, bile, or a yellow, mucoid-looking pus of a paler colour. Occasionally the pus is more fluid, and may have the appearance of ordinary laudable pus; rarely it is thin, watery, or grumous. It frequently contains micro-organisms, but in not a few instances the bacteria of pus are absent. It is further characterised in a certain proportion of cases by the presence of a protozoic organism—the *amœba coli*. This parasite consists of a minute mass of plastic protoplasm, divisible into an imperfectly defined and granular endosarc, and a homogeneous and very pellucid ectosarc. At low temperatures the *amœba* ceases to move, and assumes a globular form, having a diameter equal to about that of three or four blood-corpuscles. At the temperature of the body it exhibits active and characteristic amœboid movements. It is readily detected by a magnifying power of from 80 to 100 diameters, especially if the amœboid movements are encouraged by slightly warming the slide. See ENTOMOZOA.

When the abscess is single its most usual situation is towards the back part of the right lobe, but it may occupy the left lobe or any other part of the gland. When abscesses are numerous, they are scattered throughout the liver, especially near the peritoneal surfaces.

Death may occur before the abscess ruptures; rarely it becomes encysted or absorbed; usually it makes its way to the surface of the organ and, after adhesions

have been formed between opposing serous surfaces, opens on the surface of the body or into some of the neighbouring viscera. The most usual direction in which rupture occurs is into the right lung, next in frequency the intestinal tract, less frequently the surface of the body, and rarely into the pleural or peritoneal cavities or into the pericardium, gall-bladder, inferior vena cava, or calyx of the right kidney. Rupture into the pericardium or vena cava is necessarily and speedily fatal, but in a case recently recorded by Mr. Hulke recovery followed surgical interference after a hepatic abscess had opened into the peritoneum. Recovery may speedily ensue after rupture of the abscess, more especially if it opens into the lung or intestinal canal; but in this event, in the majority of instances, the necrotic process is not arrested, and the patient sinks after perhaps a long illness. Recovery after surgical operation, even in the case of very large abscess, is often remarkably rapid, a great cavity of many ounces capacity healing up perhaps in a week or two.

Of the intimate pathology of hepatic abscess little, if anything, is known. By the discovery of the *amœba coli* in connexion with this disease and dysentery, the question seems to have entered on a promising field. Kartulis, and more recently Osler, Councilman, and Lafleur have shown that this parasite is present in a form of dysentery, characterised by its peculiar obstinacy and tendency to relapse, and which has been named *amœbic dysentery*. Moreover, the latter observers have shown that the *amœba* not only occurs in the dysenteric and hepatic discharges, but also penetrates deep into the bases of the dysenteric ulcers, and into the tissues surrounding hepatic abscess. These circumstances, to say the least, are significant. Moreover, the *amœba* has been found in dysentery and in liver-abscess in India (Hehir), in Africa, in Europe, and in America. That it is not invariably pathogenic is proved by its being sometimes present in the healthy feces of apparently healthy individuals, so that it has yet to be shown that it is the real cause of these diseases, and not merely an occasional epi-phenomenon.

With reference to the association of dysentery and hepatic abscess, it ought to be mentioned that in some epidemics of dysentery abscess is of more frequent occurrence than in others, and this has been remarked, not only in epidemics in the tropics, but in epidemics occurring in Europe. In many of the latter hepatic abscess is almost unknown, but in others it is not so rare, as in the Dublin epidemic of 1818. Manifestly there is a specific element common to certain types of dysentery and hepatic abscess; but what this is, and how it operates, has not been determined. At one time it was held that the

abscess was caused by the absorption of pus by the portal vein from the dysenteric ulcers, and from suppurating phlebitis which was supposed to be present in the intestinal radicles of that vessel. But no such phlebitis was demonstrated; and, moreover, it was shown that the hepatitis may precede the dysentery, and therefore could not be caused by it. Further, as hepatic abscess is by no means a frequent sequela of gastric ulcer, of typhoid fever, or of tubercular or malignant ulceration of the intestine, the ulceration of the bowel in dysentery cannot *per se* be regarded as the cause of tropical abscess of the liver.

**SYMPTOMS.**—Sometimes these are fairly acute, but in the great majority of cases they are of an extremely subdued and insidious character, so much so that sometimes the first intimation of the presence of abscess in the liver is its rupture into the lungs or intestine, and a discharge of the peculiar chocolate-coloured pus. Very often the symptoms amount to little more than feelings of ill-health, vague uneasiness about the region of the liver, and a state of low irregular feverishness. Temperature in the morning may be but slightly if at all above the normal, rising during the day to 101° or 102°; or there may be spells of complete apyrexia or, occasionally, of smart fever. Profuse sweating, particularly during sleep, is generally very marked. Slight feelings of chilliness occur from time to time, and in a few cases smart rigors are met with; but too much importance must not be attached to their absence. The complexion may be muddy, and the scleræ slightly icteric; the urine, though often loaded and dark, is seldom icteric, and marked jaundice is rare. The temper is generally very irritable, and the spirits depressed. The tongue is sometimes coated; at other times it is clean. Appetite is usually poor; but occasionally it is fairly good. Vomiting may occur, especially if the left lobe of the liver is involved. The bowels may be confined; generally they are loose or irregular, often dysenteric. A short dry cough and some dyspnoea are not uncommon. The decubitus is usually dorsal or on the right side. In some cases pain of a cutting character in the region of the liver is a distressing symptom, and is regarded as evidence of perihepatitis, and the formation of adhesions between the peritoneal surfaces. In other cases the pain is of a very subdued type—more a sense of weight and uneasiness; but it is usually intensified by pressure or percussion or smart compression, and it is often observed that percussion over the lower part of the abdomen may cause pain not where the blow falls, but in the region of the liver. Firm pressure on the liver often gives rise to a feeling of nausea or faintness. Pain in the right shoulder or scapula is one of the classic symptoms of hepatic abscess, and is regarded as an indication that the mischief is in the neighbourhood

of the diaphragm. The abdomen is generally on inspection seen to be motionless and full. The liver is enlarged, but not always to a great extent; the enlargement may be upwards, a more significant sign than when it is enlarged in a downward direction. There may be a general bulging of the right hypochondrium, and obliteration of some part of one or two intercostal spaces, with a degree of oedema or even redness of the integuments. In wasted patients with normal livers the epigastrium should be scaphoid when they are lying down, but in abscess of the liver it is often flat or even bulging, and this is more apparent when the patient is made to stand up; this appearance, with fever and tenderness, is always a suspicious symptom. Friction or crepitation may be heard at some part of the base of the right lung; the presence of these indicates proximity of the abscess to the diaphragm; and if, in addition, the amœba coli is found in the sputum—as Councilman and Lafleur have pointed out—rupture of the abscess into the lung is impending. When this occurs, a thick, gummy, chocolate-brown pus—quite pathognomonic in appearance, and yielding the reactions of bile when tested chemically, and exhibiting granular and degenerated liver-cells under the microscope—is brought up. In consequence of the persistent coughing after the abscess has opened and partially emptied, there may be much hæmorrhage from its walls, and a very bloody sputum; so much so that the great bulk of the expectoration is blood, with here and there only a streak or pellet of the chocolate-brown pus. This appearance is very apt to deceive the unwary, and to lead to a diagnosis of pulmonary hæmoptysis. The stethoscope may detect approximately the point of entrance of the abscess into the lung, and occasionally it reveals peculiar and very loud bruits, synchronous with the pulse, somewhere about the upper border of the hepatic area, and sometimes of a to and fro character. These bruits probably arise from a to and fro play of air out of and into a partially empty but uncollapsed abscess-sac through a narrow sinus, and are produced by the jog communicated to the inflated abscess-sac by the systole of the ventricles. Should the abscess open into the bowel there may be one or two loose stools of a similar chocolate-brown character; and in a case of suspected abscess of the liver the physician ought always to bear this in mind, and inspect any loose motions reported to him. Rupture into the stomach may be followed by vomiting; if into the pericardium or peritoneum, by sudden collapse.

**DIAGNOSIS.**—This is often a matter of great difficulty, even to the most experienced, and at times only to be settled by the development of events, or the use of the aspirator. An excellent rule for the practitioner in countries in which hepatic abscess is endemic is as follows: Whenever persistent

ill-health is associated with a low degree of fever, and symptoms—no matter how trifling—referred to the abdomen, which cannot be readily accounted for, suspect abscess of the liver, and examine the organ daily with this possibility in view, and inquire carefully for a history of dysentery. It must be borne in mind, too, that the dysentery may have been very trifling, called perhaps diarrhoea, and that it may have occurred several months before the health began to break down. The significance of enlargement of the liver, localised hepatic swelling, pain, tenderness, a fixed and motionless abdomen, rigors, and a hectic temperature following dysentery can hardly be misunderstood. But it is seldom that in the early and more hopeful stages of abscess the indications are so unequivocal; indeed it may be, and often is, entirely overlooked, and not discovered until it has burst into lung or bowel, or until it is found on the *post-mortem* table. The presence of pneumonic crepitus about the base of the lung in liver cases is often misinterpreted; rightly understood, it is a most valuable confirmatory sign, especially if it is limited to a small patch. Sometimes the periodicity of the fever, and the severity of the rigors, and regular procession of pyrexia and diaphoresis in hepatic abscess, lead to a diagnosis of ague; but the impotence of quinine to cure the fever, and the absence of the plasmodium malarie in the blood, and of splenic enlargement—which is nearly always present when the liver is affected in malarial hepatitis, and which rarely if ever ends in abscess (Kelsch and Kiener)—ought to awake suspicion. When in a malarial subject there is a history of dysentery, and both liver and spleen are enlarged, diagnosis in the early stage of hepatic abscess is nearly impossible without the aid of the aspirator. In any case where grave doubt exists, where fever is persisting, and the patient is manifestly losing ground, exploration with a medium-sized aspirator-needle must not be delayed. This trifling operation, if attended with any risk, is infinitely less dangerous than procrastination. With proper precautions it is not a dangerous proceeding; so far from doing harm it has often a markedly curative effect in cases of hepatitis, even when there is well-marked local bulging. As an encouragement to early recourse to the aspirating needle, it ought to be borne in mind that hectic may occur in hepatitis before the formation of pus, and even in cases in which pus never does form; and the whole trouble which otherwise might have ended in abscess may rapidly subside after exploratory needling. In exploring the liver with the aspirator, in the absence of definite localising indications, the needle ought to be entered in the first instance about the seventh or eighth interspace in the anterior axillary line; if pus is not found here, a second and third puncture should be

made, the needle being driven in to its full extent if necessary, in front just below the ribs a little to the inside of the nipple-line, and again behind in a line with the angle of the scapula well below the edge of the lung.

**PROGNOSIS.**—It is impossible in the present state of medical knowledge to say how dysenteric hepatitis may terminate, whether in resolution or abscess. Should the latter form, provided it is single and opened soon and effectually drained, the prognosis is fairly good; quite 50 per cent. recover. If left too long or allowed to open of itself, the prognosis is bad, though by no means hopeless. If two or more abscesses are present, the chances of recovery are proportionately diminished; and if there are many suppurating points scattered through the liver, death is inevitable. If after operation the temperature quickly becomes normal and keeps so, prognosis is favourable; but if, on the other hand, after operation there is a distinct evening rise and a continuance of the hectic, the worst may be apprehended.

**TREATMENT.**—Hepatitis threatening abscess ought to be treated by absolute rest in bed, low diet, and, in the absence of diarrhoea or dysentery, saline aperients, such as sulphate of sodium. The right side ought to be covered with a large, hot, frequently renewed poultice, a foot in breadth and three feet in length, and which should pass from beyond the middle line behind to beyond the middle line in front. If there is dysentery, a thirty-grain dose of ipecacuanha should be given, and repeated as in ordinary dysentery (*see* DYSENTERY). In the absence of intestinal trouble, chloride of ammonium in twenty-grain doses every six hours is sometimes exhibited with advantage. Quinine is indicated in malarial complications.

Symptoms not improving, exploration with the aspirator must not be too long delayed. If pus is found, the abscess should be opened forthwith, thoroughly drained, and treated on Listerian principles. The different methods of operating do not fall to be considered here, but it may be remarked that in no surgical affection is it more necessary to observe the grand surgical principles of early opening, free drainage, and perfect asepsis. By these means of late years the mortality in this affection has been enormously reduced, and a diagnosis of liver-abscess is now no longer to be regarded, as it was in former days, as almost tantamount to sentence of death. The opinion of the physician is sometimes asked as to the propriety of making an external opening, in the case of an abscess discharging through the lungs or intestinal canal, or from an inefficient sinus in the walls of the chest or abdomen. A good rule to observe in such a case is this: If the patient is improving, fever absent, and discharge lessening, do not counsel interference; if, on the other hand,

hectic persists, the discharge increases or does not diminish materially, and the patient continues to lose ground, advise operation. If there be doubt about which course to advise, recommend change of air, which has sometimes a wonderful influence in wavering cases in inducing healing. Should fever and ill-health persist after the abscess has been freely drained, and the drain, though effective, give vent to very little discharge, suspect the presence of a second abscess, and advise further exploration, and—if pus is found—further operation. Recovery has followed after several abscesses have been so treated in the same individual. If the temperature keeps up after all abscesses have been opened, and discharge from the drainage tube is profuse, it is an indication that the necrotic process is still going on in the liver; the prognosis is then most unfavourable. Gangrene sometimes sets in around the operation-wound, and is almost invariably fatal.

When pus has formed, the diet ought to be fairly liberal; but overfeeding and overstimulation are under all circumstances to be carefully avoided.

PATRICK MANSON.

**LIVER, Albuminoid Disease of.**  
 SYNON.: Waxy Liver; Lardaceous Liver; Fr. *Dégénérescence Amyloïde du Foie*; Ger. *Amyloïde Entartung der Leber*.

**DEFINITION.**—A disease characterised by painless, more or less considerable, enlargement of the liver; due to the existence in its structure of a peculiar homogeneous substance, the exact nature of which is not known, but which has a marked relation to certain cachexias and constitutional maladies.

**ÆTIOLOGY.**—Albuminoid disease of the liver occurs in association with constitutional syphilis and diseases attended with protracted suppuration, or other protracted exhausting discharges. It has occasionally been found with chronic bone-disease, more especially of the spine, without evidence of any adequate discharge; and it is known to occur occasionally in new-growths, more particularly in the liver. It has been noticed in connexion with chronic dysentery, but the records of the Seamen's Hospital do not confirm such association. In many cases of chronic ague, with marked cachexia, which have been admitted into the hospital just named, there was an enlarged hard liver, pointing to albuminoid change; but there was probably in these cases the superaddition of syphilitic taint. Rokitansky speaks of the disease as congenital in children born of syphilitic parents.

**ANATOMICAL CHARACTERS.**—The liver has its normal shape; is more or less enlarged, sometimes to such an extent as to fill the greater part of the abdominal cavity; and is

firm, resistant, and inelastic, with a smooth, glistening surface. The organ cuts like bacon, hence the name 'lardaceous.' The cut surface is a translucent grey, like gelatine or boiled sago, or, in advanced cases, a peculiar subdued red, like that of raw ham; but sometimes it is yellowish or speckled, the liver being anæmic and often fatty also. From the incised veins a little pale blood usually oozes. The application of solution of iodine to the cut surface causes change of colour, which has been described as reddish-brown, mahogany-brown, or walnut, by different observers. For microscopical examination, a weak solution of methyl-aniline violet is at once delicate and distinctive. Supposing a lobule of the liver to be divided into three zones, the characteristic iodine or other stain will be seen, in less advanced stages of the disease, to be limited to the middle zone, where the hepatic artery is distributed; the vessels and cells here being filled with the new material, which afterwards may extend so as to implicate the entire lobule. The structures invaded by the new material have, in a section examined microscopically, a lustrous, transparent, and somewhat swollen appearance. When the entire lobule is affected, the aspect is homogeneous. The appearance of an albuminoid liver may be modified by the co-existence of fatty change, or cirrhosis, or syphilitic disease. The spleen is generally, and the kidneys are not infrequently, implicated.

**SYMPTOMS.**—Palpation, in marked cases of albuminoid disease of the liver, will readily detect a large, hard, resistant tumour, having the normal outlines of this organ; the smoothness of its surface; and the extent to which it encroaches upon the abdominal cavity. Pressure does not elicit any tenderness, nor is there usually any pain; at most, in advanced cases, there is only a sense of tension and fulness, as in other hepatic enlargements. The painless nature of the tumour is distinctive. The disease does not interfere with the portal circulation, and does not therefore directly cause ascites. When this occurs, it is the result of general cachexia, induced by the constitutional malady, and perhaps by associated renal complication. The dropsy generally affects the legs in the first instance, and afterwards the serous cavities, and is not a prominent symptom unless the kidneys are implicated. In this case the urine is usually of low specific gravity and albuminous, and the anæmia very marked. The system of bile-ducts not being obstructed by the disease, there is no jaundice; or if this occur, which is a rare event, it is from pressure on the duct externally by enlarged lymphatic glands. The evacuations are, however, frequently of a pale yellow, and at times of a clayey, colour, which may be accounted for by the extensive impairment of secreting structure,

and the consequent secretion of a poor, colourless bile. A lardaceous state of the spleen is a frequent accompaniment of the liver affection, and gives rise to increased volume and hardness of the organ. Vomiting, without the usual indications of gastric derangement, such as furred tongue, and diarrhoea, are symptoms not uncommon in advanced cases, and are due, according to Frerichs, to the implication in the disease of the vessels and villi of the stomach and intestines.

**DIAGNOSIS.**—The peculiar features of the enlargement, its painless character, the concurrence of the constitutional maladies already noticed, especially if with implication of spleen and kidneys, will distinguish an albuminoid liver from other kinds of hepatic enlargement. If there be associated cirrhosis or syphilitic disease, the diagnosis will be difficult; but, as Bamberger remarks, an error will not be of moment as regards prognosis and treatment.

**PROGNOSIS AND DURATION.**—The disease may run on for months or even years, but it generally proves fatal, either by intercurrent affections, or by anæmia, general dropsy, and exhaustion, such result being more rapidly determined when the kidneys are involved.

**TREATMENT.**—It is more than probable from the records of cases of late years, that if the discharge which produces it can be arrested—by the removal of diseased bone, for example, or the limb in cases of joint-disease—the abnormal product in lardaceous disease may be re-absorbed and the disease cured. Surgical measures, therefore, directed towards the closing of sinuses, or the stoppage of any discharge that may be present, are of the first importance. But there are many cases—in advanced phthisis, for example, where the position of the disease is a bar to treatment, or in syphilis, in which we are as yet quite in ignorance of the reason of the production of the morbid deposit—where the only treatment that can avail must be directed especially to the associated cachexia. Whether this be syphilitic or strumous, the preparations of iodine are indicated: the iodide of potassium, the tincture of iodine, or, where the anæmia is marked, iodine in combination with iron. The syrup of iodide of iron in drachm doses, three times a day, has proved useful, if not in reducing the tumour, at least in improving the general condition of the patient. The iodine mineral springs, as Woodhall Spa, Kreuznach, Adelheidsquelle, &c., are indicated, although they contain but infinitesimal doses of iodine and bromine. The baths of Aix-la-Chapelle, Ems, and Weilbach have each had its supporters in the treatment of this malady. Chloride of ammonium, in ten to twenty grain doses, three times a day, continued for some time, has been found to be efficacious in reducing large hard

livers (Budd, Begbie, Stewart). The general therapeutical indications are pure air; plain nourishing diet; the regulated use of alcoholic stimulants; and adequate protection of the skin by warm clothing and other measures.

STEPHEN H. WARD. JAMES F. GOODHART.

**LIVER, Apoplexy of.**—By this is meant hæmorrhage into the liver, in the form either of isolated patches of extravasation or of general effusion, the whole of the hepatic parenchyma being converted into a dark-red pulpy mass. This affection is rarely met with in this country, but has been often observed abroad in warm climates and malarious districts, as a result of disease of the liver, or prolonged and intense congestion. It occurs also in some cases of scurvy. Abercrombie believed that the puerperal condition predisposed to hepatic apoplexy. It has been observed also, according to Frerichs, in some new-born infants after long labours, and in cases of this kind it is usually associated with pulmonary atelectasis. A rapidly fatal case of hepatic apoplexy was reported by Andral, in which there were no indications of any efficient cause of the hæmorrhage (*Clin. Med.*, 3rd ed. t. ii. p. 259). Extravasation of blood into the substance of the liver, together with a pulpy condition of more or less of the parenchyma, may be produced by the application of violence to the hepatic region.

**SYMPTOMS.**—The symptoms that have been observed in cases of hepatic apoplexy are pain in the right hypochondriac region, and excessive tenderness; jaundice; bilious vomiting; melæna; a cold and bloodless condition of the skin of the face and limbs; and in some cases syncope.

This affection is almost invariably fatal when associated with previous disease of the liver, or with extensive laceration.

W. JOHNSON SMITH.

### LIVER, Atrophy of, Acute Yellow.

**SYNON.**: Fr. *Atrophie Jaune Aiguë du Foie*; *Ictère Grave*; Ger. *Acute Atrophie der Leber*.

**DEFINITION.**—This is a general disease, likened by Trousseau to a pyrexia. The jaundice, being so prominent a symptom, formerly drew attention too exclusively to the liver; but the same degeneration which seizes upon the liver, likewise attacks all the glandular and muscular organs of the body. The morbid change is a parenchymatous degeneration, called by Virchow and his school a parenchymatous inflammation. It consists in a filling of the cells of a gland with albuminous granules, in such numbers as altogether to hide the nucleus; the albuminous granules quickly degenerate into oily particles and drops. These morbid appearances are found in poisoning by phosphorus, arsenic, antimony, alcohol, and other agents, and in all fevers, though in a less degree than in acute yellow atrophy. Buhl was the first to

point out that the pyrexial changes were the beginnings of acute yellow atrophy.

**ÆTIOLOGY.**—Acute yellow atrophy is perhaps the rarest of all the diseases occurring in this climate. Of its causes, next to nothing is known. It seems to be more common in women than in men; and in pregnant women than in others.

It has been shown that in pregnant and suckling quadrupeds and laying hens, the liver and kidneys often show cells infiltrated with fat, a fact which may throw some light on the disposition of pregnant women to acute yellow atrophy. Emotional disturbances, such as grief and trouble, and bad hygienic conditions, have been thought by some to predispose to this disease. It is occasionally induced by alcoholism, and may supervene upon cirrhosis. Some believe that all cases may be traced to phosphorus-poisoning.

**ANATOMICAL CHARACTERS.**—After death it is not uncommon to find the liver of natural size, or even enlarged, in the early stages of acute atrophy. Later on, the organ shrinks, so that in extreme cases it may weigh as little as nineteen ounces; or even only thirteen ounces, as in the case of a girl of seventeen, recorded by Clements. It decreases in all diameters, but the left lobe is especially shrunken. The capsule is often wrinkled. On section, there is no longer any appearance of lobules, but an ochre-coloured surface without definite structure, often interspersed with reddened patches. The gall-bladder and bile-duets are either empty or contain a grey mucus. The yellow and red appearances are blended. The yellow represents the early and acute stage of the disease, and is therefore mostly present in acute cases; the red represents the later changes, in which most of the liver-cells have disappeared, the colour being partly due to distension of blood-vessels. Under the microscope, the liver-cells are found, in the early stages, to be filled with granules, which completely hide the nucleus ('cloudy swelling'); part of these granules are soluble in acetic acid, others are not. Reddish-brown rhombic crystals, resembling hæmatoidin, have been described by Dr. Coats, amongst the detritus of the liver-cells, as well as crystals of leucin and tyrosin, which have been found by most observers, especially when the liver has been kept for some time. Later on, all trace of liver-cells may be lost, nothing but a granular and oily detritus and pigment being seen under the microscope, whilst the portal canals contain a greater or less number of migratory cells.

Micrococci have been observed in the liver by Waldeyer, Klebs, and Eppinger, but their pathological significance is doubtful.

The spleen is enlarged and soft in the great majority of cases. The stomach and alimentary canal present dark-red or tarry con-

tents, the outcome of hæmorrhage; the tubular glands of the stomach are filled with fatty degenerated epithelium. The muscular tissue of the heart shows likewise fatty degeneration; and the tubules of the kidneys are filled with epithelium in various stages of fatty degeneration.

**SYMPTOMS.**—Acute yellow atrophy is commonly preceded for some days or weeks by a simple jaundice, in which nothing peculiar can be made out. Delirium and convulsions then suddenly set in, followed by deep coma, stertorous breathing, and dilated pupils. During the first part of the disease the pulse is natural or reduced in frequency, but with the appearance of the convulsions and delirium it rises to 120 or 130. The skin is always yellow, rarely deeply coloured. The urine is natural in quantity, bilious, containing leucin and tyrosin; towards the end of the disorder it also contains no urea, chlorides, or phosphatic earthy salts; a kind of peptone is present. In some cases it is necessary to evaporate the urine to a concentrated condition to detect the crystals of leucin and tyrosin (*see* LEUCIN; and TYROSIN). There is almost always constipation; the stools being at first pale, afterwards black from admixture of blood. Vomiting is very constantly present; at the end of the disease, of a black coffee-ground matter. The right hypochondriac and epigastric regions are painful and tender. The liver, at first natural in size, or even larger than natural, decreases daily in dimensions, so that at last percussion may give no liver-dulness at all, owing to the softened condition of the liver causing it to become folded on itself, the intestines rising up to take its place. With the decrease of the liver, the spleen increases in size. A hæmorrhagic diathesis likewise sets in, as shown by petechiæ on the skin, epistaxis, hæmatemesis, and melæna. The temperature is commonly low, until just before death.

**DIAGNOSIS.**—The diagnosis of acute yellow atrophy is very difficult, and may remain doubtful after death. The rapid shrinking of the liver, as evidenced by diminution of the hepatic dulness, associated with the discovery of leucin and tyrosin in the urine, the occurrence of grave cerebral symptoms, and the absence of pyrexia, are certain symptoms of acute atrophy. Poisoning by phosphorus can hardly be distinguished from acute yellow atrophy, unless the patient own to having taken the drug. The prodromal stage cannot be distinguished from simple jaundice.

**PROGNOSIS.**—The prognosis is extremely bad: only a very few suspected cases are known to have recovered.

**TREATMENT.**—The treatment must be conducted upon general principles. A few cases, in which the diagnosis of acute yellow atrophy has been thought justifiable, have recovered, and these have been treated with the mineral

acids and saline purgatives, aconite, quinine, and camphor. These are therefore the remedies which may be recommended to be used. Local symptoms, such as vomiting or bleeding, must be treated as in other diseases.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

### LIVER, Atrophy of, Chronic.—

Chronic atrophy of the liver is seen in many wasting diseases, and in old age; the liver then often shrinks, becoming tougher in consistence, but rarely granular on the surface. The cut surface is dark red or pale brown; the acini are either invisible, or else smaller than natural. Frerichs thinks that the blood-vessels are all dilated. The increased toughness seems due to the atrophy of the liver-cells, the meshes of the connective-tissue network being thus brought nearer to each other.

The symptoms of chronic atrophy are merged in those of the primary disease, against which treatment must be directed.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

### LIVER, Biliary Accumulation in.—

**ANATOMICAL CHARACTERS.**—When a permanent obstruction to the flow of bile into the duodenum has been set up, serious changes take place in the gall-ducts and the liver itself (*see* GALL-BLADDER AND GALL-DUCTS, Diseases of). At first the liver swells, apparently from the pent-up secretion. It becomes of a deep bilious or olive-green colour, the central parts of the acini being the deeper coloured; on section the dilated ducts are seen, and bile or a colourless fluid wells out of them. Increase in the consistence of the liver commences; and if the obstruction continue, the organ wastes, becomes much tougher, and shows a granular surface. This increase in consistence is due to an overgrowth of the connective tissue of the liver, as in cirrhosis, only to a less degree. The amount of overgrowth depends upon the kind of obstruction. It is greater when a rough angular gall-stone is the cause, than when a hydatid tumour with its smooth walls presses upon the gall-ducts. This overgrowth springs at first from the gall-ducts, which are greatly thickened, and thence spreads over the connective tissue of the portal canals.

The liver-cells atrophy, as in cirrhosis. They vary much in size. Their contents seem to be chiefly fat and pigment-granules, though neither is of very great amount as a rule. The arrangement in rays around the hepatic venule is quite lost. One of the most important functions of the liver is the preparation of glycogen, and this function seems to be abolished in long-continued jaundice. In animals whose bile-ducts were tied, Dr. Wickham Legg found the glycogen to disappear not many hours after the ligature was applied; and after puncture of the

fourth ventricle, no sugar appeared in the urine.

In some cases of complete obstruction to the bile-ducts, the liver-cells have been found altogether destroyed, nothing but a fatty detritus being seen under the microscope. This is not owing simply to *post-mortem* changes in the liver; but is possibly due to the long-continued action of the bile-acids circulating in the blood upon the liver-cells themselves, as Leyden has pointed out. It is not owing to the simple solution of the liver-cells in the bile, for the bile has not the power of dissolving these cells, as Th. von Dusch has asserted. In some cases suppuration takes place in and around the bile-ducts—suppurative cholangitis.

**SYMPTOMS.**—As regards the clinical phenomena of biliary accumulation in the liver, there are, of course, all the symptoms of jaundice and of the disease which leads to it. In addition, the liver at first swells, and may be detected below the ribs for two or three fingers' breadth, but rarely more; it is often painful on palpation. Later on, the liver retreats within the boundaries of the chest. Ascites sometimes shows itself, owing to the disturbance of the circulation in the liver; and the spleen often swells. Fever of an intermittent or remittent character sometimes takes place, associated, it may be, with recurring rigors, which may simulate ague. The long precedence and persistence of jaundice, with, in many cases, history or evidence of the cause of obstruction, enable it to be distinguished from ague. All these symptoms are, however, liable to be interfered with by the primary disease.

**TREATMENT.**—The treatment must be directed to the cause of the obstruction of the ducts.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

**LIVER, Cirrhosis of.**—**SYNON.**: Granular Liver; Hob-nailed Liver; Gin-drinker's Liver; Interstitial Hepatitis; Fr. *Cirrhose du Foie*; Ger. *Chronische interstitielle Leberentzündung*.

**DEFINITION.**—A chronic disease of the liver, in which the organ becomes hardened, and usually more or less diminished in size, at the same time assuming a granular or hob-nailed appearance; these changes resulting from an increase in the connective tissue, usually caused by abuse of spirituous liquors. The name *cirrhosis* was first given by Laennec to the hardened and shrunken liver, on account of the yellow colour of the granulations in this disease.

**ÆTIOLOGY.**—The most common cause of cirrhosis is, undoubtedly, the abuse of spirituous liquors. Spirits, unmixed with water, seem to be more potent in causing cirrhosis than wine or malt liquors. Next after these, but at a great distance, come syphilis, and the immoderate use, it is said, of spices—

such as curry, pickles, and powerful sauces, or of coffee. In some rare cases no cause is apparent. The fact of its occurrence in cases (rare it is true) where there have been no alcoholic excesses, and the negative results of experimental attempts to produce the disease in animals by long-continued administration of alcohol, raise the question whether when due to alcohol it is referable to the action of it *per se* on the liver, or to its indirect effects on the intestine, &c. The late Dr. Moxon suggested that in non-alcoholic cases it might be due to extension of inflammation from the peritoneum; and since then Dr. Goodhart, Sabourin, and Brieger have recorded its occurrence in tubercular peritonitis. The disease is far more common among men than women; it is very rare indeed in children. In some of the latter cases, the child has been proved to have been in the habit of drinking spirits. Cirrhosis has also been seen among the lower animals, a proof that alcohol is not the sole cause.

**ANATOMICAL CHARACTERS.**—The seat of the disease in cirrhosis is the capsule of Glisson. The connective tissue, which accompanies the vessels entering at the portal fissure, and which forms a covering for the liver beneath the peritoneum, takes on a very active overgrowth. One result of this overgrowth is compression and atrophy of the secreting cells of the liver. Another is a hindrance to the flow of blood through the liver; for, although new vessels do indeed form in the new connective tissue, yet these are by no means enough to carry on the circulation, in the place of those obliterated or destroyed by the advancing overgrowth of connective tissue.

There are several varieties of cirrhosis. In the first, the *atrophic*—that which is most common—the liver is shrunken, it may be to one-half or one-third of its natural size. This shrinking is often greatest in the left lobe, so that this may become a mere appendage to the right. At the sharp edge of the liver, there is often nothing left but a semi-transparent tissue, containing none of the elements of the gland. False membranes often join the surface of the liver with the diaphragm or other neighbouring parts. The surface itself is greatly roughened. It shows numberless granulations, varying in size from a poppy-seed to a hazel-nut. The fibrous investment of the liver is greatly thickened; and the peritoneum either tears off in layers, or leaves a granular surface behind. The liver is exceedingly hard and tough; and on section, the cut surface is seen to be made up of yellow islets, imbedded in a white translucent tissue. These yellow bodies are the representatives of the granulations seen on the outer surface, and they are the remains of the natural liver-tissue, separated from

one another by the new white connective tissue.

This is by far the commonest variety of cirrhosis, but there are others. One form is *hypertrophic* cirrhosis, in which the liver is greatly increased in size, sometimes being more than double its natural weight; but the surface is smooth, and the capsule, though thickened, leaves a smooth surface when torn off. There is toughening of the liver, though not to so great a degree, and the same appearance of the cut surface as in ordinary cirrhosis. In another variety the organ is shrunken, but the surface is smooth, and on section are seen only pins'-points of yellow tissue in the white translucent overgrowth. Whether the hypertrophic variety ever becomes shrunken is still undecided. A third variety is *fatty* cirrhosis, which may be mistaken at first sight for fatty liver. but the touch shows how tough it is. It sometimes floats in water. There is no everted edge, and on section no acini are to be made out; but the cut surface is indistinct, pale, and yellow. The surface of the liver is smooth.

Under the microscope, using a low power, the tissue of the cirrhused liver is seen to be broken up into islets, separated by broad bands of what looks like a highly nucleated connective tissue. The separation between the two appears sharply defined. In some cases the liver-cells may be seen infiltrated with fat. With higher powers, the most striking object in the field is the great abundance of what were once called nuclei, but now lymphatic corpuscles, in the new-formed connective tissue: these vary little in size or shape, nearly all being round or roundish. The prevailing opinion now is that they are emigrated leucocytes. They are arranged sometimes in clusters, sometimes in lines, and sometimes indefinitely. According to the arrangement of the cellular infiltration, the cirrhosis is said to be multilobular or extralobular; or insular, pericellular or intralobular. It is stated by some writers that in the atrophic form the cellular infiltration is multilobular, and in the hypertrophic unilobular or pericellular; but whilst this may be partially true, in both forms instances of both multilobular and pericellular infiltration are usually encountered. The origin of the clusters is uncertain; but it seems tolerably clear that the linear disposition arises from the obliteration of vessels carrying bile or blood. The connective tissue itself is highly fibrous; sometimes homogeneous or granular. The liver-cells themselves undergo great changes. They present fatty and pigmentary degeneration, lose their natural polyhedral shape, and become oblong, oval, or spindle-shaped. Between them the new connective tissue gradually insinuates itself, and the cells become lost in the advancing overgrowth. It is contended by Dr. Beale and Dr. Payne

that the process commences in the liver-cells. These changes in the liver-cells are of course best seen at the spot where the liver-tissue and the connective tissue join. In rare cases acute yellow or red atrophy supervenes. In some cases—especially it is stated in cases of hypertrophic cirrhosis—linear rows of cubical cells are found in the portal canals. These are regarded by some as newly developed biliary canaliculi, by others as degenerated liver-cells.

**SYMPTOMS.**—The first approaches of cirrhosis of the liver are commonly very insidious. Often one of the earliest symptoms is a dull pain in the neighbourhood of the liver. This is accompanied by signs of chronic gastric catarrh, of which morning sickness is, for the diagnosis of intemperance, of the greatest importance. Morning diarrhœa is sometimes an early symptom. The patients are commonly of a sallow, often almost jaundiced, complexion; at the same time the venules of the face are often distended and varicose, giving the patients' complexion a colour which to uneducated eyes is indicative of health. They grow thinner, and their strength fails. Some patients suffer from piles; in others diarrhœa occurs. Later on, the belly begins to swell, and ascites appears; the legs may become œdematous, from the pressure of the fluid in the belly on the anterior wall of the inferior vena cava. The urine is high-coloured; often deposits urates; and sometimes contains albumen, from pressure of the ascites on the renal veins, or from contracted kidneys.

An important point in the diagnosis is to determine whether the liver is of small size, and growing smaller. This is often difficult, on account of the ascites; the difficulty may sometimes be overcome by laying the patient on his left side. In the earlier stages the hard edge of the liver may at times be felt, and even though the ascites be great, by suddenly depressing the walls of the belly with the fingers. The percussion-dulness of the liver in the nipple-line may be reduced to two inches or even one inch in height.

Although in the new-formed connective tissue of cirrhosis fresh vessels form to take the place of those obliterated, yet these by no means suffice to carry on the circulation through the liver. Portal obstruction therefore arises, which relieves itself in various ways: most commonly fluid is poured out into the cavity of the peritoneum, causing an ascites, or into the cavity of the intestines, causing a diarrhœa, which should not be lightly checked. In other cases it is relieved by hæmatemesis, or by hæmorrhoidal discharge. That which is most fortunate for the patient is the formation of a varicose communication between some radicles of the portal system and the general veins; as between the hæmorrhoidal and the hypogastric, the veins of the stomach and the œsophageal.

Most important of all, however, is a vein discovered by Sappey. It arises from the left branch of the portal vein, and passes up the falciform ligament close to the ligamentum teres to join the epigastric and internal mammary veins. It is by no means the same as the old obliterated umbilical vein, although so near to it. The vein just mentioned will often be found dilated after death.

As a rule the spleen is enlarged in cirrhosis. The enlargement may be very great, but the organ is commonly about twice or three times the natural size. After death the spleen is found of softer consistence than natural, sometimes pulpy. The cause is obscure; the reason commonly given is the hindrance to the flow of blood through the liver acting on the splenic vein. The spleen, however, does not always swell when there is obstruction to the portal circulation—for example, in nutmeg liver.

Ascites is a symptom which sooner or later is sure to come on, but extreme cirrhosis may be present without ascites. It appears to arise from the venous stasis in the subperitoneal tissues. Fluctuation, and the movement of the fluid on change of posture, are very clear. The fluid, like all other dropsical effusions, contains albumen, salts, sometimes urea, sometimes sugar; and in jaundice, bile-pigment. After the ascites has set in, the feet may begin to swell, from the pressure of the fluid on the vena cava. The upper limbs and face are free from œdema. In the hypertrophic form ascites is generally wanting, and the patient is deeply jaundiced. In some cases albumen is present in the urine, from coincident Bright's disease.

The patients often complain greatly of flatulence, which adds much to their distress, and dyspnœa. Hæmatemesis and piles are of frequent occurrence, and often epistaxis occurs. Diarrhœa when it comes is salutary, as above mentioned, and should not be checked unless extreme. The urine is scanty and high-coloured; often turbid from urates; and bile-pigment is present when jaundice sets in. Jaundice may or may not be seen, according as the pressure of the new connective tissue does or does not involve the bile-ducts. In the last stages there is great emaciation; stomatitis is often present; and in many cases a scorbutic condition of the gums and petechial hæmorrhages into the skin, especially of the lower extremities, occur. Sometimes the end is ushered in by cerebral symptoms; sometimes it is determined by hæmatemesis.

**DIAGNOSIS.**—The diagnosis depends upon the history of intemperance; the size and consistence of the liver; the size of the spleen; and the appearance of ascites and other dropsies. Of importance also is the peculiar sallow earthy complexion; and the occurrence of hæmorrhages from the stomach

or intestines. The diagnosis is often easy; while at other times it is very hard or well-nigh impossible to make. Cirrhosis may be confounded with portal thrombosis; obliteration of the hepatic duct; nutmeg liver; syphilitic disease, cancer, or hydatids of the liver; and with chronic peritonitis. The most frequent error made in diagnosis is that of mistaking syphilitic disease of the liver for cirrhosis. The former gives rise to portal obstruction and its effects, precisely as in cirrhosis. The only way to distinguish it from cirrhosis is by attention to the history of the case, and by careful examination for the signs of syphilis elsewhere, past or present. In hypertrophic cirrhosis, the coincident enlargement of the liver and jaundice often lead to the diagnosis of cancer of the liver. The absence of ascites is here important. When cancer presses on the common duct it nearly always compresses the portal vein, and ascites results.

**PROGNOSIS.**—It is rare for a patient suffering from cirrhosis of the liver to live longer than one or two years after the symptoms have become so pronounced as to allow a diagnosis to be made, and many die within twelve months. Death is in nearly all cases the end of the disease.

**TREATMENT.**—In the early stages of cirrhosis it is most important to induce the patient to give up his habits of intemperance, for, without this, treatment will be of little avail. Next, the use of alkaline purgatives, with or without vegetable bitters, such as chiretta or calumba, will be very useful. A course of the waters of Carlsbad is often most useful, or of other alkaline or iodised waters. The diet must be mild, and a purely milk diet is sometimes of great service. Exercise on horseback or on foot should be recommended.

In the later stages of the disease the great object will be to keep up the strength of the patient. The ascites, which often becomes the patient's great trouble, sometimes entirely subsides when the patient is kept rigidly in bed without any medicine. When this fails, diuretics, especially copaiba, acid tartrate of potassium, digitalis and squills, and mercurial alteratives, may be employed. Paracentesis should be put off as long as possible, especially if the pulse is of good force, and the breathing not interfered with, as the end of the disease often arrives soon after the tapping, though in some cases the ascites is cured by this operation. Since the introduction of Southey's tubes, paracentesis has been deprived of many of its dangers, and certain physicians recommend early and repeated tapping. The flatulence should be combated by regulation of diet, charcoal, small doses of hydrochloric acid, and carminatives. The bowels must be kept open, but not severely acted on. See ASCITES.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

**LIVER, Congestion of.**—See LIVER, Hyperæmia of.

**LIVER, Contraction of.**—A small liver is met with in cirrhosis, in syphilitic disease, in nutmeg-liver, and in long-continued obstruction to the gall-ducts, in all of which an overgrowth of the connective tissue of the capsule of Glisson is seen. Any kind of pressure on the liver from neighbouring organs will likewise beget wasting. A small liver is seen in old age, and in the marasmus of wasting diseases. The liver likewise wastes if the portal vein be obstructed, or the capillaries in the liver be obstructed, as in pigmented liver. A shrunken liver cannot be looked upon as a disease by itself.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

**LIVER, Enlargements of.**—**ANATOMICAL RELATIONS.**—In proceeding to determine whether the liver is enlarged or not, the following points must be remembered. Normally, the dull sound yielded by percussion extends upwards in front, in a line drawn towards the nipple, to about the sixth rib; laterally, in the axillary region, to the eighth rib; and by the side of the spine, to the eleventh rib. The lower border of the liver corresponds in front and at the side to the lower border of the ribs; and the dullness behind merges into that caused by the right kidney. The left lobe of the liver extends across the epigastrium to the left of the mesial line; the dull sound caused by its upper border merging in that produced by the heart. The upper part of the convexity of the liver rises to a little more than an inch above the sixth rib, the lung dipping down in front, and giving rise to a modified percussion-sound; but for practical clinical investigation it is better to take the line of absolute dullness. The extent of the dull sound from above downwards in the right mammary line is nearly four inches, and at the side about four inches and a-half. In the middle line in front it extends from the base of the ensiform cartilage to about two fingers' breadth below its point. It should be remembered that the limits of the liver present, compatibly with health, considerable variation; that the organ is relatively larger in early than in adult life; that it is depressed in inspiration, and ascends in expiration; that it is somewhat lower down in the erect than in the recumbent position; and that there is temporary distension during digestion.

**DIAGNOSIS.**—There are various sources of fallacy which may lead to an erroneous conclusion as to the size of the liver. Thus, an intestine distended with flatus may get in front of the anterior border of the organ, and lead to the supposition that there is contraction, when the contrary is the case. When there is ascites to any extent, it is difficult to

make out the boundaries of the liver. In this case, however, by placing the patient on the left side, so as to let the fluid gravitate in this direction, a diagnosis may often be effected; also, by suddenly pressing the finger down below the ribs, and thus displacing the fluid, one may sometimes detect the enlarged organ. A rigid right rectus muscle is liable to be taken for a tumour; to obviate this source of fallacy the patient should lie on his back with his thighs drawn up, and his attention should be diverted by conversation whilst the examination is being made. Sources of fallacy may exist in the liver itself, as in malformations or malpositions of the organ; or they may be outside it, either in the abdomen or chest. Malignant disease of the stomach, omentum, or pancreas; a kidney greatly enlarged by cancerous deposit; or fecal accumulations in the colon, may be mistaken for hepatic enlargement. The following considerations will assist in arriving at a correct diagnosis—(a) enlargements of the liver, however much they may extend beyond, generally occupy the normal site of the organ, and however irregular the surface, the usual outline may be traced; (b) such enlargements usually follow the movements of the diaphragm in full respiration. Effusion into the right pleura may be mistaken for enlarged liver, especially as this organ may be depressed by it, and so appear to extend beyond its limits in the downward as well as in the upward direction. In pleuritic effusion, however, the dulness on percussion will vary with the position of the patient, and the upper line of dulness will in effusion be straight, in hepatic enlargement convex. Pleuritic effusion and hepatic enlargement may, however, co-exist. Pneumothorax, emphysema of the right lung, thoracic tumours, and even extreme pericardial effusion, may depress the liver, and affect the diagnosis.

ENUMERATION.—Dr. Bright arranged enlargements of the liver under two heads, according to the surface of the organ, namely, smooth and irregular. Dr. Murchison considered this classification open to the objection that an enlargement, usually smooth, is at times irregular, and *vice versa*, and he proposed the division into painless and painful enlargements; but similar objection may be taken to this.

The principal enlargements of the liver are associated with the following diseases of the organ: Hyperæmia or congestion; obstruction of the bile-ducts; abscess; hydatid disease; fatty infiltration; hypertrophic cirrhosis; albuminoid disease; malignant growths; syphilitic disease; and leucocythæmia. These morbid conditions of the liver will be found described under their respective headings.

STEPHEN H. WARD. JAMES F. GOODHART.

**LIVER, Fatty Disease of.**—DEFINITION.—A disease attended with painless enlargement, and diminished consistence of the liver; due to the presence of a large quantity of fat or oil in the secreting structure; and occurring in connexion with phthisis and other wasting diseases, or in persons of luxurious and indolent habits, in whom there is usually an abundant development of fat in the tissues and other organs.

ÆTIOLOGY.—Fatty liver may be due either to degeneration of cell-structure through faulty nutrition—*fatty degeneration*; or to infiltration of the cells with fat, transmitted through the portal vessels from without—*fatty infiltration*. Fatty degeneration is met with in association with other hepatic diseases, as albuminoid disease and cancer. The fatty liver which results from poisoning by phosphorus would, according to the experiments of Voit and Bauer, appear to be due to degeneration, as the dogs upon which they experimented had been kept without food previously, and were starved during the time phosphorus was administered; showing that the fat could not have come from other parts of the body, or been introduced in food, but must have resulted from the metamorphosis of tissue-material. It is with fatty infiltration that we are more particularly concerned. The fat may come either from within or from without the body. The former case is illustrated when the greater part of the fat of the tissues and organs is absorbed, as in the emaciation of advanced phthisis. Louis, who first established the association of fatty liver with phthisis, found it to exist in about one-third of the cases of this disease, and met with it much more frequently in phthisical females than in males. His observations have been amply confirmed by subsequent observers. Fatty infiltration of the liver also occurs in connexion with other wasting diseases, and is not infrequent in patients who have been long bedridden. From a therapeutical point of view, the medical practitioner is more interested in the disease under consideration, when fat is introduced from without the body. The affection of the liver is then associated with development of fat in other organs and in the tissues generally. Persons thus affected are usually given to undue indulgence in eating and drinking: to eating not only too much food, but food rich in oil and fat, and drinking beer, but especially spirits, to excess. Want of exercise of mind and body, a heated atmosphere, and general luxurious habits, materially assist in determining the affection. In illustration of this cause may be adduced the oft-cited experiments of Magendie, who induced very fatty livers in dogs by feeding them exclusively on butter; and also the production of the *foie gras* in geese, by penning them up in a heated atmosphere and cramming them.

**ANATOMICAL CHARACTERS.**—In fatty disease the liver is more or less enlarged, but seldom to any great extent; the surface is smooth; the borders are rounded; the substance pits on pressure; and the organ is either of pale yellow or drab colour, or, when partially affected, has a mottled appearance. A portion placed in water floats, showing a diminished specific gravity. On cutting into the organ, the knife is greased; and a greasy stain is imparted to blotting-paper applied to the cut surface. A portion, when held in the flame of a lamp or candle, will burn when the water is driven off. It is, however, as Frerichs remarks, only by the microscope that the degree to which the liver is implicated can be determined. In slighter grades, fat-granules and globules are seen to be limited to the outer zone of the lobules in the vicinity of the portal vessels; but in advanced cases the whole of the cells will be found to be filled either with separate globules, or with a single large drop of fat. In less extensive infiltration the liver may be marked by red spots, corresponding to the hepatic veins. Fat in limited quantity is always present in the human liver, so that the term 'fatty' can only be applied when it is in excess.

**SYMPTOMS.**—In the lesser grades of fatty liver there are scarcely any distinctive symptoms, either objective or subjective. When the affection is more pronounced, percussion will indicate more or less enlargement, usually in the downward direction; and palpation may detect a rounded border and diminished consistence, and will, at any rate, determine that the organ is not unduly hard, has no irregularity of surface, and does not differ materially in shape from the healthy liver. There is seldom, if ever, any pain—at most, in marked cases, a sense of tension and of uneasiness on lying on the left side. Jaundice is a rare event; and ascites and enlargement of the spleen cannot be classed as symptoms of the disease. In cases of fatty infiltration, dependent on luxurious habits, as regards diet, &c., there is usually more or less development of fat in other organs, as well as in the omentum and subcutaneous cellular tissue. There is also a greasy condition of skin, with peculiar odour, resulting from abnormal oily secretion from the sebaceous follicles. Dr. Addison considered a peculiar condition of the skin—presenting to the eye a bloodless, almost semi-transparent, and waxy appearance, and to the touch a feeling of smoothness, looseness, and flabbiness—as indicative, if not pathognomonic, of fatty degeneration of the liver. In cases where the liver is much enlarged, and there is much abdominal fat, the upward pressure may interfere with the action of the diaphragm, and cause, especially after meals, embarrassment of breathing. The functional symptoms likely to be present in advanced cases are

irregularity, generally sluggishness, of the bowels; more or less dyspepsia; and, perhaps, loss of appetite. In some cases a weak or irregular, or intermitting action of the heart—with tendency to faintness or giddiness, points to implication of this organ, and is indicative of possible fatal consequences.

**DIAGNOSIS.**—The enlargement of the liver, with preservation of its normal shape, without hardness or irregularity; the absence of pain, jaundice, ascites, or enlargement of the spleen; and its association either with the emaciation of phthisis or other wasting diseases, or with the habits of the gourmand and general development of fat in the body, will usually enable us to distinguish fatty from other hepatic diseases.

**PROGNOSIS.**—The prognosis of fatty disease of the liver is affected by the associated general condition of the patient, and will, of course, be unfavourable in phthisis.

**TREATMENT.**—The general therapeutical indications in fatty liver resulting from luxurious habits of living, point to reform in the direction of diet, air, and exercise. Rich, oily, and fatty articles of food are to be avoided; whilst sugar and starch should be taken in moderation. Beer, in all forms, is objectionable, and so also is alcohol, unless well-diluted, and taken only at meals. Champagne is objectionable, but other light French wines are admissible. Exercise, either on foot or horseback, should be had recourse to daily, but must be regulated according to the soundness of the heart and circulation. Free exposure to pure air, and avoidance of heated rooms, are desirable. The functions of the skin must be promoted by adequate clothing, and by the use of the bath, or by sponging with soap and warm water. The bowels must be attended to, and dyspepsia met by antacids and vegetable bitters. The Carlsbad waters—the warm Sprudel especially—are indicated, being supposed to act upon the redundant fat.

STEPHEN H. WARD. JAMES F. GOODHART.

### LIVER, Functional Disorders of.—

**INTRODUCTORY REMARKS.**—The importance of studying the functional disorders of the liver will be more fully appreciated if the intimate relations which this organ has to various functions of the body are understood. At one time, not very long since, it was supposed that the sole function of the liver was to secrete bile, chiefly for the purpose of eliminating certain objectionable materials from the system. It is now well recognised that other important and special functions are performed by this organ—an organ which is found to exist universally in one form or other throughout the animal kingdom. These functions were thus classified by the late Dr. Murchison, in his lectures on *Functional Derangements of the Liver*: (1) The formation of glycogen, which contributes to the

maintenance of animal heat, and to the nutrition of the blood and tissues; (2) the destructive metamorphosis of albuminoid matter, and the formation of urea and other nitrogenous products, which are subsequently eliminated by the kidneys: these changes also maintaining the animal heat; (3) the secretion of bile, a large portion of which is reabsorbed, assisting in the assimilation of fat and other elements; whilst a part, passing downwards, stimulates the peristaltic action of the intestines, and arrests decomposition.

When it is remembered how the functions of the liver are readily influenced by various external and internal agencies, and how they are consequently deranged, it will be of the utmost importance to the pathologist and to the clinical physician to study the character and effects of these derangements. They were classified by Dr. Murchison under nine heads: (1) Abnormal nutrition; (2) abnormal elimination; (3) abnormal disintegration; (4) derangements of the organs of digestion; (5) derangements of the nervous system; (6) derangements of the organs of circulation; (7) derangements of the organs of respiration; (8) derangements of the urinary organs; (9) abnormal conditions of the skin. The above heads, however, represent for the most part functional and general symptoms resulting from hepatic disorders, rather than the disorders themselves. The arrangement under three heads, representing the three principal functions of the organ above mentioned, will be a sufficiently convenient form for the consideration of these disorders.

**ETIOLOGY.**—It is scarcely necessary to remark that functional disorders of the liver are often secondary to or associated with disorder of its nervous and vascular systems, or with diseases of the thoracic and abdominal viscera, with febrile affections, malaria, and other causes. It is only with the causes which induce these disorders when primary that we are here concerned. They are—

1. *Errors in diet.*—The most common of these are habitual over-eating, and especially indulgence in fat and fatty articles of food, rich soups, entrées, and pastry; the undue use of sugar, or of fish or flesh containing much oily matter. Excess in the use of alcoholic drinks is another cause in frequent operation in this country. The combination of sugar with alcohol much enhances the mischief. Hot, sweet grog; sweet new wines; champagne, unless dry; Madeira; sherry; port-wine; Burgundy; liqueurs; the persistent use of malt liquors, particularly of mild ales and stout, are ready causes of hepatic derangement.

2. *Habits of life.*—Want of exercise, whether the result of necessity or of indolent habits, is a common exciting cause of hepatic functional disorders; and its effect is exaggerated when it is associated with errors in

diet. Living habitually in a high temperature, whether in a warm climate or in overheated rooms, is another source of hepatic disorders, and this is intensified when in connexion with the causes already alluded to. Depressing worries, nervous and emotional influences, must not be forgotten in considering the ætiology of the subject. So likewise chills and exposure to draughts of cold air or to damp and wet.

**SYMPTOMS AND DIAGNOSIS.**—The phenomena which are observed in functional derangements of the liver, and which give evidence of the presence of such disorders or derangements, may be in a great measure indicated according to the particular hepatic function which is affected, namely, (1) the *glycogenic*; (2) the *metabolic*, and (3) the *biliary*.

1. **Disorders of the Glycogenic Function.**—Disorders of this function of the liver, when persistent, come under the subject of diabetes, and require only to be mentioned here. See DIABETES MELLITUS.

Sugar may also be found in the urine occasionally and in small quantities, in some cases in which its presence is clearly connected with disordered liver-function. It may be present with urea in excess, or with albumen, either at the same time or alternately.

2. **Disorders of the Metabolic Function.**—It is a matter of great practical importance to observe that the derangements of the metabolic function of the liver are constantly made evident and can be recognised by changes in the composition of the urine. These changes, therefore, should be looked for, and the urine of patients examined in almost every case. It is not possible here to describe all the changes in the composition of the urine which indicate functional disturbance of the liver. It will be sufficient to indicate the more important of these—(a) the lithates and lithic acid, (b) urea, (c) albumen, and (d) colouring matter.

(a) *Lithates and lithic acid.*—The writers quite concur with, and may concisely state, the views advanced on this subject by the late Dr. Murchison. One of the immediate results of such faulty function connected with albuminous disintegration is the non-conversion of nitrogenous matters into urea, and the production of lithates and lithic acid, inducing a condition of blood to which he fitly applied the term *lithæmia*. This lithæmia may be relieved for a time by elimination by the kidneys, showing itself in deposits in the urine, on cooling, of lithic acid, lithates, and pigmentary matter. These deposits are not infrequent in persons in apparently good health, especially after any excess or error in diet or exposure to cold; and they are more or less constant in subjects of gouty habit of body, and in those who are predisposed to hepatic derangement,

or who induce it by habitual indulgence in over-stimulating diet. These deposits may continue for years without causing much discomfort; but after a time the excessive quantity of lithic acid and lithates cannot be eliminated by the kidneys; and they accumulate, causing disease of the kidneys as well as disturbances in different parts of the system, and giving rise to various more or less distressing symptoms. Of these symptoms the more prominent are—epigastric tenderness on pressure and sense of oppression, flatulent distension of stomach and bowels, heartburn, acid eructations, a sense of weariness and tendency to sleep after meals, furred tongue, unpleasant taste in the mouth, especially in the morning, appetite often good, sometimes the contrary, an excessive secretion of viscid mucus in the fauces and back of the nose, constipation and vitiated secretions. Palpitation, irregular or intermitting pulse, frontal headache, vertigo, deafness and noises in the ears, restlessness at night, irritability of temper, and hypochondriasis, are other symptoms that are more or less constantly present.

Gout, whether openly manifested, latent, or irregular, is associated with the symptoms just mentioned. It is one of the results of lithæmia, and thus has its origin very frequently in faulty hepatic function.

Urinary calculi are also a result of lithæmia, at all events those which consist of lithic acid or its salts, which, as we know, are the most frequent forms. Thus also it is that the formation of these calculi should be prevented by remedies and a *régime* directed to the liver rather than to the kidneys.

Biliary calculi are likewise a result of functional hepatic derangement, and are frequently associated with a gouty habit of body, and with lithic acid deposits and calculi.

Lithæmia predisposes to local inflammations. Individuals who are subject to deposits of lithic acid and lithates are more liable than others to local inflammations in a severe form. In reference to this fact, Dr. Murchison made the practical observation that in such persons the lithates cease to be eliminated on the advent of a local inflammation or ordinary febrile catarrh, to be again discharged freely on the subsidence of the pyrexia. In such cases, he added, the retention of lithates in the system has probably determined the attack.

Certain diseases of the skin, such as erythema, eczema, herpes, psoriasis, lichen, and urticaria, are unquestionably often induced and maintained by lithæmia resulting from hepatic derangement.

Other effects of this functional derangement, as Dr. Murchison remarked, 'by the production of peccant substances which are not readily eliminated, and which, therefore, accumulate in the system, may in the long run lead to many of the most troublesome, if

not serious, maladies, both acute and chronic, to which our race is subject.'

(b) *Urea*.—This substance may be found deficient in the urine; but if so, it will be in connexion with some structural change in, as contrasted with functional disorders of, the liver, which have just been treated, and in which is found an excess of this substance. When found in such excess, we are not able always to determine whether it is due to its formation in the liver in excess. Still this excessive elimination, whatever its source, is associated with some special and important symptoms to be presently described. When the writers speak here of excess of urea, they mean such excess as is shown by the rough-and-ready method of mixing, say, a drachm of urine with a like quantity of pure nitric acid, and placing the tube containing the mixture in cold water. After some minutes or hours a few crystals may be deposited, or a mass almost solid may be formed. When there is a free discharge of urea, persons generally complain of feeling languid, often extremely so, and of inability to accomplish any work that requires mental or physical exertion. A case somewhat unique will illustrate certain points in this affection. The writer (R. Q.) was requested to see, with Mr. (now Sir William) Savory, a gentleman who complained of exceeding languor, which seemed unaccountable. He looked well nourished, and was surrounded by every comfort. His abdomen was full, and his liver somewhat enlarged. He was abstemious, living almost exclusively on a vegetable diet, owing to his great dislike to all animal food. His evacuations were pale; and inquiry showed that he was passing some sixty ounces of urine or more daily, the specific gravity of which was over 1030. On testing with nitric acid, a mass of urea occupying more than two-thirds of the mixed fluid was deposited. Blue pill and colocynth given twice or three times a week was well borne; and he also took taraxacum, with an alkali or an acid. The excess of urea in time disappeared and the patient did well. The writer has treated many like cases, but none in which the discharge of urine in connexion with urea was so copious; indeed, in many cases the quantity of urine is less than normal.<sup>1</sup> The sense of languor and depression in these cases is suggestive of a tonic or stimulant line of treatment. This does more harm than good. The eliminating treatment acting on the liver generally succeeds, and it may be followed by the use of some vegetable bitter tonic.

(c) *Albumen*.—Albumen is often found in the urine as the result of disordered liver-function. Its presence may be merely tem-

<sup>1</sup> Dr. A. Martin (Paris, 1877) writes thus on this subject: 'It is possible to utilise in a certain degree the variations of this principle in the urine for the diagnosis of maladies of the liver.'

porary, in connexion with a temporary hepatic derangement, or it may be more or less permanent. The writer watched with interest the case of a gentleman in whose urine albumen was present more or less constantly for nearly thirty years. He did much and very responsible brain-work, and lived generously. The urine had low specific gravity. Though often looked for, casts were rarely found. This gentleman himself recognised the share which the liver had in the production of the albumen, and he regularly took as he felt occasion a grain of calomel once or twice a week as a remedy.

(d) *Bilirubin*.—The colouring matter of bile may be formed in excess, and found in the urine on adding nitric acid. Its presence indicates some disordered liver-function, and is so far useful in helping diagnosis.

**3. Disorders of the Biliary Function.**—Having thus described some special disorders of function as indicated by the presence of special materials found in the urine, we may now refer to disorders of the biliary function generally. These may be divided into (a) those due to *excessive* secretion; and (b) those due to *deficient* secretion. It is scarcely necessary to arrange those due to *vitiated* bile under a separate head, as the secretion may be vitiated whether it be redundant or deficient.

(a) *Excessive* secretion of bile is characterised by bilious diarrhoea—copious, fluid, bilious evacuations; nausea, or not infrequently vomiting; twisting, griping pains in the abdomen; and perhaps some febrile symptoms. The bile in some cases seems to be peculiarly acrid, and to cause much smarting when voided. The urine is generally high-coloured, and loaded with lithates. There is frequently headache and sleeplessness, with either irritability of temper or depression of spirits. Disturbances of circulation, in the form of irregularity of pulse, and palpitation of the heart, are also occasionally present. This excessive secretion of bile, with the attendant symptoms, is usually the result of congestion of the liver.

(b) *Deficient* secretion of bile is characterised by dyspeptic symptoms, such as furred tongue, unpleasant taste in the mouth, loss of appetite, and flatulence. The action of the bowels may be irregular, but is usually costive; and the evacuations are of a pale yellow, or drab, or whitish colour, and often of offensive odour. The complexion is usually sallow and anæmic, not often jaundiced. Disturbances of the circulation, in the form of languid or irregular pulse, occur, and in the nervous system, headache, languor, drowsiness, and hypochondriasis being frequent concomitants. Piles are troublesome, but afford relief when they bleed. The urine is generally dark-coloured, turbid, and loaded with lithates. There is also, in chronic cases, loss of flesh. Indeed, the action of

the bile in promoting the assimilation of fat, in stimulating the peristaltic action of the intestines, and as an antiseptic, is well illustrated in the symptoms which attend protracted deficiency of the secretion.

**PROGNOSIS AND TREATMENT.**—In no class of disorders do general remedial or hygienic agents act more beneficially than in functional derangements of the liver. Pure air, exercise, strict attention to the functions of the skin, and suitable diet, are the curative means on which we must mainly rely. In all cases it is of moment to promote the healthy action of the skin and lungs. This is to be done by exercise on foot, or far preferably on horseback. The latter is peculiarly advantageous, as it stimulates directly, by a series of succussions, and by contraction of the abdominal muscles, the liver and intestines, and may be had recourse to, in moderation, by those who are not very vigorous. Walking promotes the general circulation, excites the action of the skin, increases the frequency and fulness of the respirations, and indirectly tends materially to relieve a congested or indolent state of liver. The action of the skin must also be maintained by adequate clothing in all seasons of the year, and by daily use of the bath, followed by active friction of the entire surface of the body.

The diet should be of a light nourishing character; and rich gravies, soups, made dishes, sauces, pastry, raw vegetables, and such articles should be avoided. The nitrogenous foods suit best, especially where there is associated lithæmia; starch and saccharine articles being more or less objectionable, especially when sugar appears in the urine. The strong spirituous drinks are to be avoided; but the lighter wines, when pure, as claret, hock, and light dry sherry, may be taken at meals in moderation, or small quantities of whisky or brandy in water.

Among the medicinal remedies which promote the action of the liver and the expulsion, if not the secretion of bile, mercury and its preparations hold a prominent place. For notwithstanding the results of experiments upon animals, few practitioners will be content to give up the advantages which their clinical experience has taught them to expect from the judicious use of mercury. It has been said that the drug merely effects the expulsion of the bile; the result, however, is manifest and unquestionable. In excessive secretion of bile a single full dose of calomel, followed in a few hours by a saline draught, will cause a free downward discharge; and in the lithæmic condition of system, indicated by turbid urine containing copious lithates, the same treatment will afford ready relief. In smaller doses, repeated at intervals, and combined with other aperients, such as colocynth or rhubarb, mercury will be followed by beneficial results in

less active functional derangement. In some cases mercury may be inadmissible, or may disagree; and, in any case, it is well not to repeat it too frequently, or to continue it for too long a time, as its protracted use is apt to impair digestion and nutrition, and weaken the function of the organ which it at first relieves. A valuable addition to the list of cholagogues will be found in podophyllin. A quarter of a grain to a grain of the resin may be given for a dose, combined with a little hyoscyamus or half a grain of extract of *cannabis indica* to prevent griping, and a grain or two of rhubarb or watery extract of aloe, or, if it is desirable to quicken the action, with some extract of colocynth, or it may be given in solution as the tincture. When the drug acts favourably it produces one or two bulky evacuations, with copious excretion of bile, followed by a feeling on the part of the patient that the bowels have been thoroughly emptied. Its action, however, is at times neither satisfactory nor certain, and it causes occasionally much griping, irritation, and tenesmus, and subsequently depression. *Taraxacum* is useful as a mild aperient and diuretic, and probably alterative, in functional hepatic disorders, and may be given in combination with alkalis, especially the bicarbonate of potassium or of sodium, in cases where the gouty or lithic acid diathesis exists. The dose of solid extract is from ten to fifteen grains, but the fluid extract is the best preparation, especially in combination with chloride of ammonium. Nitro-muriatic acid is indicated in cases of torpid liver associated with oxaluria (*see* LIVER, Hyperæmia of). The saline aperient draught has of late been in a great degree superseded by the use of one or other of the aperient waters, the Friedrichshall, Pullna, Hunyadi, or the Rubinat, &c. These should be taken in the morning, fasting, and their effect is quickened by the addition of some warm water. The action of these waters, as of the saline purgative, is to cause a drain from the intestinal vessels, thereby relieving the congested hepatic portal system. The waters of Carlsbad, Marienbad, and other similar springs contain no sulphate of magnesium, and owe their aperient effect to the sulphate of sodium which is the preponderating ingredient. This valuable drug does not act by stimulating intestinal secretion, but, according to Buchheim, by retaining the water in which it is dissolved and that which it meets with in the bowel, and so constituting a solvent which loosens and softens and carries down solid fecal masses and tenacious mucus. The amount of carbonate of sodium associated with the Glauber's salt in these waters renders them antacid, and assists in determining a diuretic as well as aperient action. The mineral waters, or the salts obtained from them by evaporation, may be taken at home in the morning, in conjunction with sufficient warm water. For

those individuals especially who are suffering from hepatic congestion or merely functional disorder, if the result of irregular habits in eating and drinking, a visit to and course of waters at one of the spas will, if means permit, be desirable. Change of scene, regularity in diet, and absence from mental harass, are, of course, important elements in the success of a more or less protracted stay at such places; and it is remarkable how readily many individuals, who are quite unmanageable at home, submit to strict hygienic arrangements under fresh influences. Harrogate, Cheltenham, Leamington, and Scarborough in this country, Strathpeffer in Scotland, and Lisdoonvarna in Ireland, are the chief places in Great Britain. In addition to those already named, Homburg and Kissingen on the Continent are amongst the spas which enjoy special renown in the treatment of liver-affections. *See* MINERAL WATERS.

S. H. WARD. RICHARD QUAIN.

**LIVER, Gangrene of.**—*See* LIVER, Abscess of; and Inflammation, Acute, of.

**LIVER, Hydatid Disease of.**—*SYNON.*: Echinococci of Liver.

**ANATOMICAL CHARACTERS.**—The liver is the organ most frequently affected with hydatid disease. There is usually but one cyst, but there may be two, three, or more; and the size of the cyst may vary from that of a pea to that of a child's head. The cysts may exist in either lobe of the liver, but they are more frequent in the right; and they may be attached to the upper or under surface, or project from the border, or lie buried in the substance of the gland. This is more or less modified in form, and increased in size, according to the magnitude and site of the cyst. When the cyst is small and deep-seated, there will be no appreciable change in the liver, and the disease may be latent for years. When, however, the cyst is very large, it, with the liver, constitutes a tumour, which may encroach upon the thorax, and also fill a great part of the abdomen. Pressure of the cyst may induce atrophy of a portion of the liver, but, at times, hypertrophy is the result. The bile-ducts have occasionally been found to be obliterated, or a communication to have been effected between them and the cysts. When at the surface of the organ, the cysts as they enlarge may induce inflammation and thickening of the peritoneum, and adhesion to neighbouring structures.

**SYMPTOMS.**—A hydatid cyst, when sufficiently large and near the surface, generally exhibits itself as a tumour of variable size, situated either in the right hypocondrium or in the epigastric region; evenly globular in its early stages; firm, resisting, yet elastic, and, at times, with a sensation of fluctuation. Briançon and Piorry noticed a vibration or

trembling—*hydatid fremitus*—which is felt when the surface is compressed gently by three fingers of the left hand, and sharp percussion made with the right hand over the middle finger. Frerichs does not consider this sign of much importance, it having been present in only one-half of his cases. If the tumour is situated behind the liver, it will, as it develops, push this organ forwards, flatten it, and increase the area of dulness. The tumour may last for a considerable time, and go on increasing to some extent, and yet the patient remain free from constitutional disturbance, perform all his functions well, and keep in good condition as regards flesh and strength. When, however, it has attained a very large size, it will give rise to various symptoms—to a feeling of tightness and distension; if it press upwards, to embarrassed breathing, cough, possibly more or less dulness, with diminished respiration, at the base of the right lung, and palpitation; if upon the abdominal viscera, to interference with their functions. Pain is not generally present, but in some cases there is a gnawing pain, either at the epigastrium or extending forwards from the lumbar region. Edema of the lower extremities may occur when the tumour presses upon the inferior cava, or ascites from a similar effect on the portal vein. The latter is, however, not unlikely to have some associated features, tending to indicate the unusual nature of the effusion and the origin of the disease.

**DIAGNOSIS.**—Hydatid tumour of the liver is not always easily diagnosed; but the characteristic features already noticed, and its compatibility (in many cases up to an advanced stage) with a good state of health, will generally point to its nature. Abscess of the liver will be distinguished by local and remote pain; the frequent antecedence or co-existence of dysentery; and severe constitutional symptoms, such as hectic fever, rigors, &c. It must, however, be remembered that hydatid cysts are liable to become inflamed and to suppurate, when the diagnosis will not be so readily made; but even so, the suppuration of a hydatid cyst is less likely to be accompanied by the acute paroxysmal fever so characteristic, if present, of abscess of the liver. Cancer of the liver will generally be marked by irregularity of surface; the presence of pain; the cachectic aspect; and the rapidity of progress. Aneurysm of the abdominal aorta may form an epigastric tumour, of even, spherical shape; but the pulsations, frequently very forcible, coupled, probably, with bruit, audible along the course of the vessel before and behind, will determine the diagnosis. The site, the pyriform shape and uniform size, and the usual accompaniment of jaundice, will distinguish from hydatid disease the tumour caused by a distended gall-bladder. Frerichs thinks that hydatid

disease of the liver is more frequently founded with localised pleuritic effusion at the base of the chest than with any other affection. He remarks that the same signs—dulness on percussion, absence of vocal thrill, intercostal fluctuation—would be present in both cases. He rests the diagnosis on the fact that the line of dulness would present a curve which would look upwards in the one case, downwards in the other. To this sign, of uncertain presence, may be added that the vocal vibration and resonance, although perhaps somewhat lessened, are not usually damped by pressure from below the diaphragm to the extent that is effected by fluid in the pleura.

**TERMINATIONS AND PROGNOSIS.**—Hydatid tumour of the liver may last for years, and be compatible with an average state of health; or, at an early or advanced period of its existence, it may terminate in one of the following ways: 1. It may, from its bulk and position, press upon and interfere with the functions of different organs. Pressure on the large venous trunks may induce ascites and dropsy of the lower extremities; pressure upon the stomach and intestinal canal may obstruct functions connected with the assimilation of food, and induce failure of flesh and strength, and ultimately death from exhaustion. 2. The tumour may contract adhesions with the diaphragm; ulcerative action through this may be set up, and either (*a*) discharge of the contents of the sac may take place into the pleura, and fatal pleuritis result; or (*b*) further adhesions and ulceration may effect communication with the lung, pneumonic symptoms ensue, and the contents of the sac, mixed with the products of inflammation, be expectorated. The contents are sometimes expectorated, and the disease cured by this means. 3. A rare result is adhesion to, and ulceration into the pericardium, with escape of contents, and rapidly fatal results. 4. Adhesion may be effected with some part of the alimentary canal, and the contents of the sac be discharged by vomiting or by stool. 5. Rupture of the sac may be caused by a blow or otherwise; the contents be discharged into the peritoneum; and fatal peritonitis result. 6. The tumour may contract adhesions with the parietes; point externally; and be opened or effect an opening by natural process, inflammation and suppuration having been previously set up in the sac. 7. Budd and Frerichs notice a possible cure from the obliteration of the sac by the formation within it of a putty-like matter, the combined result of degeneration of the cyst and its contents, and of inflammation outside the parent cyst. These conditions are by no means rare in the deadhouse, and they are frequently associated with calcareous changes in the cyst-wall, as well as with the deposition of brown or orange biliary pigment. So generally is pigment of this kind present in various parts of these cured hydatids, that there is good ground for

supposing that the rupture of minute bile-ducts into the cyst or its wall is no uncommon thing; and, whether by killing the parasite, or by effecting slow inflammatory changes in the bed of the cyst, that it leads to a spontaneous cure in no inconsiderable number of cases. And as an illustration, although a rare one, of the extent to which the slow inflammatory changes may proceed under favouring conditions, there is a specimen in the museum of Guy's Hospital where the hydatid is surrounded by, and had no doubt determined, an enormous growth of gummatous material. 8. Communication may be effected between a hydatid cyst and one of the larger bile-ducts, and then the result will usually be fatal, although there are one or two cases recorded of recovery. 9. Similar cysts may be formed in other parts or organs of the body. 10. As a possible rare event may be mentioned communication of the sac with the ascending vena cava, escape of the contents into this, transfer of the contents to the right side of the heart, impaction in the pulmonary artery, and fatal asphyxia.

**TREATMENT.**—So long as a hydatid tumour of the liver induces no distressing symptoms, and does not affect the functions of any organs, there is no pressing cause for interference. When, however, it is rapidly increasing, is accompanied with pain or distressing distension, and especially if by upward pressure it is causing difficulty of breathing, and other symptoms, recourse should be had to tapping. If there be any doubt about the nature of the tumour, the exploratory needle may be first introduced; and should a clear fluid, free from albumen, escape, the diagnosis may be considered to be established. It is now, indeed, considered the best treatment not to wait for urgent symptoms; but when the disease is well developed, the cyst yet perhaps single, and the walls elastic, to let out the contents. This is best and most safely effected by puncturing with a fine trochar or with an aspirator. Certain precautionary measures must, however, be attended to. Prior to tapping, a broad flannel roller should be firmly applied round the abdomen, commencing from below and carrying the bandage up to the tumour, so as to assist in fixing this. It is considered desirable not to quite empty the cyst, as by doing so the chance of air entering the cyst is increased. This result may also be further prevented, and adhesion of the cyst to the parietes promoted, by applying a compress of lint over the wound and fixing it firmly with the remainder of the bandage which has been already partially applied. The patient should be kept quiet in bed for a day or two, and rest should be further ensured by the administration of morphine. The late Dr. Murchison discountenanced the use of chloroform for this operation, as the pain is but

trifling, and the chloroform may induce vomiting, which would interfere with the subsequent rest of parts, so desirable to ensure a successful result. It is not necessary to wait for the adhesion of the cyst to the parietes before puncturing, as the use of a fine trochar diminishes the risk of escape of the contents of the sac into the peritoneum; and, moreover, the escape of a certain amount of fluid will not usually induce peritonitis. A large proportion of cases thus treated have been successful.

When a hydatid cyst has suppured, it should be opened by free incision, and the contents allowed to evacuate themselves. The cyst may be opened immediately or by two stages, according as it is adherent to the parietes or not. Free drainage must be ensured in accordance with the principles of modern antiseptic surgery.

Puncture and subsequent injection of the cyst with some stimulating fluid; gradual opening of the cyst by applications of caustic potash, so as to ensure adhesion with the parietes; and a large incision with a view to effective removal of contents, are methods of treatment which have been practised, but cannot be recommended in comparison with simple puncture with a fine trochar; but, as regards incision and evacuation, the success of modern surgery is such that it may now be resorted to with less misgiving, and is even strongly advocated by some. There would, however, appear to be some risk of hæmorrhage from the bed of the cyst after incision; and inasmuch as there may be several cysts, and simple aspiration is free from any serious danger, and is often successful in inducing quiescence, at any rate, in the cyst for long periods of time, the simpler measure should, unless specially contra-indicated, be first adopted.

The late Dr. Hilton Fagge and Mr. Durham treated several cases successfully by acupuncture, and by passing a galvanic current through the contents of the cyst; but it seems not improbable that the result may have been due to the acupuncture, and not to the galvanic influence. Treatment by special medicinal agents, administered internally, has been fairly tried. Of these, common salt and iodide of potassium in large doses may be mentioned. They have, however, proved entirely useless.

STEPHEN H. WARD. JAMES F. GOODHART.

**LIVER, Hyperæmia of.**—**SYNON.:** Congestion of the Liver.

**DEFINITION.**—Uniform enlargement of the liver, with preservation of its normal shape; caused by over-distension with blood, the result of mechanical obstruction to the return of blood to the heart, or of direct afflux of blood through the portal vessels; attended with a sense of fulness and oppression in the right hypochondriac and epigastric regions,

and a dusky and sometimes jaundiced complexion.

**ÆTIOLOGY.**—Congestion of the liver may be either *active* or *passive*. Niemeyer limits the term 'congestion' to the latter, and applies the term 'fluxion' to the former. *Active hyperæmia* or congestion results from—  
 1. Excess in eating and drinking, especially in persons of sedentary and indolent habits. Determination of blood to the liver occurs, to some extent, in the process of digestion in connexion with ordinary meals, and if these consist of rich and irritating materials, and are repeated too often, or if the liver is stimulated between meals by the imbibition of spirituous liquors, the hyperæmia may become excessive and continuous.  
 2. Long exposure to a tropical or sub-tropical temperature will perhaps induce hepatic congestion, but such influence may be long resisted by persons of temperate habits; and it is probable that the association of irregularities in eating and drinking mainly contributes to the result.  
 3. A chill, after exposure to heat, may induce active hepatic congestion, which, in hot climates, may result in suppurative hepatitis.  
 4. It may be excited by injuries to the liver—such as contusions or wounds.  
 5. Hyperæmia of the liver, with enlargement, occurs in connexion with typhus fever and other acute exanthemata, puerperal fever and scurvy. It is also a result of prolonged exposure to malaria, with or without attacks of pronounced ague. This influence is the main cause of the enlarged livers with which 'old Indians' return to this country.

*Passive hyperæmia*, to which the term *mechanical congestion* is applied by some authors, is due to interference with the return of blood from the liver through the hepatic vein and inferior cava to the heart. Such interference may be due immediately to dilatation of the right heart, with affection of the tricuspid valve; to obstruction to the circulation in the course of the pulmonary artery, caused by different diseases of the lungs; or, farther on, in the line of the circulation, to disease of the mitral or aortic valves. The affections of the lungs which interfere with the pulmonary circulation are either acute, as pneumonia, and then the hepatic hyperæmia may pass off with the disease; or they are chronic, as emphysema and fibroid disease, and then the hyperæmia will persist. Mechanical congestion of the liver may result from direct obstruction to the flow of blood by the pressure on the vena cava of aneurysmal or other tumours. Mere weakness of the heart's action often keeps up a certain amount of passive congestion.

**ANATOMICAL CHARACTERS.**—A hyperæmic liver is increased in size about equally in all directions; its resistance is also increased; its peritoneal investment appears distended and shining. On making an incision blood

oozes out freely, and the cut surface is dark red—either uniformly so, or spotted with intervening lighter spaces. In the passive or mechanical form, the central vessels of the lobules—the hepatic veins—are engorged; and when persistently so, as in disease of the mitral valve, the cut surface gives the characteristic appearance to which the term *nutmeg liver* has been applied. The dark centres contrast with the pale circumference of the lobules, the light and dark parts being clearly defined, but varying according to the section; the surface gives an appearance which resembles closely that of a cut nutmeg. The central dark spots result from distension of the hepatic veins, and deposition of bile-pigment in the adjacent hepatic cells, which are more or less atrophied by pressure; the lighter spaces correspond to the interlobular veins, the light colour being due, according to most pathologists, to the presence of fat in the cells at the circumference. Dr. Wickham Legg, however, states, as the result of an examination of twenty cases of nutmeg liver, that the fat was not in excess in the majority of them. In addition to the atrophy of the cells by pressure of dilated hepatic veins, there may be hypertrophy of the interlobular hepatic tissue, with lymphoid bodies scattered through it. The term 'red atrophy' has been applied to this advanced stage of nutmeg liver, but, as Dr. Legg remarks, it is objectionable, as having been previously used by Virchow to designate a state of liver met with in wasting diseases, such as typhoid fever. The term 'atrophic nutmeg liver' answers well.

**SYMPTOMS AND SEQUELÆ.**—In slight cases of hyperæmia the liver does not extend much beyond its normal limits, but it may perhaps be felt below the borders of the ribs and across the epigastrium. In severe cases, and especially in passive hyperæmia from obstructed circulation, the organ often attains a considerable size, and is found, on percussion, to extend upwards into the mammary region, downwards nearly to the umbilicus, and across into the left hypochondrium. Its resistance is generally increased, and, in old-standing cases, such as the local condition often seen in the deadhouse as the effect of tight-lacing, it may even be hard enough to create the suspicion of malignant disease. There is often, especially in acute cases, tenderness on pressure. The patients do not exactly speak of pain, but of a sense of oppression and fulness in the right hypochondriac and epigastric regions, and of uneasiness from the pressure of clothes, or on lying on the left side. In most acute cases there is marked functional and general disturbance; a furred tongue, nausea, vomiting at times of bile, bilious diarrhœa, sallowness of complexion, or some amount of jaundice. In certain cases the bile seems to be peculiarly acrid, and causes much griping

and distress as it passes downwards, and smarting as it is voided. The urine is high-coloured, and loaded with lithates. The patients often complain of headache, are irritable and depressed in spirits, and feel languid and drowsy. Disturbances of circulation are indicated, in severe attacks, by irregularity of the pulse and palpitation of the heart. When jaundice is present in any marked degree, there is probably catarrh of the bile-ducts, and transient obstruction of these ducts, and then the evacuations are devoid of bile.

The hyperæmic condition of liver induced in India or other tropical regions, as the result of high temperature and malarious influence, may terminate in chronic enlargement, and possibly in damaged structure of the organ. The symptoms which mark this are cachexia and anæmia, with sallowness of complexion; a weak circulation, indicated by great susceptibility to changes of temperature, and by chilliness or coldness of the lower extremities; disturbances of the nervous system, shown by irritability, depression of spirits, disinclination from effort of any kind, headache, and giddiness; and other complaints. Dyspeptic symptoms are present. The bowels are either constipated or relaxed, the stools in either case showing a deficient or vitiated secretion of bile. The urine sometimes contains bile, frequently oxalate of lime, and excess of urea. The skin is dry and harsh.

In the mechanical form of hyperæmia there are, in addition to special gastric and hepatic symptoms, other symptoms indicative of the pulmonary or cardiac affection on which it depends. The complexion is more or less dusky, and there is a certain amount of lividity mixed with the jaundiced hue. The liver may be found to vary in size in accordance with the variations in the conditions which give rise to it. It may sometimes pulsate. Ascites may now become a prominent symptom, whilst, as Niemeyer remarks, the general dropsy resulting from associated heart-affection may be yielding to treatment.

**DIAGNOSIS.**—Hyperæmia of the liver, when it is the result of obstruction to the circulation caused by diseases in the chest, is easily recognised. Also, when of the active kind and acute, it is not likely to be mistaken for any other cause of enlargement. It is only when the congestion is chronic, and the liver hard and resistant, that an erroneous diagnosis may be made; but the previous history of the case, and associated general symptoms, will lead to a right conclusion. It might be mistaken for albuminoid disease, but then the liver may be really undergoing this degeneration.

**PROGNOSIS.**—Active hyperæmia of the liver usually ends favourably on removal of the exciting cause, and under appropriate treatment. The following considerations will,

however, influence the prognosis: 1. An attack of hyperæmia in an individual of intemperate habits may be but the early stage of cirrhosis. 2. A similar attack, occurring in India, may, as Dr. Maclean remarks, be but the commencement of acute hepatitis, which may end in suppuration. 3. The prognosis in the mechanical form of congestion, dependent upon disease in the chest, will be influenced by the nature and stage of such disease.

**TREATMENT.**—An attack of acute hyperæmia of the liver will usually be relieved quickly by rest; by restriction to a bland, fluid diet; and by ensuring free action of the bowels by a dose of calomel, followed after a few hours by a saline aperient, either in the form of a draught, or of one of the more active mineral waters. A single sufficient dose of calomel, four or five grains, will often rapidly relieve attendant gastric irritation and vomiting, and ensure a free downward discharge of bile. Should there be much tenderness on pressure over the liver, the application of sinapisms or turpentine stupes, followed by hot poultices, will be beneficial. So long as any acute symptoms continue, the patient should be kept quiet in bed; the diet should be light and fluid; and stimulants should be rigorously excluded. The portal system must be kept relieved by a dose of Pullna or Friedrichshall water, or by a saline draught, every or every other morning, preceded the previous night by some mercurial preparation, if the secretion seem to require this. Podophyllin may be advantageously substituted for mercury in some cases, but it is uncertain in its action. Its griping effect is counteracted by the addition of a little extract of hyoseyamus or half a grain of extract of cannabis indica, and its action will be quickened by adding a little compound colocynth pill. When the acute stage of hyperæmia is passed, taraxacum often acts well as a purgative and alterative; combined, in subjects of gouty habit, whose urine is more or less charged with lithates, with bicarbonate of potassium, or, in other cases, with diluted nitro-hydrochloric acid. This acid, in torpidity and chronic enlargement of the liver, is one of our most effective remedies. It acts by altering and promoting the biliary secretion, and by improving the tone of the digestive organs. In the chronic enlargement of liver of 'old Indians,' in that which results from malarious poison, and also in other forms of chronic congestion, it often acts very beneficially. It may be used both internally and externally. Ten to twenty drops of the diluted acid of the Pharmacopœia may be given two or three times daily, combined with taraxacum, quinine, or other drug, according to indications. The external use of the acid has been advantageously had recourse to at the Seamen's Hospital for years, either in the form of compress over the

abdomen, or by sponging the surface of the body, or by the use of baths to the lower extremities. The fluid for the bath or compress is prepared by adding eight ounces of the diluted acid of the Pharmacopœia to a gallon of water at about 98° F. The compress may be applied by soaking a flannel roller of sufficient length, about a foot in width, in the prepared acid, and wringing it so that it merely remains damp. The roller should be then applied round the body, covered with a piece of oiled silk, and worn constantly—subject, however, to renewal of acid night and morning; or the lower extremities may be immersed in the fluid for about twenty minutes, night and morning, and the inner sides of the thighs and the body be sponged at the same time. Earthenware or wooden baths should be used; and the sponges and towels after each bath must be well washed in cold water, or they will be destroyed by the acid. At times the external use of the acid causes purging; and in several cases, at the Seamen's Hospital, severe irritation of the skin, with copious papular eruption, resulted, and the remedy had to be discontinued. Chloride of ammonium and iodide of potassium must be mentioned as drugs that have been found serviceable in reducing livers enlarged by chronic congestion.

In the hyperæmia resulting from disease of the mitral or aortic valves, or from chronic pulmonary changes, it is sufficient to remark that the chest-symptoms will often be most effectually relieved by treatment directed especially to the liver.

Much benefit will result in many cases of hyperæmia from a course of mineral waters at one of the German or English spas. Marienbad, the cool springs of Carlsbad (rather than the Sprudel), Kissingen, Cheltenham, Harrogate, &c., are indicated in active hyperæmia; the more tonic waters in the more chronic forms of congestion. A more detailed notice of the different spas and waters in relation to hepatic affections will be found under other articles. See LIVER, Functional Disorders of; and MINERAL WATERS.

STEPHEN H. WARD. JAMES F. GOODHART.

**LIVER, Hypertrophy of.**—In true hypertrophy of the liver, the increase in the size of the organ is due to enlargement and multiplication of the secreting cells, without any morbid change having taken place in these, and without the deposit around them of any morbid material. The parenchyma of an hypertrophied liver is generally firm and vascular, in some few instances pale and flabby. The hypertrophy may be *partial* or *general*. *Partial* hypertrophy may either result from localised irritation of the liver, as in tight-lacing; or it may compensate for some other portion of the organ reduced or entirely destroyed by disease. as in the large

irregular lobules of hepatic tissue formed in advanced cases of syphilitic hepatitis. *General* hypertrophy has been found in some cases of diabetes mellitus; in leukæmia; and, according to Dr. Jules Simon, in a certain cachectic condition produced under the combined influences of deprivation, scrofula, and residence in a damp locality. Whether true hepatic hypertrophy can be caused by residence in hot climates, or by prolonged exposure to malaria, seems, according to Frerichs, to be open to doubt. It is probably due in some instances to prolonged congestion of the liver, and, as a result of such a condition, may occasionally be met with in drinkers and free livers, and in the subjects of pulmonary and cardiac diseases.

W. JOHNSON SMITH.

**LIVER, Induration of.**—The liver is commonly hardened whenever it is small. Simple induration is a state in which new connective tissue seems to replace the proper hepatic tissue throughout large tracts of the liver. Often the organ becomes small and lobulated; at other times it is increased in size, and a case has been known in which it weighed eight pounds, constituting a connective-tissue hypertrophy of the liver. The diseased part presents the appearance of a completely homogeneous, whitish-yellow, firm, hard mass; which under the microscope is seen to be made up of connective tissue, in which no, or very few, elements of liver-substance can be made out.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

**LIVER, Inflammation of, Acute.**—*SYNON.*: Acute Hepatitis; Fr. *Hépatite Aiguë*; Ger. *Acute Leberentzündung*.

It is usual to divide acute inflammation of the liver into *perihepatitis*, involving the investing membrane of the liver and Glisson's capsule; and *hepatitis*, in which the parenchyma of the liver is engaged—in other words, into superficial and deep-seated; but, practically speaking, perihepatitis is rarely more than an accompaniment of hepatitis, of peritonitis, or of an acute attack of a neighbouring organ. We shall not treat of the two separately, as they are constantly associated with each other.

**ÆTIOLOGY.**—Acute hepatitis is by no means a common affection in Europe, and even in the tropics congestion is far more frequent than acute inflammation. The chief causes of hepatitis are exposure to heat and to changes of temperature—both of which have a large share in what is called tropical influence—irregular habits of life, and spirit-drinking. Irregular action of the liver and of the bowels predisposes to it. Men in the tropics are far more subject to the disease than women or children—in fact, acute hepatitis is exceedingly rare in children. The disease is uncommon in men under the age of twenty, and appears to be

most frequent between the ages of twenty-five and thirty-five. Independently of the cases in which it appears to be connected with dysentery, many authors believe hepatitis to be induced by the same causes as those which produce tropical dysentery, and tropical fevers—in other words, by malarious influences. Hepatitis or perihepatitis is sometimes occasioned by falls, or by external violence, which may operate in producing them either directly or secondarily.

**ANATOMICAL CHARACTERS.**—The liver when inflamed is usually represented as softened and congested, sometimes having a granular appearance. When cut into, more blood flows from it than usual. It is sometimes infiltrated with serous fluid, with lymph, or occasionally with small spots of pus. While portions of the liver are involved in inflammation, other portions may be unaltered; but most generally it is inflamed or congested throughout, in some parts in red patches, while in other parts there are patches of a yellowish colour. These may be traced in their changes until they are converted into abscesses, which may be single, or less frequently multiple; usually these are enclosed in a cyst, which varies from the thinnest half-formed membrane to a tolerably tough one. If there has been much perihepatitis, the liver is very commonly adherent to the diaphragm or to the neighbouring viscera by organised lymph, and its capsule is thickened.

With reference to the distribution of its blood-vessels, it has been presumed that congestion of the liver is mainly caused by obstruction to the hepatic veins, consequent on affections of the heart or lungs, or by some obstruction of the vena portæ, or morbid alteration of the blood which it conveys. In inflammation, again, the capillaries of the hepatic artery are believed to be primarily engorged. But these matters are not very certain. As respects suppurative inflammation, Budd maintained, and rightly as regards many cases, that it is the result of purulent matter taken up from ulcerating surfaces of the bowel, and conveyed to the liver by the vena portæ.

**SYMPTOMS.**—These vary much in their degree of acuteness. There is frequently in the commencement chilliness, or even some shivering, followed by fever. The appetite is impaired, and there may be loathing of food. The tongue is white, and usually has a thick white coat. The bowels are sometimes constipated; more frequently there is diarrhœa. Sometimes there is great thirst, along with irritability of the stomach and bilious vomiting. A certain degree of jaundice has been often set down as a symptom of hepatitis, but it is very unusual. There is frequently pain in the right shoulder, but by no means always. There is often a short, dry cough. There is generally some fever;

the pulse is usually from 100 to 110, and the temperature of the body is increased two or three degrees; but there is seldom present that amount of irritation which one would expect to find when inflammation is so acute that it may end in suppuration. In the region of the liver the symptoms are various. Sometimes there is sharp lancinating pain, especially when the convex surface is most affected; more frequently there is a dull, heavy pain, which is increased when the patient endeavours to lie on his left side. There is generally pain in the liver on pressure. On careful examination the viscus will often be found to be increased in bulk, but by no means always so. It may be enlarged upwards and backwards, or below the edge of the ribs, or towards the epigastrium. If the inflammation be at all acute, or if there be perihepatitis, which may involve the lower surface of the diaphragm, there may be shortness of breathing, which is often very distressing. The acuteness of suffering depends very much on whether the surface or the deeper portion of the organ is most involved in the inflammation. The urine is commonly high-coloured, and contains an excess of bile-pigment. Sometimes albumen is present, but on the whole nothing very definite is known on this point.

As regards the duration of the symptoms, if there only be perihepatitis, they will probably subside in three or four days. Deep-seated hepatitis may end in about ten days in resolution, or in the formation of abscess (*see* LIVER, Abscess of); or the disease may be protracted in a less acute form for weeks or months, producing enlargement or partial induration of the liver. In such cases, and indeed in the less acute form of hepatitis, it is often difficult to draw a distinction between chronic congestion and chronic inflammation.

**DIAGNOSIS.**—Hepatitis is not very likely to be confounded with other affections. If the surface is particularly involved, when there is much shortness of breath, and the pains are lancinating, it may be confounded with local pleurisy or pneumonia. The auscultatory sounds, as also the position of the patient, will show whether the lungs are affected—for with pneumonia, with intercostal neuralgia, with inflammation of the stomach, or with the passage of gall-stones, he will not lie on the affected side, as in hepatitis; nor is there in these diseases the excessive irritability of stomach that there is in inflammation of the liver. The pain of gall-stones comes on more suddenly, is more acute, and disappears more rapidly. Sometimes hepatitis has been confounded with enlargement of the gall-bladder, but usually the pyriform shape and prominence of that organ when over-filled should be enough to distinguish it. As for the diagnosis between hepatitis and perihepatitis, it is chiefly of

importance as regards the prognosis. The pain in the latter is usually more acute, less of a dull pain, and is less persistent. The parenchyma of the viscus is less sensitive than its surface, as has appeared to the writer, on handling and cutting off two or three inches of liver which had protruded through a wound.

**PROGNOSIS.**—The prognosis of hepatitis is grave, but its termination is generally favourable in temperate climates, especially when the case is one rather of perihepatitis than of hepatitis. In the tropics the disease is much more formidable. There is always the risk of its running into abscess; or, if that be avoided, of a state of chronic hyperæmia of the organ, with enlargement and constant tendency to relapse supervening. The complication with dysentery or chronic diarrhœa is frequent and unfavourable. The ratio of mortality differs much in different places, and in the same place in different seasons; and it is difficult to ascertain the absolute mortality of hepatitis, as long as congestion and inflammation of the liver are not distinguished in statistical returns. However, hepatitis (if cholera be excluded) has long been, and continues to be steadily, the greatest source of mortality among our soldiers in India. The French found it a very fatal disease in Algiers, and it has always been one of the most serious diseases of hot countries.

**TREATMENT.**—The activity of treatment must be regulated by the acuteness of the case, and by the presence or absence of complications, such as dysentery. In former days repeated venesection used to be employed. We now find a few leeches applied over the liver or to the anus at least as effectual. Emollient cataplasms are to be kept constantly applied to the side. The bowels should be freely acted on with the neutral salts. The action of ipecacuanha in large doses is very useful, whether it produces nausea or operates on the bowels. It is particularly indicated where there is dysenteric complication. The general feeling is that the induction of vomiting operates unfavourably, but in the early stage of the disease a certain amount of this action is not injurious. It seems to relieve the liver to some extent. Calomel and opium, and the exhibition of calomel in small doses, to touch the gums, was the old treatment in India; while the French trusted mainly to the exhibition of calomel, ipecacuanha, and opium. Both modes of treatment were believed to yield satisfactory results. The popular idea that if the patient was once salivated he was secure from the formation of abscess has no foundation in fact. If there is much general fever, the use of an ordinary diaphoretic mixture, with a larger proportion of tartar emetic, will be found useful; if nausea is produced, so much the better. When the

symptoms become less acute, the steady use of moderate saline aperients, or of iodide of potassium—in short, the ordinary treatment for congested liver, should be adopted. Counter-irritants and blistering, or the application of tincture of iodine over the liver, are of more use in this than in the earlier stages. The nitro-muriatic acid bath has long been a popular remedy in the chronic stage of hepatitis. The body may be immersed, but a bath for the feet, or sponging the side with a solution of the acid, will usually be found as efficacious. The internal use of the mineral acids the writer believes to be more effective. Change of climate exercises a singularly beneficial effect; especially that obtained by a sea-voyage. Sudden exposure to cold, however, after a return from a warm climate, must be particularly guarded against. The treatment of the sequelæ of hepatitis becomes practically that of congestion of the liver. The steady use of saline aperients, or a resort to the saline-alkaline, or to alkaline baths, as Carlsbad, Marienbad, Elster, Vichy, and others, will be found beneficial. In the acute stage the diet must be low, and limited chiefly to fluids. Great attention must be paid to diet during convalescence also. This will help materially in preventing the disease from becoming chronic; and, indeed, there is no better prophylactic against hepatitis than a carefully regulated diet, with abstinence from spirituous drinks.

J. MACPHERSON.

### LIVER, Inflammation of, Chronic.

This is usually only another name for cirrhosis. Sometimes the name is given to a perihepatitis, a thickening and opacity of the capsule enclosing the liver, and beneath which the liver-substance is found hardened and tough, due to an over-growth of the connective tissue from the capsule. Most pathologists, however, look upon cirrhosis as a chronic inflammation of the liver, and the name is usually restricted to this state. A chronic inflammation of the liver is also induced by syphilis, both congenital and acquired. See LIVER, Cirrhosis of.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

**LIVER, Malformations of.**—Abnormalities in the form of the liver are not common, and are more often acquired than congenital. The following are some of the most frequently observed malformations that are *congenital*, and due to some original defect: A more or less quadrangular liver; a rounded liver; reduced proportions or total absence of left lobe; prolongation of the left lobe in the form of a narrow tongue-like process towards the region of the spleen; abnormal grooving of the surfaces of the liver; extreme depth of normal fissures. Another occasional variety of hepatic malformation consists in extensive lobulation, and the existence of one or more additional small

lobes—a condition met with in the livers of rodent animals. An extreme instance of this extensive lobulation was observed by Dr. Dickinson, and is recorded in the *Transactions of the Pathological Society* (vol. xvii. p. 160). Acquired malformation may be due to hepatic abscess; to hydatid disease; to new-growths; to some form of chronic inflammation (cirrhosis, syphilitic disease); or, finally, to compression of the organ by tight-lacing and other means. In cases of hepatic malformation caused by tight-lacing, the upper part of the right lobe may be traversed from side to side, either by a single very deep furrow, or by two or more shallow depressions with elevated transverse folds of liver-tissue between them. In those instances in which the constriction has been so applied as to 'fold up' the liver, and to increase its convexity from before backwards, a single deep transverse furrow is often formed on the under surface of the right lobe.

According to Wilks and Moxon, compression of the waist by a strap often causes a transverse groove or depression, with a fibrous appearance, running just above the anterior edge of the liver in front.

Dr. Coats has described a furrowed condition of the upper surface of the liver, in which elevated folds of hepatic structure, with corresponding depressions, take a direction from before backwards. This furrowing occurs in cases of obstructed respiration, and is held to be due to the pressure of the lower ribs on the liver, which organ is more or less fixed by depression of the diaphragm and by the strongly contracted abdominal muscles.

W. JOHNSON SMITH.

### LIVER, Malignant Disease of.—

**DEFINITION.**—Development in the liver of cancerous or sarcomatous growths, either primary, or secondary to similar growths elsewhere; causing, generally, enlargement of the organ, with irregularity of its surface; attended with pain, often with jaundice and ascites, with marked cachexia and progressive emaciation; and having usually a rapidly fatal termination.

**ÆTIOLOGY.**—Hepatic cancer is so largely a disease transported to the liver from other organs by the various vessels that supply it with blood and lymph, that its *raison d'être* is that of the various primary growths from which it emanates—in the stomach, the intestine, the sexual organs, the breast, the eye, &c. Thus it is rare in early life. Of eighty-three cases analysed by Frerichs, forty-one were between forty and sixty years of age, and the remainder in nearly equal proportions above and below that period. Climate, habits of life, over-indulgence in the use of spirituous liquors, do not seem to play any part in determining the malady. The influence of hereditary tendency to cancer must not, however, be overlooked. Malignant

disease of the liver is primary in about one-fourth of all cases; secondary in the remainder; and, in about one-half of the cases in which it is so, the primary disease has been seated in structures connected with the portal system. Of such as *originate* in the liver, the disease in the larger number commences in the gall-bladder or the bile-ducts, and is of the nature of cylinder-celled cancer. Some few, at any rate, have appeared to be the direct outcome of the local irritation of a gall-stone.

**ANATOMICAL CHARACTERS.**—Malignant disease of the liver may be either carcinoma or sarcoma, the former being either hard or soft (scirrhous or medullary), the latter rounded-celled and occasionally melanotic. Colloid cancer is rare, and spreads from the stomach or peritoneum. Cancer may occur as an infiltration of the liver-tissue, when large masses of the liver are uniformly affected, and little or no irregularity of surface results. It usually, however, occurs in circumscribed masses, smaller nodules or larger protuberances, varying in size, to use a familiar comparison, from that of a pea to that of a child's head. These masses are more or less numerous, and usually distinct; or they may encroach upon one another, and coalesce. When near the surface, they give rise to the marked irregularity which is very characteristic of the disease; and, when large and numerous, cause considerable increase in the size of the liver. The masses on the surface are sometimes flattened, and have a central depression which has been designated as 'cancer navel.' The cut surface is either white, or reddish-white, or darker red when blood has been recently extravasated, or of varying colour, from altered blood-pigment, when the extravasation of blood has been of longer date. The portal and hepatic veins are sometimes invaded by the cancer.

**SYMPTOMS.**—In the earlier stage of this disease, the symptoms may be merely subjective, and then diagnosis will be difficult. When far advanced, however, a prominent irregular swelling may be seen, raising the abdominal parietes, and occupying often a large portion of the abdominal cavity. Lesser grades of the disease may be detected by palpation and percussion. The liver will be found to extend more or less beyond its normal limits; to be hard and resisting; and, in a large proportion of cases, irregular. In a few cases, however, when the disease is of an infiltrating form, the surface will perhaps be smooth throughout. At times there is no enlargement of the organ, and the portion affected lies under the ribs, so that physical examination does not help us. There is often tenderness on pressure, especially when the peritoneal coat is inflamed. Usually, but not always, there is pain in the liver itself; sometimes merely a feeling of tightness and fullness; at other times a gnawing, aching

pain; and some patients have described the pain as 'burning.' There is frequently also pain shooting back to the spine, over the sacrum, or about the angle of the right scapula. A sensation as of a cord drawn round the right hypochondrium has been complained of. There is sometimes pain radiating down to the lower part of the abdomen; and occasionally wandering pains in the extremities and body generally are complained of. When the stomach is intact, there may be no material disturbance of its functions, but usually derangement is manifested by loss of appetite, nausea, vomiting, and other symptoms, which will be intensified if the stomach is implicated in the disease. The bowels are, as a rule, constipated in the earlier stage, but towards the close there is often dysenteric diarrhoea. Jaundice occurs in nearly one-half of the cases of malignant disease of the liver, and is due to compression of the bile-ducts by cancerous masses within the organ, or by an enlarged lymphatic gland in the portal fissure. When once established it is permanent, and the colour of the patient varies, being pale yellow, or deep olive-yellow, or greenish, or sometimes of the dark hue which has given rise to the term 'black jaundice.' The stools in such cases are white or clayey in appearance; and the urine deep-coloured from bile-pigment. The condition of the urine, when there is no jaundice, is variable; in the earlier stages of the disease it is generally scanty and pigmented, and loaded with lithates; in the last stage, according to Dr. Parkes, copious, pale, and deficient in urea. This condition he attributes to the utter failure of digestive and nutritive power. Ascites is present in more than half of the cases, and is due either to compression of the portal vessels, or to implication of the peritoneum. Sometimes the large size of the tumour, especially if ascites to any extent be present, may cause much pressure upwards, and give rise to distressing chest-symptoms, such as embarrassed breathing or palpitation. Hæmorrhage not infrequently occurs in advanced cases. The blood may come from the stomach or bowels, and be due to portal obstruction; or may be of passive character, as in scurvy or purpura; and the bleeding may take place beneath the skin, or come from the stomach or bowels. In the latter case the hæmorrhage is accompanied, according to Frerichs' experience, by intense jaundice, and usually by somnolence and delirium. The complexion of patients suffering from the disease under consideration, when there is no jaundice, is usually sallow, anæmic, earth-coloured. There is, in a large majority of cases, progressive and, towards the close, often extreme emaciation. Generally there is no fever, but a sort of hectic may occur when the cancerous development goes on rapidly, and involves several organs. When the disease of the liver is secondary to, and

complicated with cancerous affections of other organs, as the stomach—which occurs in a considerable proportion of cases—pancreas, uterus, or mammary gland, symptoms will exist indicating such complications, but need not be specially dealt with here.

DIAGNOSIS.—When hepatic cancer is somewhat advanced, and the liver large and irregular on its surface, the diagnosis will be easily effected. In the early stage, on the contrary, and throughout some cases in which the liver is not perceptibly enlarged, one must be cautious in giving a hasty or too decided opinion. Inherited tendency to cancer; the age of the patient; in women the period of 'change of life'; a sallow, earthy aspect; progressive emaciation; and pain in the right hypochondrium, point with fair probability to the disease. But nearly the same conditions and symptoms may be associated with aggravated hypochondriasis, or chronic tendency to gall-stones; and in the latter case the difficulty of diagnosis is increased, as gall-stones are often associated with cancer. Permanent closure of the bile-duct from other causes gives rise to persistent jaundice and other symptoms, as in the case of closure by pressure from a cancerous mass. Enlargement of the liver from malignant disease may be confounded with the following hepatic enlargements and malignant tumours: 1. Albuminoid or lardaceous disease. In this affection the hard, perfectly smooth surface, with preservation of normal shape of the liver, and absence of pain in the tumour and of jaundice, will be sufficiently distinctive, accompanied as it should be by associated indications in other organs. 2. In many cases of cirrhosis, as also of malignant disease, the liver is enlarged and its surface uneven, and in both diseases there is great resemblance in the aspect and general cachectic state of the patient, and similar disturbance of gastric and hepatic function. In cancer, however, the ascites is generally but slight, and the liver, instead of contracting, as it usually does in cirrhosis as the disease progresses, continues to increase, and is marked by large hard nodules and protuberances, which contrast with the more general but less stony enlargement in cirrhosis. In cancer the skin is often moist; in cirrhosis it is harsh and dry. Intemperance is not an element in the ætiology of cancer, as it is in cirrhosis. In the latter disease, as also in lardaceous liver, the spleen is frequently enlarged. 3. Hydatid tumour is to be distinguished from a localised cancerous mass by the presence of more or less distinct fluctuation; and the absence of pain, and of serious functional and constitutional symptoms. 4. A tumour caused by hepatic abscess would probably give evidence of fluctuation; be associated with or consecutive to dysentery; and often attended by rigors, hectic fever, and characteristic shoulder-tip

pain. 5. Malformations and malpositions of the liver have been mistaken for cancerous enlargement, especially in females about the period of 'change of life.' 6. A largely distended gall-bladder has been mistaken for a cancerous projection from the liver; but the smooth oval swelling, and the site of the enlargement, are distinctive, and, as Frerichs says, a practitioner who made an erroneous diagnosis in such case would be wanting in the *tactus eruditus*. 7. Cancer of the omentum would present a movable tumour, separable, probably, from the liver by a slight area of tympanitic resonance. 8. Cancerous deposits in the left lobe of the liver may be readily mistaken for cancerous affections of the stomach. The following points will assist in diagnosis: (a) Percussion in the greatest thickening of the walls of the stomach gives a tolerably clear, tympanitic sound; in cancer of the left lobe of the liver, the sound is much more deadened, and is only somewhat tympanitic on stronger percussion-stroke; (b) careful examination of the liver, and of the stomach, when full and when empty, will also lead to a correct conclusion; (c) even when the liver and stomach are both affected, careful examination may often make out the boundaries of disease in each. 9. Malignant tumour of the right lobe may be mistaken for enlargement of the right kidney. Percussion will generally give a tympanitic sound, from the presence of intestine between the kidney and liver. The hepatic tumour is also distinguished from this and other abdominal tumours by its following the movements of the diaphragm in respiration. But when the renal enlargement is very great, diagnosis is not easy. 10. Malignant disease of the ascending or transverse colon will constitute a movable, and generally somewhat tympanitic swelling; and fecal accumulations in the colon may be removed, but not always readily, by aperients and injections. Percussion, too, will often elicit a resonant space between the enlarged intestine and the liver.

**PROGNOSIS.**—The prognosis is always unfavourable. The disease, when once fully pronounced, runs its course rapidly, the fatal termination being seldom deferred beyond a year. Scirrhus has usually a longer duration than medullary cancer.

**TREATMENT.**—This can be but palliative, and directed to rendering the inevitably fatal course as smooth as possible, by relieving distressing symptoms. Remedies which in other hepatic affections are valuable, such as cholagogues, or mineral waters, are here useless, if not worse. The diet should be plain and nourishing; and the moderate use of wine and alcohol is not contra-indicated, as in other disorders of the liver. Serious gastric and other derangements must be met by appropriate remedies; it being always borne in mind that we have to soothe the patient,

and not add to his distress by the exhibition of nauseous drugs. For the relief of pain, the various preparations of opium are indicated, and, as a rule, morphine acts the best. It may be administered either internally, or by the hypodermic method, and must be repeated when pain demands it. Local applications over the liver, as poultices, spongiopiline, &c., with solution of opium sprinkled over the surface, are useful, especially when the peritoneal coat is inflamed. Tapping should not be had recourse to for the relief of ascites, unless this becomes so great as to interfere by upward pressure with the functions of the lungs or heart. The fluid soon re-accumulates, and the effect of the operation is to hasten the fatal termination.

STEPHEN H. WARD. JAMES F. GOODHART.

**LIVER, Malpositions of.**—Abnormalities in the position of the liver are much less rare than abnormalities in its form. The more frequent forms of *congenital* displacement include—Lateral transposition, the liver being found on the left instead of the right side of the abdomen; eventration, the organ being exposed in front of the abdomen of a fœtus; the presence of more or less of the liver in the chest, through congenital deficiency of the diaphragm. In *acquired* displacement the liver may be either depressed or elevated, some rotation of the organ on its transverse axis taking place in an opposite direction in each case. Depression may be caused by pressure from above, as by effusion in the right pleural cavity, and probably to some slight extent by considerable pericardial effusion, or cardiac hypertrophy. Elevation of the liver, which takes place more frequently, may be due to pregnancy, ascites, or the presence of some large abdominal tumour. Curvature of the spine, whether lateral or angular, usually gives rise to some change in the position of the liver. In Pott's disease the organ is often forced downwards towards the crest of the right ilium.

By tight-lacing both the position and the form of the liver may be altered. The organ may be forced downwards, and at the same time so twisted on its transverse axis that its convex surface looks directly forwards, and its concave surface directly backwards. When tightly compressed, the upper surface of the right lobe becomes marked by the ribs, and presents transverse puckerings. At the same time the right lobe is bent upon itself, the concavity of its lower surface being much increased. The hepatic tissue corresponding to the summit of the arch thus formed gradually wastes, until at last the lobe is divided into two portions by a deep transverse groove, which portions are connected merely by a membranous band, composed of thickened serous membrane, and the corresponding portion of the hepatic capsule.

Attention has of late years been directed to a condition of the liver analogous, in many respects, to that known as floating or movable kidney. The first case of this kind was put on record by Cantani, in 1866, and eight other instances have since been reported by Italian and German physicians. From the analysis of these cases given by Thierfelder, it seems to be quite clear, notwithstanding the absence of any confirmation by *post-mortem* observation, that a liver, normal in form and volume, and in all other respects perfectly healthy, may, under certain conditions of very rare occurrence, be capable of considerable displacement within the abdominal cavity. In the original instance published by Cantani, the organ lay entirely below the umbilicus, and part of it rested within the pelvis. There is much difference of opinion as to the ætiology of this condition. It occurs in most instances in women who have had many children, or who have been weakened by much hard work, and, from what is known at present of the condition, is probably due to the associated influences of a pendulous abdominal wall, and elongation, probably congenital, of the suspensory ligaments of the liver.

W. JOHNSON SMITH.

**LIVER, Morbid Growths of.**—Several morbid growths have been met with in the liver, of which the following are the most important:—

1. **Non-parasitic Cysts.**—These formations are not often met with in the liver. There may be a single cyst, or a number of small cysts scattered throughout the organ. The single cyst is usually of small size, but in some instances—as in those recorded by Sir Benjamin Brodie, and in 1882 by Dr. Sharkey—may acquire very large proportions. The contents are fluid, in some cases stained by bile-pigments; in others quite clear and free from any trace of bile. This single cyst is, no doubt, very often a ‘retention-cyst,’ either of congenital origin, or the result of general interstitial hepatitis. Multiple cysts may also be due to dilatation of the bile-ducts, but in most instances they represent the condition known as cystic degeneration of the liver, which is regarded by pathological authorities in this country as the result of vacuolation or some other morbid change in the hepatic cells. The association of cystic disease of the liver with a similar condition of the kidney, the frequent occurrence of which renders very doubtful the conclusion that such is a simple coincidence, seems to indicate that the morbid conditions in these two organs are both of a similar nature, and due to a common cause. A condition much resembling that of multiple cysts in the liver, may, as has been shown by Dr. Hale White, be produced by *post-mortem* changes in this organ.

2. **Dermoid Cysts.**—Mr. Hulke has recorded an instance in which several dermoid cysts in a withered condition were found attached to the surface of the liver (*Trans. Path. Soc.* vol. xxiv. p. 157).

3. **Erectile Tumours.**—A hepatic erectile or cavernous tumour consists of a small red or bluish-red formation, of a more or less globular shape, of reticulated structure, and containing fluid blood or soft coagula. Growths of this nature are often multiple, and each of about the size of a filbert; they are usually found either along the anterior margin of the liver, or on the upper surface of the organ, near the attachment of the suspensory ligament. Each tumour is enclosed in a capsule of delicate connective tissue. Though seated at the periphery of the liver, an erectile tumour seldom projects beyond the surface of the organ. Much remains to be made out as to the pathological significance of these tumours, especially with regard to their relation to malignant disease. There is some difference of opinion as to their connexion with the hepatic vascular system. Virchow and Wilks hold that they are in communication with minute branches of the hepatic artery; whilst Frerichs states that they cannot be injected through this vessel or through the hepatic veins, but only through branches of the portal vein.

4. **Lymphatic Formations.**—The liver is sometimes found studded in all parts with minute patches of tissue of soft consistence, each patch being made up of an aggregation of lymphoid cells disposed in the meshes of a delicate reticulum. These patches of tissue are in close connexion with small vessels, from the walls of which, according to Frerichs, they are developed. This condition is associated with leucæmia.

5. **Tubercle.**—Tubercle, as met with in the liver, occurs only in the form of minute miliary granulations, scattered throughout the whole organ, but accumulated more especially on the surface. These growths have been rarely observed in the liver, and in most of the instances in association with acute general tuberculosis.

6. **Cancer.**—See LIVER, Malignant Disease of.

7. **Hydatids.**—See LIVER, Hydatid Disease of.

8. **Adenoma Hepatis.**—Under this title has been included a series of growths which, though all composed of imperfectly developed liver-tissue, differ very much from each other in pathological character, some remaining quite innocent, whilst others develop into intensely malignant and infective tumours.

9. **Benign Growths.**—Fibrous and other growths have been in rare instances found in the liver, but they do not give rise to any clinical signs.

W. JOHNSON SMITH.

**LIVER, Nutmeg.**—SYNON.: Fr. *Foie Noix de Muscade*; Ger. *Muskatnussleber*.—Nutmeg-liver consists in a chronic passive congestion of the organ, a state which may always be brought about when there exists any impediment to the circulation of the blood through the heart or lungs. The radicles of the hepatic vein become filled with blood, and thus the centre of each acinus shows a deep red, while the outer parts are either yellow or of natural tint. A nutmeg appearance is thus given to the liver, which is often shrunken and tough, with adherent capsule, and granular surface. Under the microscope the centre of the acinus is seen to be filled with dilated blood-vessels, which, pressing on the liver-cells, cause them to atrophy and undergo pigmentary degeneration, whilst in advanced stages of the disease they disappear altogether, and the centre of the acinus is made up of blood-vessels only, but there is no increase of the connective tissue in the same situation. The capsule of Glisson now and then takes on an over-growth, just as in cirrhosis; and the connective tissue between the lobule and around the vessels is considerably increased. It is to this over-growth of the connective tissue that the shrinking and hardening of the liver are due.

**SYMPTOMS.**—The liver may sometimes be felt during life under the ribs, more often not. It is sometimes tender on pressure. Slight jaundice is often present. The spleen is not enlarged, but is small—the opposite condition to that found in cirrhosis. Ascites is frequently present, especially in advanced cases. The urine is usually scanty, and contains bile. The skin generally has an icteric tint, and this, with a certain degree of lividity, produces a very characteristic appearance in the physiognomy of the patient.

**TREATMENT.**—This must be directed to the condition of the heart or lung upon which the obstruction to the circulation depends. Nutmeg-liver may always be suspected when there exists any impediment to the return of blood from the hepatic veins.

J. WICKHAM LEGG. STEPHEN MACKENZIE.

**LIVER, Pigmentation of.**—In subjects who have succumbed to intense malarious fever, and in some who during life had suffered from frequent attacks of intermittent or remittent fever in hot climates, the liver may be found stained by pigment, which may be diffused throughout the whole organ, or dispersed here and there in irregular patches. This pigmentation of the liver is always associated with a similar condition of the spleen, and frequently with staining of the nervous centres, the lungs, the kidneys, and the lymph-glands. Hepatic pigmentation is one of the chief *post-mortem* phenomena of the condition known as ‘melanæmia,’ in which the blood, especially that of the portal

system, is pervaded by granules of pigment of a black or deep-brown colour, some of which are free and isolated, some held together in irregular masses by a pale jelly, and others enclosed in cells. In the pigmented liver these granules are to be found in the portal blood, in the walls of the capillaries, and outside the vessels, scattered amongst the hepatic cells, but not within these cells. In an early stage of the hepatic pigmentation the staining affects only the periphery of each lobule, but, as the disease progresses, the deposit gradually extends towards the centre, and then attacks the hepatic venous system. The arterial capillaries also contain similar pigment-granules.

**SYMPTOMS.**—The size of the affected liver varies in different cases, and according to the severity and the stage of the disease. The organ is sometimes congested and swollen; it often remains of normal size; in some few instances it finally becomes atrophied. The main symptoms of this condition of the liver are occasional intestinal hæmorrhage, diarrhœa, and ascites. These symptoms in well-marked cases of melanæmia are usually associated with albuminuria due to pigmentary affection of the kidneys, and with more severe symptoms due to cerebral complications, such as delirium, coma, and paralysis. Melanæmia has been met with mostly in warm climates, and occasionally during severe epidemics of intermittent and remittent fever in some parts of the North of Europe. In this country it has been very rarely observed. See BLOOD, Morbid Conditions of.

Pigmentation of the liver has been observed in cases of pernicious anæmia, as a consequence of a deposit of a reddish-brown pigment containing iron in the hepatic cells and capillaries.

In cases of obstructive jaundice, the liver may be stained as a result of reabsorption by the cells of bile-pigment.

W. JOHNSON SMITH.

**LIVER, Syphilitic Disease of.**—The liver occasionally becomes diseased during the tertiary stage of syphilis, or the period of gummy deposits, the hepatic affection being associated at some period with osseous and cutaneous lesions, and with syphilitic cachexia.

**ANATOMICAL CHARACTERS.**—*Syphilitic hepatitis* may attack both the capsule (*perihepatitis*) and the internal prolongations or septa of the capsule (*parenchymatous hepatitis, syphilitic cirrhosis*). In some cases a small portion, in others a greater part or the whole of the organ is affected. In the milder form and less advanced stages of the disease the capsule is slightly thickened, and marked by a few isolated white patches, while the surface of the liver is here and there slightly grooved and indented. After prolonged in-

flammatory action the liver becomes much deformed, and is made up of a number of small lobes bounded by deep depressions, the parenchyma on section being found to be traversed by well-marked bands of tough and retractile connective tissue. The secretory structures of the liver do not undergo very much change in this disease; and, notwithstanding the retractile properties of the fibrous tissue forming the white bands, the vessels and ducts usually remain permeable. The liver-cells occasionally become loaded with fatty elements, and in some rare instances undergo albuminoid degeneration. In cases of syphilitic perihepatitis, the liver is almost always bound to the diaphragm, and sometimes to the adjacent viscera, by firm adhesions.

Formation of *gummy tumours*—‘the encysted knotty tumours of the liver,’ as they were named by Dr. Budd—occurs more frequently than syphilitic hepatitis, with which condition, however, it is often associated. In this form of syphilitic disease, the liver presents on section, especially in its deeper parts, a number of globular growths, more or less firm in consistence, of a yellowish-white colour, and varying from the size of a pin’s head to that of a large walnut. A large deposit of this kind is usually soft or cheesy at its centre, and becomes more and more firm towards its periphery, where it is surrounded by a greyish and translucent zone of incipient connective tissue, which passes gradually into apparently healthy parenchyma. A full description of the minute structure of these hepatic gummy tumours will be found in a report by Dr. Payne on three specimens shown before the Pathological Society in 1870 (*Trans. Path. Soc.* vol. xxi. p. 207). In each tumour it was found that the soft central portion was composed of granular and almost amorphous material, in which were embedded certain round or irregular translucent bodies of large size, which probably represented collections of degenerated liver-cells. The soft central portion passed imperceptibly into fibro-nucleated structure. The surrounding fibrous zone was found to be composed of dense connective tissue, with crescentic and irregularly shaped interspaces, containing masses of fatty globules or granular matter. This fibrous structure was not strictly defined from the structure of the more central parts of the tumour, and on the outside passed into the interstitial connective tissue of the liver, and became converted into masses of nucleated tissue, each of which masses appeared to be formed around a small branch of the portal vein or hepatic artery. Dr. Payne supports the view held by Virchow concerning the pathogenesis of hepatic gummy tumours, and holds that the amorphous central portion is to be regarded, not as a deposit of tissue lowly organised from the first, but

as fibrous tissue in a more or less advanced stage of involution and decay.

In cases of infantile syphilis, whilst hepatitis is not infrequent, gummata are very rarely met with.

Whether true *hepatic cirrhosis* may be caused by syphilis is open to doubt, since in most cases supposed to be of syphilitic origin it has been found impossible to exclude with confidence the idea of a probable alcoholic origin.

*Albuminoid degeneration* of the liver has not infrequently been observed in the subjects both of acquired and of inherited syphilis, and very often in syphilitic subjects who had not been previously affected with caries or necrosis of bone, with cutaneous ulceration, or with profound or prolonged suppuration. This condition of the liver, when associated with syphilis, is probably due rather to cachexia and debility than to any essentially syphilitic influence. The almost, if not quite, obsolete views that the amyloid disease is to be attributed to the action of mercury, or to the combined action of this medicinal agent and syphilis, are opposed by the facts that this condition of the liver has often been observed in a syphilitic fœtus, and also in adults who had not previously been treated with mercury, and never in non-syphilitic subjects of mercurial poisoning.

**SYMPTOMS.**—The symptoms of syphilitic hepatitis and gummy tumours in the liver are in most instances obscure, so that these complications of advanced syphilis are often overlooked. The liver in some cases is enlarged, in other cases reduced in size. In the former instance it will often be found on abdominal percussion that the relative proportions of the right and left lobes have been much altered, and that there is considerable deformity of the whole organ. Firm globular elevations on the surface of the liver may sometimes be felt through the anterior abdominal wall. Advanced syphilitic hepatitis is usually associated with slight and slowly increasing ascites, and sometimes with œdema of the lower extremities. There is seldom any well-marked jaundice. The patient often complains of a sense of weight and uneasiness in the right hypochondrium, or, in some few cases, of severe pain. In almost all cases there is some hepatic tenderness. The most constant symptoms are of a dyspeptic character; the abdomen often becomes painful and distended; and there is very commonly, at an advanced stage of the disease, obstinate and profuse diarrhœa.

**DIAGNOSIS.**—The slow progress of the disease; the absence of any severe pain in the region of the liver; a clear history of syphilis, and the presence of syphilitic lesions in some part of the body; no history of cancer; and the absence of any indications of malignant disease, whether on the surface of the body or in the abdominal cavity, all serve to

support the diagnosis of syphilitic, as opposed to cancerous, disease of the liver. In ordinary cirrhosis of the liver the progress of the disease is more rapid; the dyspeptic symptoms are more severe; the ascites is more abundant; and the indications of alcoholism are generally well marked.

**TREATMENT.**—The treatment of syphilitic disease of the liver is that usually carried out in cases of tertiary syphilis.

W. JOHNSON SMITH.

**LIVER, Tubercular Disease of.**—*See* LIVER, Morbid Growths of.

**LIVER-FLUKE.**—A common name for the *fasciola*. *See* ENTOZOA.

**LLANDRINDOD, in Radnorshire, South Wales.**—Saline, sulphated, and chalybeate waters. *See* MINERAL WATERS.

**LOBULAR** (*lobulus*, a little lobe).—Of or belonging to a lobule. A term generally applied to morbid conditions affecting individual lobules of organs which are thus constituted, such as *lobular pneumonia*, *lobular pulmonary collapse*, and *lobular hepatitis*.

**LOCAL.**—This term is used in contradistinction to the word *general*. Thus, in connexion with *morbid conditions*, it is applied to those which are confined to, or seem specially to affect, a particular part. Again, *local causes* are such as act upon a limited portion only of the body, such as a blow or a burn. *Local treatment* implies the application of remedies in the same sense.

**LOCK-JAW.**—A popular synonym for tetanus. *See* TETANUS.

**LOCOMOTOR ATAXY** (*locus*, a place; and *moveo*, I move; *á*, priv., and *τάξις*, order).—*See* TABES DORSALIS.

**LOECHE-LES-BAINS (LEUKERBAD), in Switzerland.**—Thermal calcic waters and climatic health resort. *See* MINERAL WATERS.

**LORDOSIS** (*λορδός*, bent).—A term applied to abnormal curvature of the spine forwards. It is found chiefly in the lumbar region as an increase of the natural curvature; but it may also occur in the dorsal and cervical regions. *See* SPINE, Diseases of.

**LORETO, in the Romagna in Italy.** Saline waters. *See* MINERAL WATERS.

**LOS ANGELES**, the capital of southern California. A health resort, 110 miles from the Pacific coast, in a well-sheltered fertile valley at an elevation of 371 feet. Famous for its orange and lemon groves. Pasaderra, a suburb, lies 500 feet higher, and is well

supplied with hotel accommodation. Climate mild, but not marked by the degree of equability of temperature that characterises the Pacific stations. Mean temperature 61° F.; mean daily range 24° F.; and rainfall 15 inches.

**LUCCA, in Tuscany.**—Thermal waters containing sulphate of calcium with iron. *See* MINERAL WATERS.

**LUCID INTERVALS.**—No better definition of this state has been given than that of Lord Thurlow, who calls it ‘an interval in which the mind, having thrown off the disease, had recovered its general habit.’ It must be regarded as extremely unlikely that a perfect restoration to reason can take place in the course of any long-continued insanity, without full opportunity having been afforded of testing its nature. The law more readily recognises the restoration of the mind to a state of civil capacity such as will render testamentary acts valid, than such temporary recovery as would restore responsibility for crime. If a civil act be rationally performed, the law accepts that as *primâ facie* proof of the capacity of the agent; but juries very seldom convict the accused of a crime if insanity is proved to have existed within a short period of its commission.

JOHN SIBBALD.

**LUHATSCHOWITZ, in Moravia.**—Muriated alkaline waters. *See* MINERAL WATERS.

**LUMBAGO** (*lumbi*, the loins).—SYNON.: Fr. *Lumbago*; Ger. *Lendenweh*.—Muscular rheumatism affecting the muscles and fasciæ of the lumbar region. *See* RHEUMATISM, MUSCULAR.

**LUMBAR ABSCESS.**—An abscess, generally of spinal origin, occupying the lumbar region, or pointing in the loin external to the erector spinæ. *See* Psoas ABSCESS.

**LUMBAR REGION.**—Anatomically this region is limited above by the ‘sub-costal’ line round the trunk, at the level of the lowest point of the tenth costal arch, which cuts the body of the third lumbar vertebra near its upper surface; below by the intertubercular line (Cunningham), running through the tubercles on the outer lips of the iliac crests, 1½–2 inches back from the anterior superior spines; towards the mid-line by a sagittal section through the centre of Poupert’s ligament (mid-Poupert line), which cuts the ilium behind about half an inch outside the sacro-iliac synchondrosis. We thus get mapped out a region which has somewhat the shape of half a cylindrical cone tapering upwards. It includes more than the upper inch of the iliac fossa; it does not include the triangular area between the sub-costal line and the lower

edges of the tenth, eleventh, and twelfth ribs as they slope up to the spine. Clinically, the lumbar region extends up to these ribs, and down only to the iliac crest.

The *viscera* contained in this region are few. The kidney barely descends below the sub-costal line, but a large part of it lies below the last rib. Rather more of the right kidney than of the left will lie in the lumbar region. Crossing the front of the lower half of the left kidney is the tail of the pancreas. Taking a fairly vertical course through the region, just external to the lower part of the kidney is the colon (right or left); and the beginning and ending of the transverse colon cross the front of the right and left kidney respectively, lying on the latter just below the pancreas. More or less of the small intestine fills up any gaps left. Viewed from the front, all viscera above the lower ends of the kidneys would be regarded clinically, as well as anatomically, as in the hypochondriac regions. The *peritoneum* surrounds the ascending colon almost completely, but usually leaves a considerable surface (widening with distension of the bowel) uncovered on the descending colon; this surface and the meeting of the inner and outer layers of the ascending meso-colon lie on the posterior and inner aspect of the bowel. The inner layer, as it passes from the colon towards the mid-line, covers the lower end of the kidney and, mounting thence, surrounds the transverse colon and covers the front of the right kidney, but is kept from the pancreas and most of the left kidney by the intervention of the small bag. Outside the peritoneum is the sub-peritoneal *fat*, which varies greatly in amount, and which becomes more solid and suet-like where it surrounds the kidney (peri-renal fat). Outside this is the transversalis *fascia*, always delicate, but better marked in thin than in fat subjects. Next come the *muscles*. The quadratus lumborum, 2-3 inches wide, extends from the crest to the lower edge of the last rib, lying just outside the lumbar transverse processes, to which it is attached. It is contained within an aponeurotic sheath, of which the posterior and stronger layer, attached to the tips of the transverse processes, is the posterior tendon of the transversalis. The anterior layer is attached to the fronts of the same processes, and blends outside the quadratus with the posterior layer, which forms a tendinous band half an inch wide outside the edge of the quadratus, from which the fibres of the transversalis spring and pass horizontally forwards. Whilst the posterior layer of the quadratus sheath is attached, like the muscle, to the crest and last rib, the anterior layer is continuous with the external arched ligament of the diaphragm, which extends from the first lumbar transverse process to near the tip of the last rib. From this band, fibres of the dia-

phragm rise, and between the diaphragm and the upper end of the quadratus there is usually a short duplicature of pleura. Overlapping the front of the quadratus to a slight extent, especially below, is the psoas muscle, in the substance of which is the lumbar plexus. On the dorsal aspect the erector spinæ extends farther out than the psoas over the quadratus; it is bound down by a strong layer of fascia (vertebral aponeurosis) which blends externally with the posterior tendon of the transversalis. This lamina and the layers of the quadratus sheath are the posterior, middle, and anterior layers of the fascia lumborum. Between the edge of the erector and the muscle of the transversalis is the posterior tendon of the quadratus, an inch or so wide, having the quadratus beneath its inner half. Arising from the anterior two-thirds of the middle area of the crest, and from the transversalis tendon between the crest and the erector, is the internal oblique muscle, which runs upwards and forwards to the tip of the last rib. Again superficial to this, arising from the outer surfaces of the lower ribs and often from the transversalis tendon between the last rib and the erector, is the external oblique, which runs downwards and very slightly forwards, to the outer lip of the crest at and in front of the middle. Crossing the very angle between the last rib and the erector is the lowest digitation of the serratus posticus superior, running up and out; its tendon blends with the vertebral aponeurosis beneath it and the tendon of the latissimus superficially. Lastly, the latissimus springs along an oblique line from a wide aponeurosis attached to the sacral, lumbar, and lower dorsal spines, and to the outer lip of the crest almost to the middle, and blended with the subjacent serratus tendon and vertebral aponeurosis. The muscular fibres pass upwards and forwards, overlapping those of the external oblique, except at the crest, where a small space (Petit's triangle) is usually left between the edges of the two muscles and the crest, and having the internal oblique for its floor. The subcutaneous tissue is firmer, more fibrous, and in smaller quantity over the erector than elsewhere; hence, the very thick skin of the part is less freely movable over the erector than farther forwards.

The *vessels* of the region are the lumbar, five in number, running out behind the quadratus into the interval between the transversalis and internal oblique; but the first lumbar artery lies in front of the quadratus.

The *nerves* in the area are the last dorsal, which, accompanied by the first lumbar artery, runs very obliquely from below the twelfth rib downwards in front of the quadratus, through the posterior layer of its sheath, into the interval between the transversalis and internal oblique to a little above

the iliac crest near its middle, where it gives off a lateral cutaneous twig to the buttock, and then passes in, to end near the mid-line. Next we find the ilio-hypogastric and ilio-inguinal, issuing from the side of the psoas high up, crossing the quadratus, piercing the transversalis just outside the quadratus and near the anterior spine respectively, and running on between the muscles of the wall.

*Surface-marking.*—Looked at either from the front or from the back, the outline of the lumbar region is concavo-convex from above down—the concavity above being that of the natural waist, below the ribs, the convexity below being due to the bulging of the broad muscles of the abdomen over the iliac crest, which lies at the bottom of a slight groove bounded by the above muscles and those of the hip. In front, the upper part of the linea semilunaris may lie in the region. Outside this the surface varies much in form with the fatness and muscularity of the individual; generally it is slightly convex in all directions. Behind, the thick rounded edge of the erector spinae is seen; and outside this there is a distinct depression, which can be felt when it cannot be seen. The colon lies just outside the erector beneath a vertical line dropped through a point half an inch behind the middle of the crest. In thin and muscular subjects an oblique line running from about this spot up and in towards the lower dorsal spine may mark the origin of the fibres of the latissimus from the aponeurotic tendon.

This region is of much importance, for the kidney, the ascending or the descending colon, and the psoas muscle are reached through it. Through Petit's triangle an abscess or a hernia may point. STANLEY BOYD.

**LUMBRICUS.**—By many practitioners this term is still employed to designate the large round-worm (*Ascaris lumbricoides*). The title is entirely a misnomer, having originated with Tyson (*Phil. Trans.* 1683), who called the common species *Lumbricus teres hominis*. All the larger round-worms infesting man and animals are apt to be called lumbricoids. Notwithstanding their general resemblance to ordinary earth-worms, their organisation is totally different. Occasionally, in practice, patients seek to deceive the medical attendant, by placing one or more earth-worms in the night-stool or chamber-pot. The writer once encountered an instance where a large garden lobworm (*L. terrestris*), about a foot in length, had been carefully selected for this purpose. The practitioner should not only be familiar with the differences of character presented by true and false worms of this kind, but should bear in mind that earthworms cannot live in the human bladder and intestines. See ENTOZOA. T. S. COBOLD.

**LUNACY, Law of.**—The medical practitioner is frequently required to perform duties in connexion with lunacy, the satisfactory discharge of which requires that he should have some acquaintance with the legal enactments by which they are regulated. The statutes differ slightly in the three divisions of the kingdom. It will therefore be necessary, after describing what is required under the law in England, to show where its requirements differ from those which exist in Scotland and Ireland. The details to be given here will only include what is necessary for the information of the general practitioner. Anyone who intends to devote himself specially to the treatment of the insane, or to receive one or more persons of unsound mind into his house, must comply with regulations which we cannot here set forth, but which are fully described in works upon the subject. When a person living in his own home is under treatment for insanity, the medical attendant is justified by the common law in adopting any measures of restraint which may be necessary for safety or the proper treatment of the malady. This has been decided by the courts of law in recent cases. If, however, it is proposed to place the patient in an asylum, or under the charge of any person who is to derive profit either directly or indirectly from the proceeding, it is necessary that certain forms should be carefully observed.

In the case of a *Chancery lunatic*, the patient may be taken charge of by his 'Committee of the Person,' or he may be received into an asylum or other institution for lunatics, or into a private house, on an order by the committee. Such an order must, however, have annexed to it an office copy of the appointment of the committee. If no committee has been appointed, an order by a Master in Lunacy is sufficient authority for the detention of the lunatic.

In the case of *other private patients*, it is necessary, under the Lunacy Act of 1890, to have a *reception order* made by the 'judicial authority.' The judicial authority under the Act is either a judge of county courts, a stipendiary magistrate, a metropolitan police magistrate, or a justice of the peace specially appointed for such duty, having respectively jurisdiction in the place in which the patient is. This order is obtained by private application upon a *petition*, accompanied by a *statement of particulars* and by two medical *certificates*. These must be made in the forms prescribed by statute.<sup>1</sup>

**THE PETITION.**—This document must be

<sup>1</sup> These forms can be obtained at the law stationer's; but, as they are frequently wanted with the least possible delay, we mention the names of Messrs. Shaw & Sons, Fetter Lane, E.C., and of Messrs. Knight & Co., 90 Fleet Street, E.C., as firms in the habit of supplying them.

signed, if possible, by the husband or wife, or by a relative, and, if not, it must contain the reasons for its being signed by another person, and a statement of the connexion of that person with the patient. The person signing the petition must be at least twenty-one years of age, and must have seen the patient within fourteen days of the date of presenting the petition. If a petition for a reception order has been previously presented in regard to the patient, and has been dismissed, the facts relating thereto must be stated in the petition; and there must be presented along with it a copy, to be obtained from the Commissioners in Lunacy, of the statement received by the commissioners of the reasons for such dismissal. If one of the medical certificates is not given by the usual medical attendant on the patient, the reason for this must be stated in the petition. And if an urgency order (hereafter described) has been made in regard to the patient, it must be referred to in the petition. No member of the managing committee of a hospital can sign the petition for the reception of a patient into that hospital.

**THE MEDICAL CERTIFICATES.**—One of the medical certificates must, whenever practicable, be by the usual medical attendant on the patient. Each certificate must be on a separate sheet of paper, and each person who signs a certificate must have separately examined the patient not more than seven days before the presentation of the petition, and must be a person registered under the Medical Act, 1858, and in actual practice. Certain persons are disqualified for signing either of these certificates or the certificate required in support of an urgency order. These are: The petitioner, or the person signing the urgency order, or the husband or wife, father or father-in-law, mother or mother-in-law, son or son-in-law, daughter or daughter-in-law, brother or brother-in-law, sister or sister-in-law, partner, or assistant of such petitioner or person. The statute also forbids the following persons from signing certificates: The manager of the institution into which the patient is to be received, the person who is to have charge of the patient as a single patient, any person interested in the payments on account of the patient, any regular medical attendant in the institution, or the husband or wife, father or father-in-law, mother or mother-in-law, son or son-in-law, daughter or daughter-in-law, brother or brother-in-law, sister or sister-in-law, or the partner or assistant of any of these persons. Again, neither of the persons signing the certificates can be the father or father-in-law, mother or mother-in-law, son or son-in-law, daughter or daughter-in-law, brother or brother-in-law, sister or sister-in-law, or the partner or assistant of the other of them. And no member of the managing committee of a hospital can sign

a certificate in the case of a patient to be received into that hospital.

**THE RECEPTION ORDER.**—The judicial authority may, upon the presentation of the petition and certificates, make the reception order forthwith; or, if the certificates are not regarded as satisfactory, or for any other reason, he may appoint a time, not more than seven days after the presentation of the petition, for its consideration, and may, if he thinks necessary, visit the patient. The petition is then considered in private, and no one except the petitioner, the alleged lunatic (unless the judicial authority otherwise order), any one appointed by the alleged lunatic for that purpose, and the persons signing the medical certificates—all of whom are bound to keep secret the matters disclosed—can, without leave of the judicial authority, be present. A further adjournment for not more than fourteen days may then be made for further inquiry if the judicial authority shall think fit, after which he either grants the order or dismisses the petition. A reception order is sufficient authority for the person signing the petition, or some one authorised by him, to take the patient and convey him to the place mentioned in the order; but it does not continue in force unless the patient is received in the asylum or house within seven days of its date, except when the execution of the order has been suspended by reason of a medical certificate that the lunatic is not in a fit state for removal—in which case the order continues in force for fourteen days, unless a subsequent certificate is given that the patient is fit for removal, and in that case the order remains in force only for three days after the date of that certificate.

**THE URGENCY ORDER.**—If the necessity for placing a patient in an asylum or house is regarded as urgent, an order, called an 'urgency order,' may be signed as in the case of the petition by a husband or wife, or by a relative; and, if signed by another person, the reason for this must be stated, and also the connexion of that person with the patient. The person signing must be at least twenty-one years of age, and must have seen the patient within two days of the date of the order. This order must be accompanied by a statement of particulars similar to the statement of particulars which is required to accompany the petition, and by a medical certificate stating that it is expedient for the welfare of the patient, or for the public safety, that the patient should be forthwith placed under care and treatment. The certifier in this case must have seen the patient not more than two days before his reception. An urgency order is sufficient authority for a person, authorised by the persons making the order, taking the patient and conveying him to the place mentioned in the order, and it remains in force for his detention for seven

days from the date of the order; or, if a petition has been presented, it remains in force till the petition has been disposed of. An urgency order may be made either before or after a petition has been presented; but, if made before, it must be referred to in the petition, and, if made after, a copy of it must be sent forthwith to the judicial authority to whom the petition has been presented.

The information contained in the foregoing paragraphs indicates sufficiently for the purposes of the general practitioner the steps which require to be taken for placing a person of unsound mind under care and treatment. It is of the greatest importance, however, in filling up the several forms, that the most scrupulous attention should be given to the marginal notes which are attached to the forms. Great care must be taken to ensure that the instructions contained in these marginal notes are complied with in every, even the smallest, detail. For it may happen that what may appear to many to be a trifling deviation from the instructions, will render the document invalid, and thus entail much inconvenience and distress. In regard to the medical certificates, it should be kept in view that the persons who sign them must arrive at opinions in regard to two questions which are quite distinct. Each certifier has first to determine whether the patient is of unsound mind, and, next, whether it would be proper to place him under detention. In stating the facts, it must be borne in mind that they must be such as will appear to the judicial authority making the reception order sufficient evidence of insanity. Great care must be taken to state the facts both intelligently and accurately. There must be sufficient in the facts observed by the medical man himself to justify the opinion to which he certifies. A certificate founded solely on information obtained from others is invalid, and such information should be regarded by the certifier as useful merely to corroborate his opinion. It must also be kept in view that the opinion must be directly deducible from examination of the patient on the particular day and at the particular place specified. The statement of facts observed should therefore contain at least one statement of a fact, or combination of facts, which could not be made in regard to a person of sound mind. A frequent error is the stating of the facts in such an imperfect manner that, though they may have been sufficient indications of insanity as observed, the manner in which they are recorded makes them appear to be insufficient. It is sometimes stated, for example, that a patient 'believes himself to be possessed of great wealth'—without adding that this is an erroneous belief. Another kind of failure to state the facts intelligently is illustrated by an actual instance where they were given thus: 'His appearance, manner, mode of

speaking, as well as his conduct.' These were probably quite adequate to prove to the certifier that the patient was insane, but it is obvious that they afforded no proof to those who merely read the statement.

If a lunatic, who is not a pauper, and not wandering at large, is not under proper care and control, or is cruelly treated or neglected by any relative or other person having the care or charge of him, information should be given to a constable of the district or a relieving officer or overseer of the parish, who is bound within three days to take steps, prescribed by statute, to have him placed in an institution for lunatics.

The duties performed by medical men and others in connexion with the placing of patients under care and treatment have been frequently the source of great trouble and expense, from legal proceedings taken against them by patients or others who have felt themselves aggrieved. In order to afford protection against such action, when improperly taken, it is provided in the Lunacy Act of 1890 that a petitioner or certifier 'shall not be liable to any civil or criminal proceedings, whether on the ground of want of jurisdiction or on any other ground, if such person has acted in good faith and with reasonable care.' If such proceedings are taken, they 'may, upon summary application to the High Court of Justice or a judge thereof, be stayed upon such terms as to costs and otherwise as the court or judge may think fit, if the court or judge is satisfied that there is no reasonable ground for alleging want of good faith or reasonable care.' Even under the protection afforded by this enactment, it will be understood that both expense and trouble may be occasioned by vexatious proceedings, as the steps necessary to get proceedings stayed by the High Court may involve considerable outlay.

Anyone who makes a wilful misstatement of any material fact in any petition, statement of particulars, or medical certificate, is guilty of a misdemeanour; but no prosecution for such misdemeanour can take place except by order of the Commissioners in Lunacy, or by the direction of the Attorney-General or the Director of Public Prosecutions.

A private patient may be discharged from the asylum or house in which he has been detained on the written authority of the person on whose petition the reception order was made, or, if this person is dead or incapable, by the person who made the last payment on account of the patient, or by the husband, or wife, or next-of-kin. If a patient should die while under detention, it is necessary to give notice of the death to the coroner and to the Commissioners in Lunacy.

In the case of *pauper lunatics*, the procedure is somewhat different from that re-

quired for private patients. Anyone aware of the existence of an insane pauper in a parish ought, if the case is a proper one for treatment in an institution for lunatics, to give notice to the relieving officer or the overseer. When a district medical officer under the poor-law becomes aware of such a circumstance, it becomes his statutory duty to give this notice in writing within three days after obtaining such knowledge. The relieving officer or overseer may then take steps to have the patient placed in an institution for lunatics upon one medical certificate, accompanied by the order of a justice of the peace, if the justice is satisfied that the alleged pauper is either in receipt of relief, or in such circumstances as to require relief for his proper care.

In order to place either a private or a pauper patient in an asylum in *Scotland*, a petition, accompanied by a statement and two medical certificates, has to be presented to the sheriff.<sup>1</sup> In the case of a private patient, the person signing the petition must state the degree of kinship or other relation in which he stands to the patient. In the case of a pauper, the petition must be signed by the inspector of the poor. In either case, if there be reasonable ground for so doing, the patient may be placed in the asylum on what is called a 'certificate of emergency,' signed by one medical man. If, however, the order of the sheriff is not obtained within three days thereafter, the patient must be discharged. In the case of a patient placed for profit in a private dwelling-house in *Scotland*, the fact must be reported to the General Board of Lunacy for *Scotland*, and the sanction of the Board obtained.

The procedure required for placing a patient in an asylum in *Ireland* resembles that which is required in *England*. For admission to a private asylum, an order and two medical certificates must be filled up and signed, subject to regulations resembling those already described as enforced in *England*; but the facts indicating insanity do not require to be stated in the certificates. Pauper patients are placed in district asylums, and are admitted to these institutions on application being made at the asylum of the district in which the patient resides. The necessary form is obtained at the asylum. It consists of (1) a declaration to be made before a magistrate that the patient is insane and destitute, and has no friend able or willing to pay for his board in an asylum; and to this is annexed a statement descriptive of the patient; (2) a certificate by a magistrate, and a clergyman or poor-law guardian, in corroboration of the declaration; and (3) a medical certificate of insanity. When these forms have been filled up, it is necessary to

<sup>1</sup> The regular printed forms for *Scotland* may be obtained from Messrs. T. & A. Constable, 11 Thistle Street, *Edinburgh*.

wait until it is notified to some of the friends of the lunatic that there is room for him at the asylum. The procedure specially designed for the committal of dangerous lunatics is, however, frequently adopted in placing paupers in asylums; but this is a course which ought to be avoided, and which the medical practitioner ought specially to discourage. According to this procedure, the patient requires to be apprehended by the police, and brought before two justices of the peace. These call to their aid the medical officer of the dispensary district, and either discharge the patient or order his removal to the asylum. Patients who are not destitute, but whose friends are unable to pay the rates of board charged in private asylums, are received into district asylums at low rates, upon application being made at the asylum in a similar manner to that already described for paupers. The chief difference between the two forms is that, in the case of patients not destitute, the medical certificate requires to be signed by two medical men instead of only by one.

JOHN SIBBALD.

**LUNATIC** (*luna*, the moon).—SYNON.: Fr. *Lunatique*; Ger. *Mondsüchtig*.—A designation given to persons suffering from mental disorder, because such subjects were formerly believed to be peculiarly affected by lunar influences. The term is used popularly as synonymous with insane. In medical literature it is seldom employed, but the legal relations of the word are important. The adjective 'lunatic' is also used to signify that the object with which it is associated is connected with insanity, as *lunatic asylum*. See INSANITY.

**LUNGS, Diseases of.**—SYNON.: Fr. *Maladies du Poupon*; Ger. *Krankheiten der Lungen*.—Under this title there will be described in the following pages, with certain exceptions, the various morbid conditions which affect the pulmonary organs. Pulmonary phthisis is so common a disease, so complex and variable in its pathology, and so closely associated in its aetiology and symptoms with the entire organism, that it will be most conveniently described apart from the other diseases of the lungs (see PHTHISIS). Certain other diseases which involve the lungs, if not the lung-tissue proper, and which in some nosological systems are described as pulmonary diseases—namely, asthma, diseases of the bronchi, and diseases of the pleura—will also be found described apart from the present connexion, and under their several headings. Again, disorders of respiration, such as dyspnoea, orthopnoea, and 'Cheyne-Stokes respiration,' although frequently associated with diseases of the lungs, are in other instances referable to some morbid condition of other parts, such as the blood, the heart and circulation, or the nervous apparatus of breathing, and they will

therefore be discussed in a distinct article (see RESPIRATION, Disorders of). The more important special clinical phenomena of disease of the lungs—namely, cough, expectoration, hæmoptysis, and the various physical signs—also demand more detailed and complete consideration than can be devoted to them in connexion with the various pathological conditions to which they are due. See COUGH; EXPECTORATION; HÆMOPTYSIS; and PHYSICAL EXAMINATION.

After the separation of these subjects from that of diseases of the lungs, there remain for consideration under this head a large number of morbid conditions, which rank as of the first importance in practical medicine, and which will now be enumerated. The morbid processes which affect the lungs may be readily divided into two great groups—namely, first, those which are not essentially different from similar processes in other parts of the body; and, secondly, those which are quite peculiar to these organs.

First, with respect to the former group, the lungs, like the other great viscera, may present any of the ordinary morbid conditions which affect either entire organs, or the several tissues of which they are composed. Thus, the lungs may be the subject of various injuries, leading to *perforation* or *rupture*, and may present certain *malformations* and *misplacements*. They may undergo such alterations of nutrition as end in *atrophy*, *hypertrophy*, or certain *degenerations*. Disturbances of circulation give rise to well-defined pathological conditions, such as *anæmia*, *congestion*, *hyperæmia*, '*apoplexy*,' *embolism*, *infarction*, *œdema*, and *hæmorrhage*. The inflammatory process leads to a greater variety of pathological changes in the lungs than in perhaps any other organ, and which are known as *catarrhal*, *croupous*, and *chronic pneumonia*, *abscess*, *cirrhosis*, *gangrene*, and some forms of *phthisis*. *Morbid growths* of all kinds, including *malignant disease*, may involve the lungs, whether primarily or secondarily. *Syphilis*, besides actually involving the lungs, occasionally determines or modifies the occurrence of other pathological processes within them. Various *parasites*, especially *hydatids*, are occasionally tenants of the pulmonary organs.

Secondly, the morbid conditions which are *peculiar* to the lungs are such as depend upon their special structure, relations, and functions. Thus the relation between the pulmonary tissue and the pressure within and around the lungs may be so disturbed as to lead, on the one hand, to *collapse* or *compression*, or, on the other hand, to *emphysema*. Their communication with the atmosphere, and the constant interchange that is going on between the contents of the lungs and the external air, have an important influence upon the origin, distribution, progress, and

treatment of many of the diseases which affect them; whilst the length and complexity of the respiratory passages, and their liability to disease, lead to many disturbances of the pressure, the circulation, and the nutrition within the lungs, and thus to collapse, hyperæmia, inflammation, and even destructive disease. The relation of the lungs to the circulation has an equally important influence upon them from a pathological point of view. Constituting as they do the channel of communication between the right and the left sides of the heart, the pulmonary vessels are involved in all the disturbances which affect the cardiac circulation, whether due to actual disease of the valves or of the walls, or to simple functional derangement of that organ. Congestion, œdema, embolism, infarction, hæmorrhage, and some forms of inflammation of the lungs, are the ordinary results of such circulatory disturbance of a temporary kind; and when it is more protracted, *brown induration*, as well as diseases of the bronchi and pleura, are likely to result.

Such are the principal conditions which determine and influence diseases of the lungs; and we shall here enumerate these in the alphabetical order in which they will be found referred to in the following pages: (1) Abscess. (2) Albuminoid Disease. (3) Anæmia. (4) Apoplexy. (5) Atrophy. (6) Brown Induration. (7) Cancer. (8) Cirrhosis. (9) Collapse. (10) Compression. (11) Congestion. (12) Consumption. (13) Degenerations. (14) Embolism. (15) Emphysema. (16) Gangrene. (17) Hæmorrhage. (18) Hydatids. (19) Hyperæmia. (20) Hypertrophy. (21) Induration. (22) Infarction. (23) Infiltrations. (24) Inflammation—Croupous, Secondary, Catarrhal, and Chronic. (25) Inflation. (26) Malformations. (27) Malignant Disease. (28) Malpositions. (29) Morbid Growths. (30) Œdema. (31) Perforation. (32) Rupture. (33) Syphilitic Disease; and (34) Tuberculosis.

**LUNGS, Abscess of.**—SYNON.: Fr. *Abcès du Poumon*; Ger. *Lungenabscess*.

DEFINITION.—Circumscribed suppuration of the pulmonary tissues.

ÆTIOLOGY AND PATHOLOGY.—An acute *primary* inflammation of the lungs may occasionally lead to the formation of abscess. Much more commonly, however, pulmonary abscesses are the result of *secondary* inflammations, and they are then, for the most part, associated with pyæmia.

Of acute *primary* inflammations of the lung, as causes of abscess, we have to consider those due to mechanical injuries, and those associated with acute pneumonia and with gangrene. With regard to the former, it is only necessary to remark that mechanical injuries, such as fractured ribs, penetrating wounds of the thorax, the lodgment of foreign bodies, &c.,

may cause suppuration, and so occasionally lead to the formation of abscess. That acute pneumonia may, in rare cases, terminate in abscess of the lung, has already been stated. Such a result appears to be favoured by a bad constitution, and by any circumstances which tend to impair the general health, especially the abuse of alcohol. The abscess is more common in the upper than in the lower lobes. Lastly, circumscribed gangrene of the lung occasionally terminates in abscess. This takes place by the expulsion of the necrotic tissue through the bronchi, and the formation of a pyogenic membrane from the walls of the cavity, which generates pus. The cavity may ultimately close by granulation and cicatrisation. Abscesses of primary origin are usually single.

*Secondary* abscesses of the lung owe their origin to the dissemination of infective substances by means of the blood-vessels or lymphatics, and are usually pyæmic. The secondary abscesses are of two kinds—those which follow upon infarction, and those in which there is no evidence of such antecedent change. These abscesses are almost invariably multiple. They vary in size from a pin's head to a walnut, and are usually most abundant near the surface. They are commonly surrounded by a thin zone of dark red consolidation; and, when adjacent to the pleura, this membrane over them is always inflamed. Pulmonary abscesses may also result from the aspiration of putrid substances into the bronchi, as is common after operations on the mouth, nose, and trachea. See LUNGS, Inflammation of: C. Broncho-Pneumonia.

**SYMPTOMS AND PHYSICAL SIGNS.**—The formation of abscess in the lung is rarely attended by any marked clinical phenomena, the symptoms of the disease in the course of which the localised suppuration takes place being, for the most part, but little modified by its occurrence.

When acute pneumonia terminates in abscess, either the rapid fall of the temperature which constitutes crisis does not occur, or, what is more common, its occurrence is followed by pyrexia of an irregular type. The physical signs of consolidation also persist, and there is usually great prostration. Sometimes, owing to the opening of the abscess into a bronchus, pus is coughed up; and then, if the communication with the bronchus remain free, signs of cavity may be discoverable. Before such partial evacuation of its contents, the detection of the abscess by physical examination is usually impossible. The expectoration of sputa containing large quantities of pus, and often a little blood, may continue for some weeks; the signs of prostration may gradually increase; and death may ensue in the course of from two to three months, and often earlier. Partial or complete recovery may, however,

take place, the cavity becoming quiescent and secreting only diminishing quantities of pus; and complete cicatrisation may ultimately occur. In exceptional cases the abscess opens into the pleural cavity.

Abscesses of the lungs occurring in the course of pyæmia rarely give rise to any special symptoms or physical signs. They are usually much smaller than primary abscesses; and death commonly ensues before any of them have attained sufficient magnitude to influence the general phenomena of the disease.

**DIAGNOSIS.**—The diagnosis of abscess, occurring in the course of pneumonia, rests mainly upon the persistence and characters of the pyrexia; upon the physical signs of excavation supervening on those of pulmonary consolidation; and upon the expectoration of sputa containing pus. Pyæmic abscesses rarely admit of diagnosis. Their existence may be suspected if, in cases of pyæmia, pleural friction-sounds are audible over different portions of the chest.

**PROGNOSIS.**—Abscess resulting from pneumonia very commonly proves fatal in from one to three months; it may, however, as already stated, ultimately terminate in partial or even complete recovery. The development of abscesses in the lungs in the course of pyæmia does not appear to influence the general prognosis.

**TREATMENT.**—Abscesses of the lungs rarely admit of any special treatment. Their occurrence, however, indicates the importance of doing all that is possible to maintain the strength of the patient.

T. HENRY GREEN.

### LUNGS, Albuminoid Disease of.—

In advanced cases of albuminoid disease, the lung-tissues may present more or less of this morbid change. It is of no practical importance, for it does not give rise to any evident symptoms, nor does it appear to have any specially injurious effect upon the patient.

### LUNGS, Anæmia of.—SYNON.: Fr. *Anémie du Poumon*; Ger. *Lungenanämie*.

**DEFINITION.**—A deficiency of blood in the lungs, which may be *general* or *local*.

**ÆTIOLOGY.**—Besides hæmorrhage and the other causes of general bloodlessness, there are certain local causes which produce anæmia of the lung. In senile atrophy and in pulmonary vesicular emphysema, anæmia is associated with destruction of capillaries. Local or partial anæmia of the lung is the immediate result of embolism of the branches of the pulmonary artery. It rarely happens that the main vessel is entirely obstructed by an embolus; but it, or more commonly one of its main divisions, may be compressed or obliterated by the invasion of a malignant growth or aneurysm. Aneurysm of a branch

of the pulmonary artery within the lung usually causes anæmia of the portion to which the vessel is distributed.

**ANATOMICAL CHARACTERS.**—In extreme anæmia, as after death from hæmorrhage, the lungs and the bronchial mucous membrane are exceedingly pale from absence of blood. They are of course lighter in weight than natural, but in other respects unchanged. In the general disease known as anæmia, the lung partakes with other organs of the general deficiency of red blood; but in this condition, it being not so much in quantity as in quality that the blood is deficient, the lungs are of normal weight, but paler and more moist than natural, sometimes slightly œdematous.

**EFFECTS.**—The consequences of pulmonary anæmia, when long-continued, are atrophy of its texture, as in senile atrophy and vesicular emphysema, and in local deficiency of blood from partial obstruction of a large branch of the pulmonary artery. In complete obstruction of vessels from embolism, death and sloughing of the deprived area of lung is the consequence. The sudden arrest of circulation through a limited portion of the lung gives rise to stress on the collateral circulation, the result of which is often hæmorrhage. Orth asserts that continued anæmia of lung favours the occurrence of phthisis, and attributes the proneness of the apices to attack by tubercle to their relative anæmia; he also draws attention to the tendency of people with stenosis of the pulmonary artery to succumb to phthisis (*Lehr. d. Path. Anat.*, Bd. i. p. 374).

**SYMPTOMS.**—The dyspnœa and palpitation observed in anæmia are traceable to the anæmic condition of the lungs, and have their *rationale* in the necessity for an increased diligence of respiration, to enable the diluted blood to gather sufficient oxygen for carrying on the various combustion-processes of life. The remarkable gasping and restlessness seen in cases of fatal hæmorrhage, are really the signs of asphyxia from pulmonary deprivation of blood. The symptoms of general or local pulmonary anæmia, dependent upon emphysema, embolism, &c., are lost in those of the more important diseases.

**TREATMENT.**—There is no special treatment for pulmonary anæmia.

R. DOUGLAS POWELL.

**LUNGS, Apoplexy of.**—A synonym for extravasation of blood into the lungs. See LUNGS, Hæmorrhage into.

**LUNGS, Atrophy of.**—**SYNON.**: Senile Emphysema; Fr. *Atrophie du Poumon*; Ger. *Lungenatrophie*.

**DEFINITION.**—A wasting of the constituent elements of the lungs, from defective nutrition.

**VARIETIES.**—Atrophy of the lungs may be—(a) *general*, in which all the tissues of the whole of both lungs are wasted, as in senile atrophy; (b) *local*, in which all the tissues of a portion of the lung are wasted, as in the atrophy that results from a local diminution of blood-supply; or (c) *partial*, in which some of the tissues are atrophied coincidentally with increased growth of other tissues, as in some cases of so-called ‘hypertrophous emphysema,’ and in ‘cirrhosis’ of the lung.

**ÆTIOLGY.**—The cause of simple atrophy of the lungs is that general failure of nutrition which is natural to advanced age. Hereditary predisposition may determine an earlier failure of nutritive change in the lungs. The strongly marked tendency of vesicular emphysema to recur in successive generations is certainly in favour of such a tendency to premature impairment of tissue being inherited.

Over-stretching of the walls of the air-cells in emphysema, with the consequent impediment to circulation, is an important cause of subsequent atrophy in this disease. Collapse and anæmia of lung—from pressure from without, or from the pressure of a growth or aneurysm upon one of the pulmonary vessels, cutting off the blood-supply, or on a large bronchus, diminishing the respiratory function—may cause atrophy of the whole or of a part of one lung.

**ANATOMICAL CHARACTERS.**—The appearance of an atrophied lung may be best seen in a case of natural or senile atrophy. The organ is small, light, anæmic, more or less deeply pigmented, drier in texture and less firm and resisting than natural, pitting on pressure from want of elastic resilience, and capable of being squeezed into a very small compass. The air-cells appear to be increased in size; and at some portions, if the lung be inflated and dried, large cells may be seen, evidently resulting from the coalescence of two or more infundibula. Across such cells filaments may extend the remnants of small bronchi and blood-vessels. The pulmonary artery and its branches are diminished in size, and the bronchial tubes are also thinned.

**Microscopical characters.**—The atrophic process commences at the vesicular septa, which project inwards to subdivide the infundibula or alveolar spaces of the lung into true air-cells or alveoli. The process is one of simple withering and obliteration of capillaries, dependent on diminished respiratory function and blood-volume. The septa dwindle down to mere ridges upon the infundibular walls; and these walls in their turn become thinned even to perforation and coalescence of several air-spaces. Thus, without any corresponding enlargement of lung, there is an apparent enlargement of air-cells from the simplification of structure. A certain degree of fatty degeneration, affecting especially the minute vessels and the nuclear

remains of the pulmonary epithelium, is associated with this simple atrophy, as with all other atrophic processes.

When atrophy of the lung is associated with, or the result of, other diseases, as emphysema or forcible collapse, the process is essentially the same, but is combined in the former case with over-stretching of the air-cells, and more or less thickening of the fibrous tissues derived from the bronchial and perivascular sheaths, from repeated congestions. Thus we have a larger and heavier lung; and, in the later stages, more marked fatty degeneration of its fibrous texture.

In cases of atrophy from the long-continued pressure of fluid in the pleura, the pleura is always thickened from the original inflammation, and fibrous processes are directed inwards from it between the lobules, so as to render difficult any subsequent expansion of the lung.

In the case of atrophy from compression of the lung by fibrous growth or fluid effusion, we have again often a heavier lung from increase of fibrous tissue. It is obvious that the increase in weight must always be due to attendant, often determining, disease.

**EFFECTS AND SYMPTOMS.**—The consequences of the partial atrophy of lung which accompanies the 'large-lunged' emphysema of advanced middle life are very grave. Extensive obliteration of the pulmonary capillaries, without a corresponding diminution in the blood-volume, induces a stress of circulation, a mechanical congestion, which ultimately tells back through the right heart upon the whole venous system. The damaged elasticity of the lung impairs the mechanism, as the atrophy of the alveoli impairs the function of respiration. In senile emphysema, however, the lung-atrophy, being but a part of a general atrophy of all the tissues and of the blood, causes no discomfort, provided no extra effort is attempted, and no bronchitis supervenes. Local atrophy of the lung has its symptoms merged in those of the predominant disease.

**Physical Signs.**—In senile atrophy of the lungs the chest-capacity is diminished in all directions to accommodate the small lungs. The lower ribs are approximated and their obliquity greatly increased; the upper intercostal spaces are depressed. The chest-movements are very limited. The percussion-resonance is generally increased over the chest, except over the præcordial region, which is less covered by lung than natural. The respiratory murmurs are simply enfeebled, not altered, unless there be some bronchitis present. It has been said that there may be some effusion into the pleura in atrophy of the lung, to fill up the space vacated by the shrunken organ. The mechanism of such an effusion is, however, quite inconceivable.

**COMPLICATIONS.**—There are no complica-

tions necessarily incident to senile atrophy of the lung. Bronchitis not uncommonly, however, supervenes, and proves fatal to the patient.

**TREATMENT.**—The treatment of senile atrophy of the lungs simply consists in shielding the aged person from the causes of bronchitis.

R. DOUGLAS POWELL.

### LUNGS, Brown Induration of.—

The condition recognised under this name by Laennec and Virchow, is one which is sometimes observed after prolonged mechanical congestion of the lungs, particularly that which results from disease connected with the mitral orifice. The morbid change consists mainly in excessive pigmentation, the pigment accumulating not only in the interlobular tissue, but also in the alveoli and minute bronchi, where it is enclosed in enlarged epithelial and granular cells. At the same time the capillaries are dilated, the interstitial tissue is increased, and probably the alveolar walls are thickened. The pigment is granular, and of a yellowish colour; it is derived from the blood, and seems to be of the nature of hæmatoidin. It may become brownish, reddish, or even black; and ultimately may be found free. The extent and degree of brown induration vary much in different cases. When the change is marked, the lungs are enlarged, heavy, firm, incompressible, and inelastic, not collapsing on exposure. They present various tints, from yellowish- to reddish-brown. This alteration in colour is also evident on section, and red spots are often seen, shading into black, while a brownish fluid may be expressed. Brown induration is associated with congestion of other parts of the lungs, and often with infarctions. It cannot be clinically recognised apart from these conditions, unless the affected organs should present physical signs of more or less extensive chronic consolidation in cases of known pulmonary congestion from mitral disease. No special treatment is called for.

FREDERICK T. ROBERTS.

**LUNGS, Cancer of.**—See LUNGS, Malignant Disease of.

**LUNGS, Cirrhosis of.**—A synonym for chronic pneumonia. See LUNGS, Inflammation of: D. Chronic Pneumonia.

**LUNGS, Collapse of.**—SYNON.: Apneumatosi; Fr. *Affaïssement Pulmonaire*; Ger. *Lungencollapsus*.

**DEFINITION.**—Simple diminution in size of the whole or of a part of a lung, with reduction of the volume of the contained air; caused by interference with its free entrance in inspiration.

**ÆTIOLOGY.**—The causes of collapse of the lung are either *intrinsic* or *extrinsic*; and

frequently the two classes of causes are combined. The *intrinsic* causes present actual obstruction of the respiratory passages, and include all diseases of the larynx, trachea, and bronchi attended with inspiratory dyspnoea, whether due to the pressure of external tumours, to affections of the passages themselves, or to the presence of inflammatory products, blood, or foreign bodies within them. To this class of causes belongs the collapse of the lung which is apt to follow infantile bronchitis, when the tubes become obstructed, and there is no power to expectorate. All causes that interfere with respiratory efficiency favour the occurrence of the condition named. A plug of mucus may be drawn, in inspiration, deeper and deeper into the bronchial tubes, which it obstructs, and, acting like a 'ball plug,' allows the expulsion of air in expiration, but interferes with inspiration; the air not being replaced, apneumatoses is developed; and as there is no air behind the plug of mucus, cough is powerless to expel it. In children, bronchial inflammation is exceedingly common; and, the smaller tubes being proportionately smaller in the child than in the adult, the danger of collapse is increased. When children under five years of age die of bronchitis and allied affections, apneumatoses is almost invariably present; and 25 per cent. of the total mortality of infants may be safely set down to this cause. Partial collapse of the lung from pressure on the respiratory passages will be found described in the article *MEDIASTINUM*, Diseases of. The *extrinsic* causes of pulmonary collapse are certain conditions of the walls of the chest, which diminish the force of the inspiratory act, such as paralysis or debility of the inspiratory muscles, and softness of their bony attachments. Muscular paralysis is seen in injuries to the cord. Debility of the respiratory muscles may often be observed before death. Collapse of the lung is sometimes, although rarely, met with in adult life, when great prostration occurs in the course of fever, and respiration is impeded by pulmonary congestion. Associated as it is with softness and weakness of the ribs, rickets is one of the most frequent causes of collapse of the lungs. The action of the inspiratory muscles may be still further interfered with by abdominal distension, or by the binding up of the abdomen of the infant with tight bandages. The danger of collapse is lessened when the ribs have gained firmness and fixity, and when, raised by the respiratory muscles, the thoracic cavity is enlarged, and the lungs are consequently expanded.

**ANATOMICAL CHARACTERS.**—The whole of one lung or of one lobe may be affected, but a lobe or a part only of the lung is usually involved, the affected lobules being abruptly separated from those adjoining. As a rule,

several patches of collapse occur in each lung, having a darker colour and more depressed surface than the healthy parts. The lower margin of the left lower lobe is most frequently affected. The collapsed portions of lung are similar to the liver in consistency: they resist pressure, are non-crepitant, are smooth on section, and sink in water. The bronchi are filled with mucous fluid; there is an entire absence of air in the collapsed parts. On inflation, the affected portion assumes a natural appearance, unless considerable congestion exists; whereas in pneumonia inflation cannot restore the lung to its natural appearance. In pneumonia pleurisy is rarely absent; but in collapse, uncomplicated with diathetic disease, the pleura is invariably healthy.

**SYMPTOMS.**—The symptoms of collapse of the lung vary greatly with the cause, rapidity, and extent of the morbid condition. In severe cases, for example, in the collapse that follows bronchitis in very young subjects, the symptoms are peculiar. There is great prostration, debility, restlessness, and sleeplessness. The temperature falls; the surface becomes cold, blue, or dusky; the eyes become shrunken; and the pulse is quick and small. There is a constant feeble whining cry. Respiration is very quick and shallow, as high as 70 to 80 or even 100 per minute. The rhythm is changed, the interval being between inspiration and expiration, instead of after expiration. There is no pain as in pleurisy. The cough is constant and impotent; is often followed by a cry of impatience; and differs much from the suffocative cough of bronchitis.

On examining the chest, the lower part is found retracted and diminished in diameter. The intercostal spaces sink in inspiration, and move outwards slightly in expiration. When the collapse is extensive, the percussion is dull and resistant, unless the affected lobules are interspersed among the healthy ones. The respiratory murmur is lost over the affected parts; though conducted breath-sounds, of a bronchial character, and rhonchi are generally audible almost universally. In the simpler cases of collapse of the lungs, such as occur in pertussis during the severe fits of coughing, some of these symptoms and signs may be suddenly developed, and again speedily disappear.

**DIAGNOSIS.**—Apneumatoses may be distinguished from croupous pneumonia by the comparative rarity of the latter disease in infancy; and by absence of the great heat of skin, and of fine crepitation on auscultation. From extensive miliary tuberculosis it is diagnosed by the absence of advancing symptoms of constitutional disorder, though the two conditions may co-exist. In pleurisy, the dulness on percussion, and the absence of respiratory sounds at the base, are much more marked than in apneumatoses. Con-

genital collapse or *atelectasis*, is a condition which has to be distinguished from infantile collapse or *apneumatosi*s. Readily separable by symptoms, these two conditions may be indistinguishable by physical signs. In *atelectasis* the lung retains, in whole or in part, its fetal condition, nature having failed to establish respiration and fit the child for its new mode of existence. In *apneumatosi*s the once permeable lungs cease to admit air, and thus death from *apnoea* occurs without any apparent structural change being discoverable, save that the respiratory organs bear the appearance of fetal, unexpanded lungs.

**PROGNOSIS.**—The prognosis in collapse of the lung is favourable if the affection is recent, and the child healthy, with fair muscular power, and under favourable hygienic conditions. On the contrary, the disease is generally fatal if it involve a considerable extent of lung, especially if it supervene on *atelectasis*. Death usually occurs from slow asphyxia, the effect being the same as if the size of the lung were reduced by the removal of the affected parts. As much as half of the entire lungs has been found involved, thus fully accounting for the quickened respiration, the distress, and the dyspnoea, and for the bloodlessness and extreme pallor, with cold, blue extremities. The fatality of whooping-cough in infants is mainly due to the ready collapse of the lungs, specially when the child is badly nourished and breathing impure air. The natural course of the disease is from bad to worse: more lung is involved each day; and death occurs after two or three weeks from slow asphyxia. If collapse follows acute bronchitis, death often ensues rapidly; but if recovery takes place, the lungs are slow to regain activity, and the seeds of future mischief may remain. After an attack of pneumonia, complete absence of breath-sounds may exist for a time, and then suddenly—after a blow, shock, or violent cough—air enters the collapsed portion of lung, and the respiratory sounds assume a normal character.

**TREATMENT.**—When this affection was looked upon as a form of pneumonia it was treated by depletion. Now that we realise that it is not of an inflammatory character, our object must be not to lower vitality but to diminish excessive secretion. Slight counter-irritation, by means of stimulating embrocations, is useful. An emetic of ipecacuanha will help to remove accumulation if the patient is not too weak. Expectoration may be promoted by small doses of the same drug. When the lungs are extensively involved, vital power must be kept up by the help of ammonia, steel, phosphate of iron, port-wine, and beef-tea; and the food must be selected so as to be digestible by the stomach of the infant.

E. SYMES THOMPSON.

**LUNGS, Compression of.**—**SYNON.:** Fr. *Compression du Poumon*; Ger. *Lungen-compression*.

**DEFINITION.**—Diminution in size of the whole or of a part of a lung, associated with reduction of the volume of the contained air; caused by pressure on the pleural surface.

**ÆTIOLOGY.**—Compression of the lung may arise in the course of numerous diseases or injuries affecting the chest; the compressing influence being either gaseous, liquid, or solid.

First, the admission of *air* to the pleura from without, through a perforating wound, as from a sword or bayonet thrust; or from within, as by rupture of an air-cell, or the extension of pulmonary ulceration through the pleura,—produces in either case compression. If no previous pleurisy has existed, the compression is complete; but if, on the other hand, long-standing pleurisy has caused adhesion, compression cannot take place, or will be but partial.

Secondly, compression may arise from the presence of *fluid*, such as pleuritic effusion, acute or chronic; passive, non-inflammatory effusion, as in hydrothorax; or blood, as in hæmatothorax.

Thirdly, compression of the lung by *solids* is seen in the case of various tumours of the chest, whether originating in the mediastinal structures, in the lungs, or in the thoracic parietes.

In a fourth class of cases compression of the lung is the result of the enlargement of neighbouring parts, other than the thoracic viscera; and especially of the abdomen, as in ascites, and tumours of the liver, spleen, or ovaries.

**ANATOMICAL CHARACTERS.**—Compression of the lung may be either general or local, complete or partial. A lung compressed by pleuritic effusion is found to be reduced in volume, non-crepitant, dense, but not quite insusceptible of inflation. The blood is coagulated in the affected lobes, the clot being often decolorised and adherent to the walls of the vessels, many of which are impervious, or altogether obliterated; while the pervious vessels and the air-cells of the adjacent parts are distended, and emphysema is produced. In other cases, the compressed lung proves to be anæmic, tough, and dry.

In cases of slow recovery from chronic empyema the lung is often found bound down and thickened. The thoracic cavity vacated by the shrunken lung is occupied by the displaced heart, and sometimes by the extension of the sound lung across the middle line.

**SYMPTOMS AND PHYSICAL SIGNS.**—The symptoms of compression of the lung vary greatly in accordance with its causes, the rapidity of onset, and the extent and degree of compression. If pleuritic effusion be very rapid, the dyspnoea may be exceedingly

urgent. After perforation of the pleura with sudden collapse of the lung, there also occur acute pain, dry cough, and painful spasms of the intercostal muscles. The pulse is frequent, feeble, and often irregular. Symptoms, more or less acute, of inflammation may follow. In other instances the symptoms are those of hydrothorax, or of intrathoracic tumour.

The physical signs of compression of the lung are chiefly those of the associated cause, such as pneumothorax, pleurisy, hydrothorax, or intrathoracic tumour; and partly certain phenomena characteristic of the physical condition of the lung itself. The latter vary considerably with the degree and extent of compression, but they may be described in general terms as follows: Either increased clearness of the percussion-sound over the area of compressed lung, with tubular or rarely even tympanitic quality, especially in children, or in extreme cases of compression complete loss of resonance; indefinite, weak, but occasionally rather blowing or tubular respiratory sound, sometimes mixed with scanty, dry, subcrepitant rhonchus; and exaggerated loudness and ringing quality of vocal resonance. A further description of these symptoms and signs will be found under the headings of the various causes of compression referred to.

**DIAGNOSIS.**—The diagnosis of compression of the lung is in general simply the diagnosis of the condition on which it depends.

**PROGNOSIS.**—The prognosis depends on the cause of the compression. Thus in pneumothorax it is unfavourable, though recovery may take place. In hydrothorax the ultimate prognosis is unfavourable, as it is usually an evidence of formidable, if not incurable, organic disease. In pleurisy, if the effusion has been rapid, met by prompt treatment, and uncomplicated with hectic, complete recovery may take place without much compression of lung or distortion of chest; but incompletely cured pleurisy is too frequently the first incident in the history of phthisical disease. In empyema the prognosis is more favourable than in pneumothorax or hydrothorax.

**TREATMENT.**—Little need be said as to the treatment of lung-compression. It resolves itself into that of the primary or causative diseases. Bearing in mind the injury done to the lung by compression, efforts should be made to relieve the lung before it has been irremediably bound down. The early adoption of paracentesis thoracis is the most practical means of gaining this end in pleuritic effusion. Remedies calculated to remove effusion and thus relieve the lung should be given, remembering that the more speedy the relief given to the lung the more complete will be the cure. Suitable movements of the chest might be ordered subsequently, with the view of promoting expan-

sion of its walls and of the lung. Nor must a mention of the value of elevated health-resorts, the Engadine for instance, be omitted as a means of promoting the expansion of a lung which has been compressed.

E. SYMES THOMPSON.

**LUNGS, Congestion of.**—See LUNGS, Hyperæmia of.

**LUNGS, Consumption of.**—See PHTHISIS.

**LUNGS, Degenerations of.**—Changes of a degenerative character in connexion with the lungs constitute an important element in some pulmonary diseases, or they may be the sole morbid condition present. They are of the following nature:—

1. *Albuminoid.*—This is only very occasionally noticed, in extreme cases of general albuminoid disease.

2. *Fibroid.*—Changes leading to a more or less fibroid condition of the pulmonary tissue are of common occurrence, but it is not always easy to determine whether they should be regarded as due to a chronic inflammatory process, or to degeneration, and pathologists differ in their views on this point. As a degeneration, the fibroid change may be considered as most important in connexion with emphysema, and it is regarded by some authorities as an element of much consequence in the causation of many cases of this disease. It also follows long-continued congestion, and collapse or compression of the lung from any cause. Of course much fibroid or fibrous tissue is found in the lungs in many cases of phthisis, and in connexion with pleuritic adhesions and other conditions, but this lesion must be looked upon mainly as of inflammatory origin. The effects of these changes are to make the lung-tissue firmer and tougher, but at the same time to diminish or destroy its elasticity, the elastic tissue being more or less displaced. Hence, if the lungs be exposed to any distending force, they cannot recover themselves properly, and the air-vesicles remain more or less permanently dilated.

3. *Fatty.*—This degeneration is also regarded by some pathologists as one of the main elements in originating many cases of emphysema of the lungs, and also as one of the essential changes in this disease. Here, again, the lung-tissue is impaired in its elasticity and resisting power to distension, but it is not tough. Granular fat may be visible under the microscope.

4. *Pigmentary.*—The lungs become the seat of more or less pigmentation with increasing age. They are also markedly affected in certain occupations in which carbonaceous or other materials are inhaled. In so-called brown induration of the lungs there is an abundance of pigment.

5. *Senile*.—The lung-tissue undergoes atrophy, with more or less loss of elasticity, owing to wasting of the elastic tissue with increasing age, and even a fatty degeneration may take place. Hence, in such subjects emphysema is readily set up by causes which would have no similar effect on younger persons.

6. *Secondary*.—Under this head may be included those degenerative changes which take place in morbid formations in the lungs, such as inflammatory deposits, tubercle, or cancer. These belong mainly to the fatty or caseous variety of degeneration, but calcification may also occur.

FREDERICK T. ROBERTS.

**LUNGS, Embolism of.**—See LUNGS, Anæmia of; and LUNGS, Hæmorrhage into.

**LUNGS, Emphysema of.**—SYNON.: Fr. *Emphyseme du Poumon*; Ger. *Lungen-emphysem*.

**DEFINITION.**—An excess of air in the lungs, whether due to a dilated condition of the air-sacs, or to the presence of air in the interlobular tissue.

**VARIETIES.**—There are two forms of emphysema of the lungs, namely: A. *Vesicular Emphysema*. B. *Interlobular Emphysema*.

A. **Vesicular Emphysema.**—Vesicular pulmonary emphysema exists in three forms, namely, (1) *partial lobular*; (2) *lobular*; and (3) *lobar*. The last form involves the whole of a lobe, or the whole of one or both lungs. The first form is rarely seen alone, but is generally associated with the second form, which is very common, and is found in connexion with diseases, such as bronchitis, which are attended with violent or long-standing cough. The third form is by far the most important, and will be more especially referred to in the present article. It more frequently attacks both lungs than one, and the lower as well as the upper lobes. It is a serious malady, and sometimes destroys life at an early period. Its features are characteristic: the lung-substance has a peculiar doughy feel; pits on pressure; is wanting in healthy crepitation; and has a colour very closely resembling that of a calf's lung. It has been described as 'large-lunged vesicular emphysema.'

**ETIOLOGY.**—*Determining causes and mechanism.*—With reference to the determining causes of emphysema, there are two theories, namely, the inspiratory theory, and the expiratory theory. On the first view the dilatation and rupture of the air-sacs are accounted for by the over-distension of the lungs in inspiration. On the second view these changes are considered to be caused by the strain to which the lung-tissue is subjected in violent expiratory efforts, especially the act of coughing. It has been thought by others that emphysema must be looked upon

as a complementary lesion, arising in consequence of the over-distension to which the healthy portions of the lungs are subjected in cases of pulmonary collapse. Without entering into any critical examination of the theories as to the mechanical causes of emphysema, it may perhaps be sufficient to say that there can be little, if any, doubt that the lobular forms of the disease are mainly produced by expiratory efforts, such as violent cough, or blowing wind instruments. They have their seat in those parts of the lungs which become most distended by such acts. With regard to the lobar form of the disease, however, this explanation of its mechanism does not suffice. In this affection the inspiratory power is that which distends the lungs. The pulmonary tissue has lost a portion of its elasticity, it yields to distension, and no longer reacts perfectly when the distending power ceases. Further distension follows; reaction diminishes still more; until at length in some instances the lungs become greatly enlarged.

In senile cases the loss of elasticity of the chest-walls aids in preventing the pulmonary reaction.

**ANATOMICAL CHARACTERS.**—In the early stages of emphysema there is simply a dilatation of the air-sacs; an increase in the size of the alveoli; and a diminution in the height of the alveolar walls, which, yielding with the distending cavities, become partially obliterated. As the disease progresses, the air-sacs become more distended and the walls of the alveoli sometimes completely obliterated, so that the air-sacs are quite smooth, instead of honeycombed. Then follows perforation of the air-sacs—at first slight, here and there an oval opening being discoverable; afterwards the openings become larger and more numerous. The subsequent progress of the disease is attended with further distension of the air-sacs, and rupture of the fibres of their walls. The openings thus caused coalesce, until at length the walls are simply represented by membranous shreds, and even large vesicles may form. These changes, varying in degree, characterise all the forms of emphysema. In the lobar form, however, perforation takes place to a much greater extent, *quoad* the amount of dilatation, than in the lobular or partial lobular form.

The emphysematous lung is anæmic; and its blood-vessels become widely separated, and often ruptured and atrophied. The bronchial tubes are sometimes dilated, especially in old-standing cases, and in these there is frequently found an increased development of the circular muscular fibres.

There is a form of lobar emphysema which is met with in old age, and which differs in some respects from that already described. The lungs are not so large; they are universally distended, however, to a greater or less

extent; and they present a somewhat atrophied appearance. The alterations, of which they are the seat, are probably the result of those changes which age produces in the chest-walls, impairing their elasticity. This loss of elasticity may also affect the lung-tissue. See LUNGS, Atrophy of.

**SYMPTOMS.**—A constant and generally gradually increasing dyspnoea is one of the most important and most frequent of the symptoms of emphysema. Cough, with expectoration, is generally more or less present. Hæmoptysis is rare, and, when it does occur, is slight. The patient usually complains of no pain, but of a feeling of oppression, or a 'smothering in the chest.' In severe cases of lobar emphysema this last symptom and the dyspnoea are often the only circumstances which attract the attention of the sufferer to his malady. In other instances, however, and especially when the disease is only partial, a close examination will elicit the fact that there have been bronchitic symptoms. Few cases of emphysema exist for any length of time without the occurrence of asthmatic seizures. In advanced cases the aspect is peculiar. The countenance is dusky, leaden, and puffy. The nostrils are dilated, and expand widely on inspiration, whilst the angles of the mouth are drawn down. The voice is feeble. The whole body has a cachectic appearance, and is sometimes much wasted. General dropsy often ensues.

A knowledge of the changes produced by emphysema affords an explanation of the peculiar character of the respiratory movements and sounds, as well as of the other physical signs and symptoms of the disease. The lungs being the seat of general expansion, the thorax is kept abnormally distended. Thus it can undergo but little enlargement at each inspiration. As there is no impediment to the passage of air to the air-sacs, inspiration is accomplished rapidly. Not so, however, with expiration. The lung-tissue has in great measure lost its elasticity, and reacts slowly after distension; and this results in laboured, slow, and ineffectual efforts to expel the air. Further, as the lungs are more or less riddled with perforations, their aërating surface is diminished, and this necessarily causes dyspnoea whenever any increased demand is made on the respiratory function. The quantity of blood circulating through the lungs, even from the earliest stages of the affection, is also diminished; and the destruction of the capillary vessels, which ensues when the disease is more developed, further decreases the vascularity of the pulmonary tissue. Hence its pale, anæmic appearance after death, a circumstance which serves to explain how rarely it is the seat of pneumonic inflammation.

**PHYSICAL SIGNS.**—Amongst the most important of the physical signs of emphysema

are the following: The upper part of the chest and the clavicles are prominent; the neck seems shortened; the fossæ above the clavicles are deepened; there is increased curvature of the dorsal spine; and the sternum is arched. The gait is stooping; the ribs are prominent; and the intercostal spaces are depressed. There is indeed a general increase in the size of the chest, usually most marked at the upper part. These are the features of the disease when it is extensive. If partial, or confined to one lung or part of a lung, the prominence of the chest exists on one side only, and the other symptoms and signs are less marked. The movements of the chest in respiration are peculiar. The breathing is for the most part superior thoracic, but the chest is not much expanded on inspiration, for the lungs are already inordinately distended. The lower end of the sternum and the lower ribs are drawn in during inspiration. In some cases during inspiration there is marked protrusion of the abdomen. The respiration presents other features. The inspiration is short and quick, and is followed by a prolonged and often wheezing expiration. Coughing is performed feebly, and expectoration is attended with difficulty. Percussion and auscultation elicit important diagnostic marks of the disease. When it is general, there is increased, and in some instances almost tympanitic, resonance over the whole of the chest, most marked towards the apices of the lungs, and along their anterior borders; and in partial cases almost confined to these spots, or to one side. The præcordial region is generally resonant, owing to the distended lungs coming between the heart and the wall of the chest; and the cardiac impulse can often be felt beneath the lower end of the sternum. The respiratory murmur is faint, and characterised by peculiarities which a knowledge of the anatomical condition of the lungs and of the chest-walls enables us to explain. The inspiratory murmur is short, and is followed by a prolonged expiratory murmur. This latter is unlike the sound heard in any other affection, and is, in fact, pathognomonic of emphysema. In some advanced cases the respiratory sounds are scarcely audible, if the bronchial tubes are free from mucus, and no spasm exists. Laennec described a *râle* which he thought was peculiar to emphysema. He called it *râle crépissant sec à grosses bulles*. A *râle* such as Laennec described is often heard in emphysema, but it is not a dry *râle*. It is probably produced in the finest bronchial tubes, and is a modification of the sub-crepitant *râle* of bronchitis. Although valuable in aiding diagnosis when present, yet, from its frequent absence and the difficulty of distinguishing it from the ordinary sub-crepitant *râle*, it loses much of its diagnostic import.

COMPLICATIONS AND SEQUELÆ.—Bronchitis is one of the most frequent of the diseases associated with emphysema of the lungs. It is rare for the latter affection to exist for any length of time without the supervention of the former. Bronchitis presents some peculiarities when it affects an emphysematous lung. It is rather the result of congestion than of inflammation. It often attacks the finer bronchial tubes; and, when severe, is attended with profuse secretion—a circumstance which, coupled with the fact that expectoration is less easily accomplished than when the lungs are healthy, seriously complicates the affection, and increases the danger of death from asphyxia. The inflammation sometimes runs on very rapidly, and copious purulent or puriform expectoration occurs. Even when this is the case, an examination of the tubes after death may reveal but little vascularity of the mucous membrane. These severe bronchitic attacks are very apt to be attended by the formation of fibrinous clots in the heart and the large vessels arising therefrom. Bronchitis, in a sub-acute or chronic form, is a very constant cause of winter cough in emphysematous patients.

Asthma, occurring with greater or less severity, is a frequent attendant on emphysema. The attacks come on for the most part during the night, and may possibly be due to the congestion of the lungs which takes place during sleep, or when the body is long in the recumbent posture. This congestion probably sets up an irritation, which gives rise to reflex spasm of the bronchial muscular fibres.

Secondary affections of the heart are constantly met with in advanced cases of emphysema. Many pathologists have believed that the right cavities alone become affected; but more recent observations have shown that the cardiac disease is not confined to one side. There is, in extensive emphysema, a general hypertrophy of the heart, with dilatation of all the cavities, especially of the ventricles. But hypertrophy is not the only change which takes place—valvular disease is frequently found. The deposits and thickening which occur about the valves are no doubt secondary to the changes in the muscular walls, and must be attributed to the general malnutrition produced by the disease. It is not difficult to understand how it happens that in emphysema there is general cardiac hypertrophy. The impediment which exists to the circulation of the blood through the lungs necessarily gives rise to an overloaded state of the right side of the heart; hence results increased action of the right cavities, and hypertrophy of their walls. Again, the overloaded state of the venous system, and the consequent impediment to the capillary and arterial circulation, call for increased action of the left ventricle;

and this is followed by its dilatation and thickening. There exists also another cause, which probably has some influence in producing this cardiac hypertrophy, namely, the altered position of the heart. This organ is pushed downwards, and its impulse is often felt strongly in the epigastrium. The position of the ventricles is therefore changed, and the direction of the axis of their cavities is altered with reference to that of their great vessels. This must lead to embarrassment of the circulation.

As a consequence of the changes in the heart and venous system in emphysema, dropsy often results. Many cases go on for a long time without any dropsical symptoms, whilst in others there is only slight œdema of the legs. In advanced cases, however, there is frequently general dropsy complicated with albuminuria.

General emphysema is attended in its progress with symptoms of cachexia and anæmia. In some cases there is much wasting of the muscular system, even before dropsical effusions occur. Further, the patients often have a sallow and anæmic appearance, not unlike that met with in renal and other serious organic diseases. There has been an impression that emphysema and phthisis are incompatible diseases, but recent researches have shown that this view is not correct. Indeed, in most cases of death from phthisis, patches of emphysema, involving the whole or part of a lobe, are met with; and doubtless have been produced by the fits of coughing so common in the disease. But the great question is whether tubercular deposit ever takes place in lungs which are the seat of lobar emphysema; and this question must be answered in the affirmative, although the concurrence of the two diseases is uncommon. Pneumonic consolidation is very rare in an emphysematous lung.

Pleurisy not infrequently exists in connexion with emphysema; pleuritic adhesions being often found after death. The occurrence of pleurisy must, however, be considered as an accidental circumstance. In the most extensive cases of emphysema pleuritic adhesions may not be found.

**PATHOLOGY.**—The pathology of emphysema involves some important points for consideration. The great question is whether there is any degeneration of tissue preceding or attending the affection. When the disease is partial, and has followed or is attended by bronchitis, or some other affection in which there has been violent or long-standing cough, the emphysema may be the result of mechanical violence, without pre-existing degeneration of the lung-tissue. When, however, it is of the lobar form, degeneration is probably the primary step in the affection. The facts which tend to confirm this view are: (1) the insidious manner in which the disease sometimes comes on, and the development which

it attains, without any previous history of violent or long-standing cough; (2) the frequency with which it attacks the whole of both lungs; and (3) its hereditary character. The exact nature of the degeneration has not been satisfactorily made out. Fatty matter has been found in a few instances, but not in all cases. The degeneration is probably one primarily involving the elastic fibres and other structures of the walls of the air-sacs. Whatever be the nature of the degeneration, there can be no doubt that lobar emphysema is a malady resulting from some form of malnutrition of the lung-tissue. There is reason to believe, too, that this form of emphysema is sometimes associated with gout.

**TREATMENT.**—This must be referred to under two heads, namely (1) the treatment of the disease; and (2) that of the secondary affections, which follow or are associated with it.

1. *Treatment of the disease.*—Too little attention has been paid to emphysema of the lungs as a substantive disease. Considered in the main as the result of bronchitic affections, the treatment has been chiefly directed to the control of these attacks; and in regard to the partial forms of emphysema, this is a most important object. But in reference to lobar emphysema, if we recognise the fact that it is primarily due to some degeneration of tissue, it is obvious that the treatment should be directed to check, if possible, this process. It can scarcely be expected that, when once perforation and rupture of the air-sacs have taken place, the normal condition of the lung can be restored. But, whilst we admit this, it is by no means implied that the disease is beyond control. That condition of lung-tissue which precedes the perforations—the simple distension of the air-sacs—admits of great amelioration, and further degenerative changes may be, if not prevented, at least much retarded.

The main principles of treatment should be such as guide us in the management of other constitutional diseases attended with degeneration. All measures which tend to invigorate the system, to give tone to the heart, and to improve the condition of the blood, should be resorted to. Amongst the remedies for internal administration the most useful is iron. It should be given in small and continued doses. Quinine is valuable, as are also the various bitters and other remedies for dyspepsia, from which emphysematous patients often suffer. Cod-liver oil is very useful in some cases. Strychnine has been recommended with the view of improving the tone of the muscular fibres of the bronchial tubes. It has not been found useful in this respect; nor need we wonder at this, for the disease is one primarily of the air-sacs, and not of the bronchial tubes, and, if the muscles of the latter are secondarily affected, it is rather with spasm than paralysis. Small doses of

strychnine given for dyspeptic symptoms may be useful. In some cases iodide of potassium has apparently been beneficial. Probably these cases have been associated with gout.

Breathing compressed air has been strongly advocated, and no doubt it has afforded in some cases decided temporary relief, and good effects are said to have followed its use. *See AIR, Therapeutics of.*

The regulation of the diet, and the general management, are most important. The diet should be nourishing, and a moderate amount of stimulants should be allowed. The food should be easy of digestion, and nutritious in proportion to its bulk. The stomach should never be overloaded, as that condition will give rise to dyspnoea. Errors of diet must be avoided, and the functions of the bowels should be carefully regulated. Another point is to give the lungs as little work as possible, and to let the patient breathe a pure air. All violent exercise, or physical exertion of any kind, must be strictly prohibited; moderate exercise is, however, to be recommended. Moderate walking, yachting, carriage exercise, riding at a quiet pace and on an easy horse, are important adjuvants in the general treatment of emphysema. The condition of the skin should be carefully looked to: warm clothing should be constantly worn; and the greatest care should be taken to ward off bronchial inflammation. Residence during the winter in a warm and dry climate is to be recommended.

2. *Treatment of secondary affections.*—Amongst the most important of the affections secondary to emphysema is bronchitis. No depressing measures should be used in this disease, but such as will promote expectoration, and check the secretion of the bronchial tubes, if, as is very frequently the case, this be excessive. Ammonia, the various stimulating expectorants, and iron are the most valuable remedies, together with moderate counter-irritation (*see BRONCHI, Diseases of*). The dyspnoea attendant on emphysema admits only of palliative treatment, but is often greatly improved by the observance of the rules laid down for the general management of the disease. The dyspnoea is always increased by the presence of bronchitis; by the stomach or bowels being overloaded; and by the general over-distension of the venous system, which necessarily ensues as a consequence of the impediment to the flow of blood through the lungs. Care should be taken to prevent any flatulent distension of the stomach or intestines, and to keep up a good action of the bowels, liver, and kidneys. For the relief of the asthma which frequently exists in connexion with emphysema, full doses of iodide of potassium are often useful. Stramonium may also be smoked, and other measures beneficial in ordinary spasmodic asthma may be tried (*see ASTHMA*). In refer-

ence to the treatment of the dropsical symptoms, which follow as a secondary consequence of emphysema, the reader is referred to the articles DROPSY; and HEART, Dilatation of.

**B. Interlobular Emphysema.**—This condition, in which the excess of air in the lungs is contained, not in the air-sacs and alveoli, but in the connective tissue between the lobules, is described under the head of EMPHYSEMA, SUBCUTANEOUS, to which article the reader is referred.

A. T. H. WATERS.

**LUNGS, Gangrene of.**—SYNON.: Fr. *Gangrène du Poumon*; Ger. *Lungenbrand*.  
DEFINITION.—Death of a portion of the substance of the lungs.

Gangrene of the lungs is of two kinds, namely, (1) *diffused*, in which the whole of one lobe or lung is affected; and (2) *circumscribed*, in which a portion only of a lobe undergoes gangrenous change.

ÆTIOLGY.—Gangrene is sometimes a result of acute pneumonia. It has been known to follow the inhalation of noxious gases. When it occurs independently of these causes it is an evidence either of extreme constitutional depression, or of pressure interfering with the circulation and nutrition of the lung. An aneurysm or mediastinal growth, pressing upon the main arterial, venous, and nervous trunks at the root of the lung, is perhaps the most common cause of diffused gangrene; whereas the more circumscribed form of the disease is a sequela of acute and limited pneumonia, cancer, bronchiectasis, or rapid phthisis in a debilitated subject. In pulmonary apoplexy gangrenous change sometimes occurs, the gangrene being limited to the portion of lung involved in the originating extravasation of blood. Pneumonia caused by a foreign body in the air-passages is apt to run on to gangrene. The diffused gangrene that occurs in drunkards and lunatics, in asthenic fever, measles, small-pox, and typhus, evidences excessive nerve-prostration and loss of nutritive power. In children, gangrene of lung sometimes follows cancrum oris, as well as the eruptive fevers.

ANATOMICAL CHARACTERS.—The colour of a gangrenous lung is dark, dirty olive, or greenish-brown. It is moist or even wet; and either of the consistence of engorged lung, or softer and more diffuent. The odour is that of external gangrene or decomposed flesh, and is distinctive during life, rendering the room in which the patient lies horribly offensive. When scattered patches of gangrene occur, there is often in one part a solid mass of greenish lung-tissue, and in another a central sloughy or gangrenous cavity, surrounded by a broad rim of soft infiltrated lung. The seat of circumscribed gangrene is usually the periphery of the lung, and the lower lobes. If a bronchus open into the

gangrenous patch, inflammation of the bronchial membrane is set up. In rare cases the pleura is involved, and pyo-pneumothorax is induced. Sometimes the pulmonary arteries are found plugged, and more often the bronchial arteries. In those rare cases in which recovery takes place, interstitial pneumonia is set up, leading to encapsulation of the gangrenous spot; the sloughs are ejected; and cicatrization follows, as in pulmonary abscess.

In diffuse gangrene the whole of one lung is sometimes involved. The pulmonary tissue is then converted into a black, putrid substance, saturated with blackish purulent fluid; or the gangrenous part merges gradually into œdematous or hepatized tissue.

Embolism, arising from the introduction of putrid matter into the veins, and leading to abscess in various organs, may follow either form of gangrene. Secondary gangrenous change is frequently met with in other parts of the same or in the opposite lung.

SYMPTOMS.—It is seldom possible to diagnose gangrene of the lung until the purulent discharge reaches a bronchus and is ejected. Then the sputa are found to separate speedily into layers—a superficial froth, a liquid middle layer, and a lower sediment. The smell of the sputa and breath is pathognomonic. The dyspnœa and prostration are usually great. The physical signs are those of softening and excavation, percussion being either dull or tympanitic, and loose crepitation being soon replaced by gurgling and perhaps amphoric breathing. The passage of the circumscribed into the diffused form may be traced by watching the physical signs.

DIAGNOSIS.—Suppurative ulceration of the bronchial cartilages gives rise to great fœtor of breath. Sometimes a gangrenous odour in the breath occurs when the putrefactive change is limited to the secretions; and it may be present also in pyo-pneumothorax with internal fistula. These several diseases must be excluded by a careful estimation of the history and the physical signs. See BREATHE, The.

PROGNOSIS.—The prognosis of pulmonary gangrene is hopeless in the diffused form; and it should be given very cautiously, even when the symptoms or signs point to a limitation of the mischief.

TREATMENT.—Every attempt must be made to support the strength by nourishing food and stimulants; bark and ammonia, quinine and acids, iron and cod-liver oil being indicated. The inhalation of creasote or carbolic acid in spray may be tried, or of turpentine given off from hot water.

When a gangrenous abscess of the lung exists, and it is evident that the passage of fœtid matters through the bronchi is setting up dangerous irritation, leading to exhausting discharge, or threatening to poison the system, the question of tapping the gangrenous

cavity should be entertained. The introduction of a drainage-tube sometimes affords immediate relief in such cases. The fœtor of breath ceases; the offensive secretions, being no longer locked up in the lung, lose their putrescent character; and the relief to the constitution is great. An accurate diagnosis is in such cases essential, the danger of the operation being greatly enhanced if the abscess is at a distance from the chest-wall, and if the lung is not adherent to the costal pleura. The operation should only be performed when there is but little prospect of recovery without it, but must not be delayed until the vital powers are too reduced to allow of ultimate convalescence.

Besides the general treatment which co-existing disease may require, special attention must be given to the removal of the gangrenous odour from the atmosphere of the room, from the sputa, and from the patient, who is apt to exhale from the skin a similar odour to that given off in the breath. Sulphurous acid, carbolic acid, or chloride of lime fulfils the first indication; Condyl's fluid the second; and this may also, when diluted, form a useful wash or gargle. The sulpho-carbolates, when administered freely, have a distinct value in removing the fœtor from the skin, and making the patient less unapproachable.

E. SYMES THOMPSON.

**LUNGS, Hæmorrhage into.**—SYNON.: Extravasation of Blood into the Lungs; Pulmonary Apoplexy; Fr. *Hémorrhagie du Poumon*; Ger. *Lungenblutung*.

**ÆTIOLOGY AND PATHOLOGY.**—In the article HÆMOPTYSIS will be found enumerated the causes which lead to pulmonary hæmorrhage. In the present article only those extravasations of blood into the lungs are included which do not depend upon direct injury to the organ, or upon exposure and rupture of vessels in the course of destructive disease affecting it.

Hæmorrhage into the substance of the lungs may be *diffuse*, *punctiform*, or *circumscribed*.

(1) *Diffuse pulmonary apoplexy.*—The diffuse extravasation of blood into the lungs is an extremely rare condition. Some cases have been recorded, however, in which it has arisen from primary disease of a branch of the pulmonary artery. The lung-tissue is broken down by the hæmorrhage into it; and the patient soon succumbs.

(2) *Punctiform hæmorrhage*, and (3) *circumscribed or nodular pulmonary apoplexy*, are not of uncommon occurrence, and are attendant upon the same morbid conditions of the lung. Mitral disease—mitral stenosis especially, but also mitral regurgitation—is the chief remote cause of these two forms of pulmonary apoplexy.

In certain purpuric states of system, which

we need not here specify, punctiform hæmorrhage in the lungs is possible; the purpura much more frequently affects, however, the pleural surface or bronchial mucous membrane. Mechanical congestion of the lungs, from the above-mentioned forms of heart-disease, is by far the most common condition upon which this minute and interstitial form of hæmorrhage supervenes, giving rise to no additional symptoms, but causing considerable and peculiar pigmentation of the lung. The interstitially thickened lungs acquire a brownish tint, from the absorption of the blood-spots, leaving hæmatin behind; and the appearance has given rise to the term *brown induration* of the lungs. See LUNGS, Brown Induration of.

Nodular or circumscribed pulmonary apoplexy is often associated with the petechial hæmorrhage just described, and, like it, mostly supervenes upon the mechanical congestion of the lungs arising from heart-disease. There are two ways in which this form of hæmorrhage may be produced. The first way is by rupture of capillaries or small veins under the heightened pressure of the pulmonary circulation. An effusion of blood thus occurs, which fills up one or more lobules, and coagulates to form the dark firm consolidations so characteristic of the lesion. Or a branch of the pulmonary artery becomes obstructed by an embolus, for instance, by a fragment of coagulum conveyed from the right auricle, or from one of the systemic veins (phlebitis, varicose veins); and its territory becomes at once filled with blood, in the following manner. The pulmonary arterial vessels do not communicate with one another, each branching separately to its capillary distribution; the pulmonary veins, on the contrary, inosculate freely, and, moreover, are not provided with valves. Thus, when the onflow of blood is arrested through the obstructed arterial branch, venous regurgitation through the capillaries from collateral pressure fills up the precluded vascular area with stagnating blood, and the intra-alveolar tissues become speedily occupied with its effused corpuscles. In some cases the vessel may not at first be accurately closed by the embolus; the onward current is then retarded instead of being quite arrested; the mechanism is, however, practically the same.

**ANATOMICAL CHARACTERS.**—A lung that is the seat of this form of hæmorrhage is usually toughened and heavy. Some hard, and more or less square, flat surfaces may be felt and seen raised above the general surface of the lung, which has shrunk below their level. The pleura covering such patches is darkened in colour, and presents flakes or granulations of lymph, impairing its translucency and smoothness. On making a vertical section through one of the surfaces, it is found to form the base of a more or less conical mass,

which has a firm damson-cheese-like section, and is sharply defined from the surrounding tissue. In its axis is seen a branch of the pulmonary artery, occupied by partially altered clot. There is usually some staining of the pulmonary tissue immediately surrounding the apoplectic nodule, from imbibition. Such hæmorrhagic nodules vary greatly in number and size; there are usually several in each lung, of about the size of a walnut; but one may occupy a whole lobe. They also vary in appearance according to the date of their occurrence; their colour, at first that of dark blood-clot, passes through pale chocolate or catechu tint to yellowish-red or pale yellow, as the colouring matter becomes gradually absorbed. The whole extravasation may be gradually and completely absorbed, leaving the restored lung but little damaged; or a shrunken fibrinous deposition or blood-cyst, with surrounding induration, more rarely a calcareous centre, may mark the site of former hæmorrhage. It should be added that these extravasations, although generally near the pleural surface of the lung, are not always so, but may occur deeply in its substance; they occur more frequently in the right lung, and generally in the upper lobe.

**SYMPTOMS.**—Amidst the distressing symptoms which are attendant upon the conditions leading to pulmonary apoplexy, it would be difficult to single out any diagnostic of this special lesion. An exacerbation of dyspnoea already terrible enough, or a sudden failure of pulse, may perhaps be noted. Dark scanty hæmoptysis is, however, the pathognomonic sign, the frothy mucous expectoration containing some streaks, or small clots, of dark coagulated blood. Some circumscribed patches of dulness, with bronchial breathing and neighbouring crepitation, may perhaps be made out, especially in the mammary and mid-axillary regions.

**PROGNOSIS AND TREATMENT.**—These lesions are among those which often close the scene in the heart-disease to which they are accessory; and are therefore irremediable. Sometimes when, from any cause, their occurrence appears to have been hurried forward; when the lividity is great, the dyspnoea urgent, and yet the disease is not of long duration; wet-cupping or bleeding from the arm will certainly give great temporary relief, and perhaps avert immediate danger.

R. DOUGLAS POWELL.

**LUNGS, Hydatids of.**—**SYNON.**: Fr. *Kystes Hydatiques du Poumon*; Ger. *Lungenechinococcus*.

**DEFINITION.**—A disease due to the presence of hydatids in the lungs.

**ÆTIOLOGY.**—Hydatid cysts in the lungs rarely occur in this country as a primary disease of these organs, but they are not infrequently met with as an extension of disease from the liver.

The general causes of hydatid disease are elsewhere discussed, but the circumstances which determine the localisation of the hydatid are not clearly ascertained. In Australia, where the affection is very common, it is met with sometimes in the lung without any other organs being affected; although, in many cases in which the lungs are diseased, evidence of the existence of the same disorder elsewhere is not wanting. Of 100 cases, the liver was the organ affected in 70, the lung in 12. The great prevalence of the disease in Melbourne is said to be due to drinking from water-holes frequented by sheep, and from eating watercress or uncooked salads containing the ova of the parasite.

**ANATOMICAL CHARACTERS.**—The general anatomy of hydatid disease will be found in the article describing this parasite (*see* ENTOMOZOA). Single sacs of acephalocysts are by far most usual, varying in size from a pigeon's egg to a man's fist. Sometimes the upper and sometimes the lower lobes are the seat of the cysts. They are developed in the interstitial pulmonary tissue; as growth proceeds, the neighbouring parenchyma is converted into fibro-cellular tissue, and undergoes obsolescence. The parent sac, containing the echinococci, is adherent to the surrounding tissue.

If the parent sac be destroyed by inflammation and consequent suppuration, a communication is established between the cavity and the bronchi, through which the daughter-cysts may be ejected; just as in more common instances they may escape from the liver after perforating the diaphragm and lung. Not infrequently the pulmonary sac communicates with a similar sac in the liver. It is natural to infer in such cases that the disease originated in the liver. Indeed, it has been stated that primary hydatid of the lung is unknown.

**SYMPTOMS AND PHYSICAL SIGNS.**—Hydatids may exist in the lung for a considerable time without giving rise to any noticeable symptoms; but as the tumour enlarges and presses upon the surrounding tissues, hæmoptysis occurs, as also bronchitis, pneumonia, or even gangrene. Sometimes the cysts perforate the pleura and cause pneumothorax, or make their way through the diaphragm into the abdominal cavity. The more usual course is the converse of this, namely, that a hydatid of the liver exists perhaps for years; causes abdominal distension; and eventually discharges its contents through the diaphragm into a bronchus; then expectoration of blood occurs, with gooseberry-like 'skins,' varying in size from a nut (in which case the cyst may be expelled whole) to an orange (in which case the sacs are shrivelled and empty), and causing suffocating cough during expulsion.

If the site of the tumour be superficial, altered breath-sounds and percussion-note

may be observed; but if it be deeply seated, the physical signs may escape detection. If bronchitis or pneumonia be set up, the signs and symptoms of these disorders mask those of the originating disease. Often the symptoms are like those of rapid phthisis, namely, cough, muco-purulent expectoration, hæmoptysis, night-sweats, and emaciation. The meaning of these symptoms is apparently confirmed by the physical signs, namely, dulness on percussion; absence of breath-sounds, or prolonged expiratory murmur; and, when the cysts burst, gurgling and pectoriloquy. Unless the daughter-cysts or hooklets are expectorated, there is nothing to point unmistakably to the nature of the disease. When perforation of the diaphragm occurs, hepatic symptoms or those of pleurisy supervene. The patient looks anxious; the features are collapsed; the skin is clammy and livid; the extremities are cold; incessant paroxysmal cough occurs, with vomiting; and by degrees sallowness and jaundice make their appearance. Symptoms of acute pneumonia may occur—of consolidation, followed by excavation; the expectoration being at first rusty, then bile-tinged, muco-purulent, and fetid, and containing, besides shreds of lung-tissue, entire cysts or portions of them.

**DIAGNOSIS.**—It may be difficult to distinguish a large hydatid cyst from pleuritic effusion, as the lung may be displaced, the chest bulges, and the intercostal spaces become prominent and fluctuating. The rounded outline of the dull space, the absence of acute symptoms, the history of gradual onset, the absence of ægophony and of alteration of physical signs on change of posture, will guide the decision; and an exploratory puncture, which gives exit to a clear, saline, non-aluminous fluid, containing possibly hooklets or fragments of cysts, will confirm the diagnosis. The conduction of the heart-sounds and impulse, and the tense unyielding condition of the bulged side on palpation, may lead to the suspicion of mediastinal tumour; but in the case of hydatids there is seldom any visible venous engorgement, or laryngeal or œsophageal pressure-sign, as in aneurysmal or other mediastinal growths. There is, moreover, generally a freedom from cachexia or constitutional disturbance. In circumscribed abscess the neighbouring lung is rarely so free from disease as in hydatid.

**PROGNOSIS.**—Although the symptoms may be so severe as to threaten immediate death from suffocation, recovery occurs in at least half the cases in which hydatid disease begins in the lung, and one-third of those in which it spreads from the liver. If the cyst is allowed to burst of itself, recovery takes place in from 30 to 40 per cent. of cases. But the mortality is greatly reduced by early tapping.

**TREATMENT.**—Palliative treatment must be directed mainly to the mitigation of pain and other urgent symptoms. Curative treatment consists in destroying the vitality of the cyst. If the fluid contents are drawn off, or allowed to escape, the death of the hydatid may occur, or inflammatory action may be set up, leading to suppuration. If the fluid re-collect and pressure-signs recur, iodine or other stimulating fluid may be injected. The iodide and bromide of potassium, with kama-la, have gained among Australian physicians reputation in these cases, especially when combined with the use of the trochar.

E. SYMES THOMPSON.

**LUNGS, Hyperæmia of.**—**SYNON.:** Congestion of the Lungs; Fr. *Hyperémie du Poupon*; Ger. *Lungenhyperämie*.

**DEFINITION.**—Excess of blood in the lungs, whether local or general.

**VARIETIES.**—Pulmonary hyperæmia may be *active, passive, or obstructive*.

The morbid appearances and symptoms are different in these three kinds of hyperæmia, and they may, therefore, be best considered separately.

**A. Active Hyperæmia of the Lungs.**  
**SYNON.:** Active Congestion; Active Affluxion.

**DEFINITION.**—A determination of blood to the lungs.

**ANATOMICAL CHARACTERS.**—Active hyperæmia, or congestion, may affect any portion of the lung, which remains crepitant and little changed, save that it is more crimson in colour, and contains slightly more blood than natural. The condition is indeed rather a vital or physiological one; and, as in active hyperæmia of the skin, may present no *post-mortem* appearances. On section, however, the lung usually exudes some frothy serum, tinged with blood; and sometimes, especially when the hyperæmia is local and arises from collateral stress of circulation, there is found hæmorrhage into the lung. The mucous membrane of the bronchial tubes is minutely injected, or it may be quite natural in appearance.

**ÆTIOLOGY.**—The causes which produce inflammation of the lung will also produce active hyperæmia, namely, cold, irritation, adjacent inflammation, &c. Increased action of the heart during violent effort or excitement, whether from mental emotion or from drink, will produce the same effect. It is said that the pressure of blood in the pulmonary artery increases more rapidly than that in the aorta during exertion (Colin). Hæmoptysis from pulmonary hyperæmia is commonly produced by the excessive imbibition of stimulants. Predisposition, either hereditary or in consequence of present disease, renders these last-named causes much more readily operative. Obstruction to the passage of blood through one portion of the pulmonary system of vessels, for ex-

ample, by embolism, or by destruction of capillaries, will cause increased collateral activity of circulation. Sudden suppression of menstruation may cause active determination of blood to the lungs. Sudden diminution of the atmospheric pressure within the chest, as during violent inspiratory efforts, whilst the trachea is closed, for instance, in croup, laryngismus, or whooping-cough, may cause active determination of blood to the lungs.

**EFFECTS.**—Several important effects may be produced by active hyperæmia of the lungs. Hæmorrhage is rarely extensive, unless there be attendant organic lesion present. Active hyperæmia of the lung constitutes the first stage of exudative or croupous pneumonia, with fibrinous exudation into the air-cells. Pulmonary œdema may result from the excessive blood-pressure, the serum exuded being frothy and blood-tinged. Vesicular catarrh is not so distinctly a consequence of hyperæmia, with which, however, it is often associated.

**SYMPTOMS.**—The symptoms of active pulmonary hyperæmia are dyspnœa; more or less pyrexia; cough; and sometimes copious hæmoptysis, in which case precedent organic disease must be suspected. The rusty sputa of the first stage of pneumonia is that most typical of pulmonary hyperæmia.

**DIAGNOSIS.**—The diagnosis would rest mainly upon the suddenness of attack, and the evidence of a sufficient determining cause, with or without predisposition.

**PROGNOSIS.**—Active hyperæmia, save in some cases of collateral afflux, is necessarily a transient affection, subsiding in a few hours, or passing on to inflammation.

**TREATMENT.**—The first point in the treatment of this condition is to secure absolute rest in bed, with silence, and removal of all causes of excitement. Derivatives; mustard or linseed poultices to the chest; perhaps cupping, or even blood-letting; warmth to the extremities; saline purgatives; and a low diet, without stimulants, may all be employed. The special cause of the hyperæmia should be treated. As a rule, astringent medicines should be avoided. Digitalis is useful to calm the circulation, especially after excitement from alcohol. If the pyrexia be very marked, pneumonia may be expected, and saline diaphoretics are especially indicated.

**B. Passive Hyperæmia of the Lungs.**  
SYNON.: Passive or Hypostatic Congestion.

**DEFINITION.**—An incomplete stagnation of blood in the lungs.

**ÆTIOLOGY.**—Passive or hypostatic congestion of the lung is a condition of hyperæmia affecting by preference the most dependent parts of the lung. Failure of heart-power, an inability to propel the blood clear through the pulmonary capillaries, is the chief cause of this condition. In states of

exhaustion from low fevers, especially typhus and typhoid; after severe surgical operations; in extreme old age; or towards the end of prostrating illness, this failure of heart-power, and consequent stagnation of blood in the lower parts of the lung, usually the bases, is apt to supervene, and is one of the common modes of death. An altered condition of the blood, so as to render its passage through the capillaries more difficult, is also stated to be a cause of this form of congestion of the lungs and of other organs. In uræmic and icteric conditions, and in the febrile state, the blood does not pass through the capillaries with the same facility as in health, and hypostatic congestions are more apt to occur. Finally, when the vessels have lost their tone, and the heart fails in power, gravitation exercises its influence in attracting the blood to the most dependent parts.

**ANATOMICAL CHARACTERS.**—Passive hyperæmia almost always affects the bases of both lungs, although often not in an equal degree, the difference depending mainly upon the position of the patient during the last days of life. The affected lung is dark-coloured, and engorged with dark blood. Its tissue is more or less deeply stained with blood, and is less crepitant than natural, yielding also more readily than natural under the pressure of the finger. If thoroughly washed, however, in a gentle stream of water, the lung-texture will be found to be but little altered. This condition very readily passes into a low form of pneumonia, and thus portions of the lung may be found consolidated, having much the appearance and consistence on section of a congested spleen (splenification, hypostatic pneumonia). The bronchial tubes and pleura are affected by *post-mortem* staining.

**SYMPTOMS.**—The symptoms of this form of congestion of the lungs are lividity, especially of the lips and extremities; and quickened shallow breathing; superadded to those of extreme prostration. Dulness on percussion, with enfeebled breathing and moist crepitant *râle*, are found over the bases of both lungs, but in greatest extent on that side to which the patient has been inclining.

**TREATMENT.**—Passive hyperæmia of the lungs being never the primary affection, and always being a sign of failing power, its treatment consists in vigorously supporting the patient by alcoholic stimulants frequently administered, and with nutritious food. Nutritive enemata are often of great value. In all exhausting diseases this condition should be anticipated, and warded off if possible by timely support and stimulants, and above all by frequently turning the patient from one side to the other, thus calling in the aid, rather than permitting the hindrance, of gravitation to the circulation through the lungs. Of medicines, ammonia,

ether, bark, and quinine are of the greatest value; and musk, sumbul, and lavender may be useful adjuncts.

### C. Obstructive Hyperæmia of the Lungs.—SYNON. : Mechanical Congestion.

**DEFINITION.**—Hyperæmia from obstruction to the escape of blood from the lungs. Mechanical congestion of the lungs is a condition differing essentially from either of the two preceding.

**ÆTIOLOGY.**—The origin of this form of hyperæmia is purely secondary and mechanical, and is included in that of the primary disease. The obstruction may be at the mitral valve, as in mitral stenosis or regurgitation; or, again, the obstruction may be at the left ventricle, when this cavity is dilated and imperfectly emptied, as in the advanced stages of constrictive or regurgitant aortic disease. Whether there be an absolute narrowing of the blood-channel between the pulmonary and systemic circulations—that is, at the mitral orifice or at the commencement of the aorta; or whether, from enlargement of the mitral orifice or from disease or injury of its valve, regurgitation be permitted, so that each contraction of the right ventricle is met and opposed, more or less, by a counter rush of blood from the left ventricle—in any case, and still more in the combination of two or more of these causes, it is clear that the pulmonary circulation can only go on at an increased pressure by the contraction of the right ventricle becoming more vigorous; and that hyperæmia must result from the damming back of the blood through the pulmonary veins.

Of the causes named, mitral constriction is that which leads most simply to obstructive hyperæmia of the lungs.

**ANATOMICAL CHARACTERS.**—It is obvious that obstructive hyperæmia is of general distribution, affecting the whole of both lungs. The result of the heightened blood-pressure from increased force of injection into the lungs, to overcome an impediment to the escape of blood from them, is most felt in the pulmonary capillaries. These capillaries gradually become lengthened, tortuous, and dilated even to three times their normal dimensions (Rindfleisch). From chronic engorgement of the lungs, the nuclei of the interlobular areolar tissue and of the connective tissue surrounding the minute vessels and bronchi, and pervading the parenchyma, proliferate. The thickened and tortuous capillaries intrude upon the air-spaces; and, the elasticity of the lungs being also impaired, their vital capacity is diminished. It is stated that the muscular fibres, which proceed from the bronchial terminations to form loops upon and encircle the infundibula, become hypertrophied (Rindfleisch), thus, perhaps, compensating for a diminution in the more mechanical elastic property of the lung. Sometimes minute hæmorrhages take place

into the parenchyma of the lung; sometimes larger escapes of blood fill the alveoli of circumscribed patches (pulmonary apoplexy). The total result of the intimate changes described is an uniform increase in the size and weight of the lungs, with an increased density and toughness. On section, the lungs are found to be more pigmented and solid-looking than natural, sometimes of a brownish hue (brown induration); they are, however, crepitant throughout, except here and there, where they may present the firm, dark, damson-cheese-like section, fading to brown-red, of recent pulmonary apoplexy. There may be some œdema present. The pulmonary arteries and veins are enlarged and congested; and the bronchial mucous membrane is usually the seat of chronic catarrh. Patches of atheroma are frequently to be seen in the larger branches of the pulmonary artery.

**SYMPTOMS.**—Dyspnoea and cough, both brought on or increased by effort, with palpitation, and oppression or tightness, usually referred to the epigastrium, are the most constant symptoms of obstructive hyperæmia of the lungs. Patients suffering from this condition have repeated attacks of bronchial catarrh, and hæmoptysis is of common occurrence. The hæmoptysis may be considerable, but more usually the expectoration is streaked with blood or contains small dark coagula.

This form of hyperæmia commonly occurs before middle life, during the usual period of mitral heart-disease. The signs of heart-disease, and most often of constriction of the mitral valve, are present. The subjects of this affection are often undersized and badly nourished; the pigeon-breasted type of chest being common, especially in those cases in which the disease manifests itself early in life. Small, frequent pulse; more or less lividity of lips; and other signs and symptoms of the cardiac disease, of which the pulmonary condition is the consequence, are to be observed. A fine inspiratory crepitant *râle* over the lungs may be heard. During the repeated bronchial catarrhs, with increased pulmonary hyperæmia, to which such patients are especially prone, all symptoms are much aggravated.

**DIAGNOSIS.**—The existence of obstructive heart-disease suggests the presence of corresponding hyperæmia of the lungs. The fine crepitant *râle* and the hæmorrhagic symptoms and signs more positively point in the same direction.

**PROGNOSIS.**—The prognosis rests chiefly upon the heart-condition present. Increasing frequency of catarrhal complications, and especially of hæmoptysis, shows the turning of the balance against the patient. The condition may in favourable cases continue for years, especially when dependent upon simple constriction of the mitral valve.

**TREATMENT.**—The treatment is essentially that of the heart-disease; with the avoidance of all causes which quicken respiration, and which tend to produce catarrhs, to which these patients are so especially prone.

R. DOUGLAS POWELL.

### LUNGS, Hypertrophy of.

**DEFINITION.**—Enlargement of the lungs with increased functional power. A condition only clinically met with as a compensatory affection of one lung, or of a portion of one lung, to make up for more or less loss of pulmonary tissue by disease.

**ÆTIOLOGY AND PATHOLOGY.**—The process by which hypertrophy of the lung comes about is almost a mechanical one. Destructive disease having removed a large portion of one lung, or permanent collapse or blocking of such portion having occurred from any cause, the diminished power of the affected lung to occupy its apportioned space in the chest-cavity during inspiration, is compensated for by an increased expansion of the healthy lung, together with a certain recession of the softer parts of the chest-wall on the affected side. The increased expansion of the healthy lung encourages an increased afflux of blood, and an increased current of blood is at the same time propelled through it, owing to the partial obstruction to circulation through the diseased lung. These changes taking place gradually, the increased function and increased afflux of blood are, in accordance with our experience of similar conditions in other parts, attended with increased nutrition of the lung, and a true hypertrophy of it is thus little by little established.

Hypertrophy of the lung, therefore, is not a diseased condition, but an excessive development in consequence of injury to some other portion of the same or of the opposite lung. Its occurrence may be best noted during the gradual absorption or removal of a pleural effusion, which has long compressed the opposite lung. Any disease, however, which, after destroying or placing in abeyance a certain proportion of one lung, becomes arrested, tends to cause hypertrophous development of the remaining lung. Chronic pneumonia, cirrhosis, arrested phthisis, and atelectasis in the child, are the chief diseases of the kind. While disease is actively progressive, the conditions present—namely, fever, anorexia, and pleuritic pains—are unfavourable to the extra-development of the sound lung; but when these unfavourable conditions subside, the hypertrophy proceeds with great rapidity, and may be established in a very few months. The effect of the rarefied air of mountainous regions is to develop the natural capacity and function of the lungs.

**ANATOMICAL CHARACTERS.**—An hypertrophied lung is larger and heavier than natural; and its anterior and inferior margins are

thick and rounded, and are found to extend beyond their normal thoracic limits both laterally and inferiorly. The texture of the lung is firmer and more resilient than usual; and it is plentifully supplied with blood. The air-cells are slightly enlarged, but not obviously dilated. On microscopic examination the nutrition of the alveolar walls and capillaries is found to be perfect; there being neither the thinning of the alveoli, nor the excessive growth of fibrous tissue met with at different stages of so-called 'hypertrophous emphysema.' Nor are the capillaries tortuous and dilated, as in the indurative 'hypertrophy' of the lung from heart-disease.

**Extent of lung affected.**—We have considered, for the sake of simplicity of description, those cases in which the whole of one lung is hypertrophied, and such cases are very common. But a single lobe of a lung may become hypertrophied, the seat of the hypertrophy depending upon the seat of the disease to which it is compensatory. However, unless the pulmonary destruction is limited and circumscribed, other conditions come into play, and we are more likely to get emphysema than hypertrophy. It may be said then that hypertrophy of the lung is (at least so far as we can appreciate it clinically) a one-sided affection, except in those cases in which it has been occasioned by some general external cause, as rarefied air.

**PHYSICAL SIGNS.**—The side on which is the hypertrophied lung is expanded, both relatively (the opposite side being usually flattened and contracted) and absolutely. The nipple-level is raised. There is increased percussion-resonance over the side, extending across the median line, so that sometimes the line of resonance indicating the inner margin of the enlarged lung will reach the mid-clavicular vertical line of the opposite side. The lower limits of resonance are also extended in front and behind. The respiratory murmur has that peculiar coarse vesicular character, with somewhat prolonged expiration, which is known as 'puerile' or 'exaggerated' respiration. The heart is more or less displaced towards the contracted side, and the displacement is often apparently increased by the heart becoming on the one side covered by the expanded lung, and on the other side unduly exposed by the recession of the diseased lung. This is especially the case when the hypertrophy affects the left lung. There are no morbid sounds heard over the enlarged lung unless (as in many cases of phthisis) it becomes or has been affected by disease. With the expansion of the lung the general symptoms improve, and the dyspnoea lessens.

**DIAGNOSIS.**—The diagnosis must be made between hypertrophy of the lung; hypertrophous emphysema; and mere dilatation. The unilateral nature of the hypertrophy; its arising secondarily to some disablement of

the opposite lung; the absence of preceding or present general bronchitis or asthma; together with the observation of its occurrence being commensurate with improvement in the condition of the patient, are the main features distinguishing it from large-lunged or hypertrophous emphysema. Nor could the puerile breathing of hypertrophy be easily confounded with the short, weak, or inaudible inspiration and wheezy prolonged expiration of emphysema. In persons of broken-down constitution, with contractile disease of one lung, the opposite lung may become dilated, and assume the shape and dimensions of hypertrophy; but the breathing of the patient does not improve, the respiratory sounds are enfeebled, and it is clear that the condition present is one of emphysema rather than true hypertrophy.

**PROGNOSIS.**—The prognosis is always *pro tanto* favourable to the patient, the hypertrophy being an important element of his recovery.

**TREATMENT.**—Compensatory hypertrophy of the lung is a condition carefully to be encouraged, when all active symptoms attendant upon the original disease are past. Mild courses of calisthenics, and a temporary sojourn at some elevated health-resort during the warm season, are most valuable, if not attempted too soon. Abundance of fresh air throughout the year, with the careful avoidance of fresh catarrhs, such as may be obtained by spending a winter and spring or two seasons in the South of France, in Italy, or at Madeira; or a well-planned sea-voyage to Australia, are the most useful requisites during the months of convalescence. A generous unstimulating diet is indicated, and tonic remedies and cod-liver oil are useful, more especially in the early stages of the wished-for hypertrophic development.

R. DOUGLAS POWELL.

**LUNGS, Induration of.**—*See* LUNGS, Inflammation of.

**LUNGS, Infarction of.**—*See* LUNGS, Hæmorrhage into.

**LUNGS, Infiltrations of.**—Certain morbid formations in the lung assume the arrangement of an infiltration, the tissues, especially the interlobular cellular tissue, being permeated with the morbid material. In some instances it involves even the epithelial cells. The best examples of this mode of distribution are observed in connexion with certain cases of pulmonary fibrosis; in infiltrated cancer; and in those forms of disease where the lung-tissue is the seat of a deposit of substances introduced from without, being inhaled in various occupations, such as carbonaceous matter and coal-dust, stone-grit, iron-filings, particles of cotton or wool, and other materials. Albuminoid dis-

ease, and some forms of pigmentary change, also present a kind of infiltrated arrangement. These conditions need not be further considered here, as they are discussed in their several appropriate articles.

FREDERICK T. ROBERTS.

**LUNGS, Inflammation of.**—**SYNON.:** Pneumonia; Fr. *Pneumonie*; Ger. *Lungenentzündung*.

**DEFINITION.**—The term 'pneumonia' has been employed simply to designate inflammation of the lung-tissue. Inflammatory processes in the lungs, however, occur under such diverse circumstances, and are accompanied by such diverse clinical phenomena and histological changes, that 'pneumonia' used in this sense includes widely different diseases.

**VARIETIES.**—Pneumonias are divisible into the following varieties: A. **Acute Pneumonia**; B. **Secondary Pneumonia**; C. **Broncho-, Catarrhal, or Lobular Pneumonia**; and, D. **Chronic or Interstitial Pneumonia**. In addition to these there are those intense and concentrated forms of pulmonary inflammation which lead to the formation of *abscess*.

There are certain other forms of lung-consolidation which have sometimes been described as pneumonic, but which are really, for the most part, non-inflammatory in their nature, and will, therefore, be only briefly alluded to in the present article. These are: (1) that condition of collapse and hyperæmia, mainly due to weak inspiratory power, feeble circulation, and gravitation, which is so common in the more dependent portions of the lungs in many acute and chronic diseases. (2) Consolidations of the lung resulting from mechanical congestion and embolism, such as are met with in certain diseases of the heart.

The several varieties of pulmonary inflammation must now be considered separately, in the order just indicated.

Pulmonary abscess is separately described. *See* LUNG, Abscess of.

**A. Acute Pneumonia.**—**SYNON.:** Fr. *Pneumonie Aiguë*; Ger. *Croupöse Pneumonie*.—This is pneumonia *par excellence*. It is the disease to which the writer would be inclined to restrict the application of the term. It is sometimes termed *croupous pneumonia*, from the supposed resemblance of the histological process to that of croup. It is also known as *lobar pneumonia*, inasmuch as a large area of the lung is usually involved in the inflammation.

**DEFINITION.**—Pneumonia may be described generally as an acute disease, characterised clinically by sudden onset, severe febrile symptoms, cough, expectoration, and dyspnoea; by the physical signs of pulmonary consolidation; and by a rapid abatement of

the general symptoms between the fourth and tenth days. Anatomically it is characterised by an acute inflammation of the lung-tissue, and by the accumulation of the inflammatory products within the alveoli, which products consist, in the main, of a fibrinous exudation and leucocytes.

**ETIOLOGY.**—*Age.*—No age is exempt from pneumonia. It is met with between the ages of one and five years. Here, however, it is liable to be confounded with broncho-pneumonia and with collapse of the lung, so that the results of statistics are less reliable at this than in the subsequent periods of life. It may be stated, notwithstanding, that pneumonia is less common during infancy than has been generally supposed, and that amongst the pneumonias which are so frequent during this period of life the broncho-catarrhal forms preponderate. After the age of five years the liability to pneumonia diminishes, but it again becomes exceedingly frequent between the ages of twenty and forty, during which period the liability to the disease reaches its maximum. It is also quite common in old age.

*Sex.*—In adults more males than females suffer. This is probably owing to the former being more exposed to atmospheric influences. In early life this difference does not obtain.

*Social position, &c.*—Pneumonia is more common amongst the poor and badly fed, and amongst those whose occupation necessitates an irregular mode of life and great exposure, than amongst the upper classes of society.

*Constitution, and health.*—Those who are constitutionally weak, and those whose vitality has been impaired by intemperance, insufficient food, anxiety, overstrain, or other causes, are more prone to the disease than the strong and vigorous.

*Climatic influences.*—Conditions of weather and climate are probably the most important of all known agencies in the causation of pneumonia, and 'catching cold' is the most common exciting cause of the disease. The influence of cold and damp in increasing the liability to acute inflammatory diseases of the chest is well known. This influence is marked in pneumonia, although to a much less extent than in bronchitis and pleurisy. Pneumonia is more common in temperate climates than in those regions which are characterised by great heat or extreme cold. Climates and seasons which are liable to sudden changes of temperature, and winds from the north and north-east, appear to be especially favourable to the development of this disease.

*Epidemic influences and contagion.*—Pneumonia sometimes prevails epidemically; it is occasionally endemic in a house; and under certain circumstances, at present unknown, evidence tends to show that it may be communicated from one person to another. Such facts tend to support the view—the most probable in the present position of our

knowledge—that some micro-organism is concerned in the causation of the disease.

*Septic causes.*—Sewer-gas emanations appear to play some part in the causation of certain cases of pneumonia. Such cases are said to differ somewhat in type from ordinary pneumonia, and have been termed *pythogenic*.

*Previous diseases.*—Pneumonia, as is well known, often occurs in those who are the subjects of other disease. It is impossible to speak with certainty as to the relation which subsists between the pneumonia and the disease in the course of which it supervenes. In some cases it may be merely an accidental complication; whilst in others the previous disease may exercise more or less influence in the causation of the pneumonia. Most of the pulmonary consolidations, however, which occur in the course of other diseases do not belong to the category of true pneumonia, but are either local inflammations, caused by some abnormal state which the pre-existing disease has induced, or conditions of hyperæmia and collapse, in which an inflammatory process plays but little part.

*Exciting causes.*—In many cases of acute pneumonia evidence of the existence of any exciting cause is entirely wanting. Of discoverable causes, that which is most common is a sudden chill, or, less frequently, more prolonged exposure to cold and damp. Excluding cold, no conditions can be mentioned which have any marked influence in determining the disease.

**ANATOMICAL CHARACTERS.**—The changes occurring in the lungs in acute pneumonia are commonly described as consisting of three stages:—

1. *Stage of engorgement.*—This is the stage of inflammatory hyperæmia and œdema, and it is characterised microscopically by overfulness and slight tortuosity of the pulmonary capillaries, and by swelling of the alveolar epithelium. The lung is of a dark red colour; it is heavier and less crepitant than natural; it pits on pressure; and its cut surface yields a reddish, frothy, tenacious liquid.

2. *Red hepatisation.*—Here there is an exudation of liquor sanguinis and blood-corpuscles. The exuded liquids coagulate within the alveoli and terminal bronchioles, the coagulum enclosing numerous white and a few red blood-corpuscles. The alveolar epithelium is swollen and granular. It is stated by some German pathologists that the coagulum is in part produced by certain changes in the epithelium like those believed to occur in croup. The lung is now much heavier than in the preceding stage, and is increased in size, so as to be often marked by the ribs. It is quite solid; sinks in water; and cannot be artificially inflated. It is remarkably friable, breaking down with a soft granular fracture. The cut surface has a markedly granular appearance, seen especially when

the tissue is torn, and due to the plugs of coagulated exudation-matter which fill the alveoli. The colour is of a dark reddish-brown, often here and there passing into grey. This admixture with grey sometimes gives a marbled appearance. The pleura covering the solid lung always participates more or less in the inflammatory process. It is opaque, hyperæmic, and coated with lymph.

3. *Grey hepatisation*.—This stage is characterised by a continuance of the process of inflammatory cell-emigration, and by more marked changes in the epithelium. The white blood-corpuscles continue to escape from the vessels, and the alveolar epithelium becomes more swollen and granular. The alveoli thus become more completely filled with young cell-forms, so that the fibrinous exudation is no longer visible as an independent material. Many of these cells, especially those in the vicinity of the alveolar walls, are larger than leucocytes, and nucleated. These are evidently the alveolar epithelium. The fibrinous exudation now disintegrates, and the young cells rapidly undergo fatty metamorphosis. The alveolar walls themselves, with few exceptions, remain throughout the process unaltered; although very occasionally, when this stage is unusually advanced, they may be found here and there partially destroyed. Owing to these changes, the reddish-brown colour of the lung becomes altered to a greyish or yellowish white. The granular appearance is much less marked; the solid tissue is much softer and more pulpy in consistence; and a puriform liquid exudes from the cut surface of the organ. This stage, when advanced, has been termed 'suppuration or purulent infiltration' of the lung.

Although these three stages of the pneumonic process have been described as succeeding one another in orderly succession, it must be remembered that each stage does not occur simultaneously throughout the whole of the affected area of the lung. The changes advance irregularly, so that whilst one portion of the lung is in the stage of red hepatisation, another may be in the grey stage—hence the mottled, marbled appearance of the consolidation. The rapidity with which the several stages succeed one another is also subject to marked variations. In some cases the pneumonic consolidation very rapidly becomes grey, whilst in others the time occupied in the transition is much longer. These differences will be again alluded to when considering the clinical history of the disease.

*Terminations*.—The natural and almost invariable termination of the histological process is in *resolution*—the lung gradually returning to its normal condition. This is effected by the fatty and mucoid degeneration, and consequent liquefaction, of the inflammatory products which have accumulated within the alveoli. As the liquefaction

proceeds, the circulation in the alveolar walls is gradually restored; the softened products are removed by absorption, and to a much less extent by expectoration; and the lung ultimately regains its normal characters. The other exceptional modes of termination in *gangrene*, *abscess*, and *chronic pneumonia* will be alluded to subsequently.

*Site*.—The local lesion in pneumonia is in the majority of cases limited to one lung. When double, one lung is usually involved before the other. The right lung is more commonly affected than the left. The part of the lung usually involved is the lower lobe (about 75 per cent.). The consolidation may extend upwards and implicate the whole lung. Pneumonia of the upper lobes is more frequently double than basic disease. It is quite rare for the pneumonic process to commence in two different portions of the lung. When the consolidation is met with in both lungs, or commencing in the upper and middle lobes, the pneumonia is often a secondary affection, and has supervened in one whose health has been previously injured, as by alcohol; and such distributions of the local lesion should always make the physician look carefully for evidence of pre-existing disease.

*PATHOLOGY*.—The old view that pneumonia is a simple local inflammation accompanied by a symptomatic pyrexia, would appear to be no longer tenable; and although a complete pathology of the disease must await further investigations, the present position of our knowledge makes it in the highest degree probable that it is a *general infective disease*, closely allied to the acute specific fevers, of which the lung-inflammation is the characteristic local lesion. What is known of the ætiology of the disease, the absence of any constant relation between the local inflammation and the intensity of the fever, the 'typical' course of the fever, the fact that the disease is incapable of being produced by mechanical or chemical injuries of the lung, and, lastly, the discovery of peculiar micro-organisms, all tend to support this view of its pathology.

*Micro-organisms*.—During recent years certain organisms have been described by various observers in connexion with pneumonia, which are supposed to be the causes of the disease. Friedländer demonstrated the almost constant presence in the exudation and lymphatics of the lung of oval, short rod-shaped cocci, contained in oval capsules, from two to four or more in each capsule. Subsequently Fränkel and Weichselbaum described the presence in pneumonic lungs of another 'capsule-coccus,' the *diplococcus pneumonicæ*. This consists of round or oval cells, usually in pairs, but often in chains. This coccus is believed by Baumgarten to be constantly present in the pneumonic lung, and it is also found in the blood. Whether

either of these organisms is to be regarded as a cause of the disease, or only as an accidental parasite, is at present quite uncertain, the results of cultivation and inoculation being thus far inconclusive. But, as already stated, the present position of our knowledge points to the probability of some specific organism or organisms being concerned in the causation of the disease. See MICRO-ORGANISMS.

**SYMPTOMS.**—The onset of pneumonia is in the majority of cases sudden, not being accompanied by any premonitory symptoms. Much less frequently, certain premonitory symptoms precede the more severe phenomena which characterise the invasion of the disease. These symptoms include general malaise, headache, chilliness, pains in the back, and loss of appetite.

**Invasion.**—The invasion in adults is, in most cases, announced by a rigor, much less frequently by vomiting. This rigor is more marked in pneumonia than in almost any other disease. It is usually single, and is rarely repeated, either at the commencement or in the course of the illness. In old subjects the rigor is very frequently absent, and in children its place is often taken by convulsions or vomiting. The rigors or other phenomena marking the invasion of the disease, together with the attendant pyrexia, are usually quickly followed by symptoms pointing to the lung-affection. These symptoms commonly supervene in the course of from twelve to twenty-four hours, although in exceptional cases not until after the lapse of two or three days. The earliest of them are pain in the side, dyspnoea, and cough. These more local symptoms, together with the pyrexia, acceleration of pulse, thirst, and prostration, gradually develop up to the second day of the disease, by which time (and sometimes before this) the pulmonary lesion is usually sufficiently advanced to yield unequivocal physical signs. The general aspect and symptoms of the patient are now tolerably characteristic. The flushed and sometimes dusky face, anxious expression, hurried breathing, hot skin, rapid pulse, short frequent cough, and marked prostration, supervening quickly upon the well-marked initial rigor, indicate pretty clearly the nature of the disease.

**Pain.**—The pain in the side, which is increased by deep inspiration and by cough, usually corresponds in situation with that of the affected lung, although it is occasionally experienced in other parts. This symptom may occur coincidentally with the rigor, although it more commonly succeeds it. In quite exceptional cases it precedes it, being the first symptom noticed. The pain is due to the implication of the pleura in the inflammatory process; and its severity consequently varies considerably according to the extent and situation of the pulmonary lesion. When this is confined to the central por-

tions of the lung, pain may be entirely absent.

**Respiratory phenomena.**—Increased frequency of respiration, dyspnoea, and cough, are early and prominent symptoms. The respiration—usually regular—ranges from 30 to 50, and in children reaches even to 70, whilst the pulse may be only from 90 to 120. This perversion in the pulse-respiration ratio is important in diagnosis. The breathing is shallow; inspiration is abrupt; and, when the pain in the side is severe, respiration is sometimes irregular. The accelerated respiration is accompanied by marked expansion of the *ala nasi*, and by more or less dyspnoea. There is, however, no definite relation between the last-named symptom and the frequency of the respiratory act. Owing to the pain, and to the frequency and difficulty of breathing, speech may be interfered with, and is often rendered exceedingly difficult. Cough is an almost constant symptom, except in the very old. It is short and hacking, rarely paroxysmal like that of bronchitis. It is usually in the early stages attended with severe pain in the side, so that the patient endeavours to repress it. The cough is usually attended by expectoration; but in children and the old expectoration is often absent, and occasionally such is the case at other periods of life, even throughout the whole course of the illness. The sputa of pneumonia are very characteristic. They are viscid, glairy, and remarkably tenacious, so that they cling to the mouth of the patient, and adhere closely to the sides of the vessel containing them. In colour, they present various shades of red, brown, and yellow, owing to the admixture of blood. The appearance so well-known as ‘rusty’ is that most commonly met with. Sometimes they are much more diffluent, and of a dark purple colour, somewhat resembling prune-juice. The characteristic sputa are usually met with on the first or second day of the disease, but their appearance is often preceded by a frothy aerated expectoration, like that of bronchitis. The amount expectorated is small, and sometimes the pneumonic is associated with more or less of the frothy catarrhal sputa throughout the whole of the disease. During the period of resolution the sputa become less viscid, and more catarrhal in character, and they usually contain small particles of black pigment. The histological elements met with in the sputa are leucocytes, red blood-cells, and altered epithelium from the alveoli and air-passages; and towards the decline of the disease, fat-granules, pigment, and occasionally fibrinous masses, which are casts of the alveoli and terminal bronchioles.

**Pulse.**—The pulse in adults usually ranges from 90 to 120, and may be even more frequent. It is commonly much more rapid in children, and less so in the old. In the early stage of the disease it is often full

and strong, but soon becomes smaller and easily compressible. It may be irregular, intermittent, or dicrotous. The smallness of the pulse is probably due partly to diminished cardiac power, and partly to the diminished amount of blood which is propelled from the left ventricle, owing to the overloading of the right cardiac cavities which results from the obstructed circulation in the lungs.

*Pyrexia.*—The pyrexia of pneumonia is continuous, with slight morning remissions and evening exacerbations. The temperature rises very suddenly, with the invasion of the disease, to from 102° to 105° F.; and this high temperature is usually maintained until the period of crisis. This sudden rise and maintenance of a high temperature is very characteristic. The amount of elevation varies in different cases. As a rule, it does not exceed about 104° or 105° F., but temperatures of 107° have been known to terminate favourably. In fatal cases it may reach 109° shortly before death. The maximum temperature is usually met with on the second or third day of the disease, but it occasionally occurs immediately before the crisis. The daily variations are usually as follows: The temperature is lowest about 7 or 8 A.M. In the forenoon, or somewhat later, it commences to rise, and attains its maximum in the early evening. It then falls, but a slight exacerbation occasionally occurs again at midnight, after which it gradually falls. The difference between the highest and lowest temperatures is usually not more than 1° F. The pyrexia runs, for the most part, a uniform course until the period of crisis, when the temperature falls rapidly, in the manner to be hereafter described; but in exceptional cases the course of the fever is much more irregular.

*Nervous system.*—Headache, restlessness, and sleeplessness are almost always prominent symptoms in pneumonia. Slight delirium is also common, especially towards evening, when the pyrexia is at its maximum. Sometimes the delirium is more marked and violent. It constitutes a more prominent symptom in the old, and in the debilitated and intemperate. In drunkards it is constantly present, and here it often assumes the character of delirium tremens. Convulsions are common in children, especially at the period of invasion. They are rare in the adult. These nervous symptoms are sometimes so prominent as to mask the nature of the disease.

*Digestive organs.*—The symptoms of pneumonia referable to the digestive system are similar to those met with in other severe febrile diseases. There is thirst, with loss of appetite. The tongue is more or less thickly coated with a white fur, and it tends in severe cases to become dry and brown. Herpes usually makes its appearance about the lips,

and sometimes on other parts, from the second to the fourth day of the disease. Vomiting, which is a common symptom of invasion, is an occasional complication, as is also diarrhoea; constipation, however, is the rule. Sometimes there is slight jaundice.

*Urine.*—The quantity of urine is considerably diminished, and its specific gravity increased, so that abundant urates are deposited. The excretion of urea is greatly increased, and it may amount to as much as seventy-five grammes in the twenty-four hours. The uric acid is likewise augmented. The chlorides are much diminished, and during the height of the disease may entirely disappear. Slight temporary albuminuria is perhaps more common in pneumonia than in almost any other acute febrile affection. The amount is usually in direct proportion to the severity of the disease. Bile-pigment is occasionally met with.

*PHYSICAL SIGNS.*—The earliest physical signs of pneumonia are usually discoverable within forty-eight hours of the invasion of the disease. They often appear within twelve or twenty-four hours; but occasionally, when the local lesion is deeply seated, nothing abnormal is to be detected until the third or fourth day. It will be well to describe them in the order in which they commonly make their appearance. The time occupied in their evolution will vary according to the rapidity with which the several stages of the pneumonic process succeed one another; and it must be remembered that only some of them are usually observed in practice. In a large number of cases, bronchial breathing and some impaired resonance (the two signs most frequently met with) are the only abnormal signs discoverable.

The earliest abnormal physical signs are due to the pain caused by the movement of the affected side; to the hyperæmia of the pulmonary capillaries; and to the commencing exudation into the air-vesicles. The respiratory movements of the side are more or less impaired. This is partly owing to pain, and partly to diminished elasticity of the lung-tissue. The breath-sounds are usually somewhat weak and harsh, but not distant; although, as stated by Stokes, they are occasionally in the earliest stage harsher and louder than natural. Percussion during this stage is usually not markedly altered. The resonance, however, in the upper portions of the chest is sometimes appreciably tympanitic in quality. The vocal fremitus is increased. The most important sign, however, of the congestive stage is *fine crepitation*. This *râle* consists of a number of fine, dry, crackling sounds, following one another in rapid succession, which were aptly compared by the late Dr. C. J. B. Williams to the sounds produced by rubbing the hair between the fingers close to the ear. It occurs during the later period of this stage, when the process of exudation from the pulmonary capillaries is

commencing to take place. Its production is probably due to the partial agglutination of the walls of the air-vesicles, and their forcible separation during the inspiratory act.<sup>1</sup> The *râle* is almost exclusively limited to inspiration. It is intensified by deep inspiration and also by cough, and it is sometimes necessary to make the patient cough in order that it may be produced. A precisely similar *râle* is often heard with deep inspiration in portions of the lung which have been imperfectly expanded. Such imperfect expansion is common in the posterior parts of the lungs of patients who have been confined to bed from acute or chronic disease, and in whom, owing to muscular weakness, inspiration is incompletely performed. The *râle* produced under these circumstances is distinguished from pneumonic crepitation inasmuch as it completely disappears after a few deep inspirations; whereas the pneumonic *râle*, when once established, persists until the consolidation of the lung is tolerably complete.

The physical signs of the stage of hepatization are due to the more or less complete consolidation of the lung. The fine crepitation which characterised the later periods of the preceding stage continues during the process of consolidation, but ceases as the filling of the air-vesicles becomes complete; although it may often still be heard at the confines of the more firmly consolidated lung. It occasionally happens, however, when the consolidation is very rapidly induced, that no crepitation is heard throughout the course of the disease until the period of resolution. The situation of the cardiac impulse is not altered. The vocal fremitus is usually increased. To this general rule, however, there are exceptions, and it not infrequently happens that it is unaltered, and it may even be completely absent. This diminution in the vocal fremitus is sometimes due to the blocking of the smaller bronchi with the inflammatory exudation, but more frequently it appears to result from an accumulation of mucus. In the latter case it may sometimes be restored by cough. Coincidentally with the increase of vocal fremitus there is usually increased vocal resonance, and sometimes whispering pectoriloquy (Walshe and Wilson Fox). The percussion-sound now is much more deficient in tone, and it is often more or less amphoric. There is also a great increase in the sense of resistance, but neither the dullness nor the resistance is so marked as in pleural effusion. In basic disease, percussion under the clavicle often yields a distinctly amphoric note, whilst the lower portions of

the chest may be almost absolutely dull. The auscultatory sign of this stage is bronchial breathing. This is usually remarkably superficial, high-pitched, and metallic in quality (tubular breathing). Sometimes, however, it is less metallic and softer (diffused blowing—Walshe). These respiratory phenomena, like the vocal resonance and fremitus, may be absent over larger or smaller areas of the consolidated lung, owing to the obstruction of the bronchi by catarrhal secretion. The slight pleurisy which constantly accompanies the pneumonia is rarely susceptible of physical demonstration during this stage. This is probably owing to the immobility of the solid lung. During the period of resolution, as expansive power returns, friction-sounds are occasionally audible.

Resolution usually commences in those portions of the lung which were the last to become consolidated. The most important and the earliest of the signs of resolution is the return of crepitation. The crepitation, however, differs from that met with in the earlier stages of the disease. It is larger, coarser, and more liquid in character—*redux crepitation*; and its liquid character gradually increases until it may become distinctly bubbling. When resolution is very rapid, *redux crepitation* may be absent (Wilson Fox). The bronchial breathing now loses its metallic ringing quality; the percussion-dullness gradually disappears; and the respiration regains to a great extent its normal characters.

The commencement of resolution and of the improvement in the physical signs occasionally takes place coincidentally with the establishment of crisis; but more commonly it is not observed until from twenty-four to forty-eight hours after the temperature has reached the normal standard. The time occupied in the completion of resolution varies. Sometimes all physical signs almost completely disappear in twenty-four hours. Usually, however, resolution is less rapid, and marked signs of consolidation remain for periods varying from two or three days to two weeks. A slight amount of dullness and some weakness of respiration often persist at the posterior and inferior portions of the lung for even still longer periods. This is especially the case if the pneumonia is complicated with pleurisy. When marked signs of consolidation exist after the third week, there always exists more or less probability that the pneumonic process may become chronic.

**COURSE AND TERMINATIONS.**—The symptoms which have been described continue with often increasing severity up to about the end of the first week, sometimes longer, when there is usually an abrupt subsidence of the pyrexia—*crisis*, and the disease generally terminates in *recovery*. In other cases *death* occurs either before or after the crisis. The

<sup>1</sup> This sound is well illustrated by that emitted in the crackling of bubbles in a viscid liquid such as beer poured into a glass. The sound may be explained by supposing that a thin layer of the viscid fluid is drawn across the orifice of the air-vesicle during inspiration to form an operculum, which is immediately ruptured.—EDITOR.

disease may also terminate in *gangrene* of the lung; in *pulmonary abscess*; or in *chronic pneumonia*. These several modes of termination must be considered separately.

*Complete recovery*.—This is the most common termination of pneumonia in young and healthy adults, and the improvement usually begins with the *crisis*. The time at which this crisis takes place, as indicated by the sudden fall of temperature, varies from the third to the twelfth day. In the majority of cases it is on the fifth, sixth, or seventh day; occasionally as early as the third day; and sometimes it is prolonged into the middle of the second week.

The supervention of the crisis in pneumonia is sometimes indicated by a change in the pulse, which becomes softer, and somewhat irregular in force and rhythm. The most marked phenomenon attending it is the abrupt fall of the body temperature. This fall usually begins late in the afternoon or evening; and the temperature very often reaches the normal standard in from sixteen to twenty-four hours, usually within forty-eight hours; the morning remission and evening exacerbation occurring during the period of defervescence. The temperature not infrequently falls 1° or 2° F. below normal, and may remain so for two or three days. Occasionally a marked increase in the pyrexia is observed immediately before the commencement of defervescence.

With the fall of temperature all the symptoms rapidly improve. The skin becomes moist and often perspires profusely. The amount of urine increases. The respiration falls in frequency; and, to a less extent, the pulse. The cough becomes looser, and the expectoration more copious. The sputa gradually lose their tenacity and rusty colour, and become more bronchitic in character; they are now usually mingled with more or less black pigment. An improvement in the physical signs is sometimes observed at the same time; more commonly, however, this does not take place till one or two days later. The patient often falls into a deep sleep, and on waking, with the exception of great weakness, declares himself pretty well, and begins to ask for food. In some cases, however, the amount of prostration following the crisis is so great that the return to health is more gradual; and a condition of collapse may ensue, which often terminates in death. There is a liability to diarrhoea at this period which it is all-important to bear in mind.

In the majority of cases pneumonia terminates abruptly in the manner above described. Sometimes, however, the temperature falls more gradually—by *lysis*; and recovery is more protracted. Defervescence may not be complete till the end of the second week. The critical fall of temperature is occasionally interrupted by more or less marked exacerbations, due either to the

implication of fresh portions of the lung, or to the supervention of one of the complications to be hereafter alluded to.

In some cases, after the occurrence of crisis, there is a slight return of fever of a hectic type. This irregular fever, which may last from three or four days to a fortnight, is probably due to the contamination of the blood by the absorbed pneumonic products (Parkes). Lastly, a distinct relapse may occur after the completion of crisis; but the relapse is in most cases shorter and less severe than the primary attack.

*Death*.—When pneumonia terminates fatally, it usually does so towards the end of the first, or quite at the beginning of the second week, and often *after* crisis. Death is commonly due mainly to failure of cardiac power, and partly to apnoea. Apnoea is the least important element in the causation of dissolution. The danger from it increases with the extent of lung involved, and it is consequently usually greater in double than in unilateral disease. Failure of cardiac power is undoubtedly the most important means by which pneumonia destroys life. There are several conditions in the disease which tend to damage the contractile power of the heart. First and foremost of these is that damage to the tissues generally which occurs in all acute febrile, and especially in acute infective, diseases. This damage is prominent in pneumonia, which, although a disease of short duration, is severe whilst it lasts. Then, again, the condition of the lung itself constitutes another element tending to damage the contractile power of the heart, and especially of the right ventricle (Jurgensen). The lung-consolidation not only presents more or less obstruction to the pulmonary circulation, and hence necessitates increased action on the part of the right ventricle; but, owing to the diminished respiratory surface, this ventricle is obliged to do more work in order that the proper interchange of gases may be effected in the lungs.

Such being the modes by which pneumonia tends to destroy life, it will be readily understood that the earliest and most important signs of unfavourable augury are on the side of the circulation. The pulse becomes more frequent, small, irregular, and often dicrotous. The frequency of the respiration, the dyspnoea, and the cyanosis increase. The cough becomes feeble and ineffectual. Owing to the engorgement and failure of power of the right ventricle, general pulmonary oedema usually supervenes, so that moist *râles* are audible at both bases. The extremities become cold, and there is often profuse perspiration. The mind wanders, and a condition of partial coma supervenes before the close. In some cases a rapid rise of temperature takes place before the fatal termination, whilst in others there may be

a considerable fall in the thermometer. In those exceptional cases in which pneumonia tends to become more or less chronic, death may occur during the third or fourth week. Death may also result from the complications.

*Gangrene.*—This is quite rare. It is most common in chronic drunkards, and in those of debilitated constitution. Its occurrence appears to be due partly to blocking of vessels, and partly to the influence of septic inflammatory products. It is usually limited to a small area of the pneumonic lung; and is either diffuse, or becomes limited by a zone of inflamed tissue. It commonly supervenes late in the disease; and the most reliable signs of its occurrence are marked fœtor of the breath and the expectoration, and great prostration. Portions of lung-tissue are occasionally found in the sputa. Gangrene is almost invariably, but not necessarily, fatal. See LUNGS, Gangrene of.

*Abscess.*—This is also very rare. See LUNGS, Abscess of.

*Chronic pneumonia.*—Acute pneumonia in very exceptional cases becomes chronic, and leads to induration of the lung.

VARIETIES.—The clinical phenomena of pneumonia are occasionally somewhat different from those which have been described. Variations are met with resulting from differences in the individual, and in the ætiological factors concerned in the disease. The following varieties must be alluded to:—

(a) *Latent pneumonia.*—Pneumonia has been termed *latent* when the characteristic symptoms of the disease are absent, or but little pronounced; or when they are masked by some other clinical phenomena. It is in the pneumonia of the aged that marked latency is so often observed. Here invasion may be unattended by rigor or other prominent symptoms. The elevation of temperature is much less than in adults. Cough, expectoration, pain, and dyspnoea may be completely wanting. If cough be present, the sputa often do not present the rusty tinge, but are simply transparent or mucopurulent. Slight pyrexia, with some increased frequency of breathing and prostration, may be the only symptoms present. This latent course of pneumonia it is important to bear in mind, as it indicates the necessity of making a most careful physical examination of the chest in all febrile illnesses of the aged. In the pneumonia of drunkards and of young children, also, the accompanying nervous phenomena may be so prominent as to mask the nature of the disease.

(b) *Asthenic (typhoid) pneumonia.*—When pneumonia occurs in those who are debilitated by previous disease, by the abuse of alcohol, by age, by privation, or other causes, the phenomena of invasion are usually not pronounced, and symptoms of intense prostration occur early. In many respects the

course of the disease closely resembles that which has been just described as so common in the aged. The initial rigor and pain in the side are often observed; but cough is slight; and the expectoration, instead of the rusty-brown tint, may present a dirty-brown or prune-juice appearance. Various symptoms of an asthenic type soon become prominent; the most important of which are low delirium, alternating with stupor; tremors; and paralysis of the sphincters. The tongue is brown and dry; sordes form on the teeth; the pulse is exceedingly rapid and feeble; and there are often slight jaundice and albuminuria. Death usually supervenes some time during the second week of the disease. After death the lung is usually found to be less firmly consolidated and less granular than in sthenic forms of pneumonia. The stage of grey hepatisation in some cases is exceedingly advanced, constituting what has been termed 'suppuration' of the lung (Sturges).

(c) *Intermittent pneumonia.*—An intermittent variety of pneumonia is sometimes met with in malarial districts, which appears to be one of the results of malarial infection. According to Grisolle, it occurs in intermittent and remittent forms. The former is characterised mainly by the complete intermissions which occur in the pyrexia. The temperature falls suddenly at the end of twenty-four hours; profuse sweating occurs; and the physical signs of the pneumonia almost entirely disappear. A return of the pyrexia and physical signs takes place at the expiration of twenty-four or forty-eight hours, followed by another intermission, and this by a third or fourth, the disease presenting either a quotidian or a tertian type. Both lungs are liable to be involved. In the remittent form there is a much less complete disappearance of the physical signs during the remission.

(d) *Pythogenic pneumonia.*—This term has been applied to certain cases of pneumonia, in which sewer-gas emanations appear to be concerned in the causation of the disease. Such cases are said to be characterised by a more prolonged and fluctuating pyrexia (Douglas Powell). Some of the cases thus described are probably pyæmic.

COMPLICATIONS.—*Pleurisy.*—Pleurisy of slight intensity, and unaccompanied by effusion, is, as already stated, almost invariably met with in pneumonia over those portions of the lung which are consolidated. This is natural to the disease, and cannot be regarded as a complication. Pleurisy of greater intensity, and attended by effusion, occurs in from 5 to 15 per cent. of the cases. Signs of effusion are not usually discoverable before the third or fourth day of the disease. The amount of liquid varies with the extent of the lung-consolidation. When this is considerable, involving nearly

the whole lung, there is but little room for effusion. The supervention of pleurisy does not commonly materially modify the course of the disease. It may, however, protract the period of defervescence. Its influence in interfering with the disappearance of the physical signs has been already alluded to. When pleurisy occurs on the side opposite to the pneumonia it constitutes a more dangerous complication.

*Bronchitis.*—This is also a common complication, especially in the aged and in young children. Many cases, however, which have been described as pneumonia, associated with bronchitis, have doubtless been cases of broncho-pneumonia. The bronchitis almost invariably affects both lungs. Its supervention is attended by an increase in the cough and in the amount of expectoration. When it involves the smaller tubes it constitutes a serious complication.

*Pericarditis.*—This is much less frequent. It may result from the direct extension of the inflammatory process from the pleura. When extensive, it is a grave complication, and greatly increases the mortality.

*Jaundice.*—A slight yellowish tinge of the conjunctiva, and even of the skin, is not infrequent in pneumonia, and has no clinical significance. It is probably in most cases owing to the congestion of the liver which results from the impeded pulmonary circulation—the distended portal veins pressing upon the bile-ducts. Much more extensive jaundice is also occasionally met with, which appears usually to be due to duodenal catarrh, and is attended by gastric symptoms. In other cases, especially in asthenic forms of the disease, a non-obstructive jaundice sometimes occurs, resulting from changes in the red blood-corpuscles. This is commonly associated with nervous symptoms, such as stupor, delirium, and a tendency to collapse, which are of grave prognostic import. See JAUNDICE.

*Parotitis.*—This is a very rare and exceedingly serious complication. According to Grisolle, it is usually unilateral; the inflammatory process is very acute; and commonly leads to suppuration or gangrene.

**DIAGNOSIS.**—The diagnosis of pneumonia, usually easy, is sometimes attended with difficulty. Although it cannot be made with absolute certainty prior to the appearance of physical signs of pulmonary consolidation, in the majority of cases a very confident opinion can be formed as to the nature of the illness in the earliest stages, before such signs are discoverable. The phenomena which are of the most diagnostic value at this early stage of the disease are the pyrexia, the altered pulse-respiration ratio, the pain in the side, and the cough. The sudden and rapid rise of the bodily temperature, which usually reaches its maximum in forty-eight hours, is very characteristic; such a rapid

attainment and maintenance of a high temperature being perhaps more common in pneumonia than in any other disease (Wilson Fox). Of the other symptoms, the increased frequency of respiration, and especially the alteration in the pulse-respiration ratio, are of the most valuable diagnostic import. When physical signs of pulmonary consolidation are discoverable, which they usually are within forty-eight hours, the diagnosis becomes certain.

Difficulties in diagnosis may arise in those cases in which the local process in its earlier stages is deeply seated. Here, characteristic physical signs may be wanting for four or five days, during which time some doubt may exist as to the nature of the disease. In the aged the disease so often runs a latent course, that its existence may be easily overlooked unless a careful examination be made of the chest. Then, again, in the intemperate its nature may be masked by delirium and other nervous symptoms.

The diagnosis of pneumonia from other diseases of the lungs is rarely difficult. The disease with which it is most liable to be confounded is pleurisy; and when a pleural effusion complicates pneumonia, some care may be required to avoid error. In pleurisy, however, there is not, as a rule, such a sudden and rapid attainment of a high temperature as in pneumonia; and when effusion has taken place, the physical signs are in most cases sufficiently distinctive. In pleural effusion the displacement of the heart; the absolute dulness and sense of resistance on percussion; the weak and distant character of the respiration; and the diminished vocal fremitus and resonance, contrast with the signs of pulmonary consolidation.

Another disease with which pneumonia may be confounded is that somewhat rare form of very acute phthisis, in which a large area of the lung becomes rapidly consolidated—the consolidation being often in the main indistinguishable histologically from that met with in pneumonia. Here, however, although the whole lung may be involved, the disease usually commences in the upper lobes, so that abnormal physical signs are more marked at the apex. The onset of the disease also is commonly much less sudden, and its course is more protracted. Then, again, the rapid consolidation of the lower portions of the lungs, which sometimes supervenes in more chronic forms of phthisis, might be mistaken for the consolidation of acute pneumonia; but the history of the case, a careful examination of the upper lobes of the lungs, the irregular course of the pyrexia, and the protracted duration of the disease, will easily serve to distinguish them. It may be stated generally that, in all acute consolidations of the lung, a protracted course of the pyrexia, and the occurrence of marked exacerbations and of remissions of the fever at irregular intervals,

afford grounds for the supposition that the consolidation is tuberculous.

The diagnosis of pneumonia from broncho-pneumonia and collapse of the lung will be considered when treating of broncho-pneumonia. See C. Broncho-Pneumonia.

**PROGNOSIS.**—The mortality from pneumonia varies at different periods, the disease being more fatal in some years than in others. This is probably partly owing to variations in what is called 'epidemic influence.'

Of all the circumstances which influence the prognosis of pneumonia, that which is perhaps the most important is the state of the general health. In those whose vitality has been damaged by previous disease, by privation, or by their mode of life, pneumonia is exceedingly dangerous. The prognosis is, however, especially grave in those injured by the long-continued abuse of alcohol, the mortality being, according to Huss, from 20 to 25 per cent.

Of equal importance is the age. In healthy children the mortality from pneumonia is comparatively small. The fatality formerly ascribed to the disease at this period of life was probably owing in great measure to the inclusion of cases of broncho-pneumonia in the statistics; the latter disease being exceedingly fatal. Healthy young adults rarely die; after the age of thirty the mortality increases considerably; and in the old pneumonia is an exceedingly fatal disease.

Pneumonia is more fatal in females than in males, the mortality being in the proportion of three to two. Pregnancy renders the disease more dangerous.

The danger of pneumonia increases somewhat with the extent of lung implicated. It is, however, the implication of both lungs which renders the prognosis especially grave. With regard to the situation of the consolidation it may, perhaps, be stated generally that pneumonias commencing in the upper lobes are rather more serious than basilar disease. The gradual extension of the consolidation late in the disease, and the spreading of the inflammatory process from one centre to another, constitute elements of gravity. When resolution is much protracted, the fact that the disease in very exceptional cases terminates in an indurative consolidation of the lung is not to be forgotten.

The mortality of pneumonia is greatly increased by the existence of complications. The prognostic importance of these has already been considered.

Of individual symptoms, the pulse is of chief importance. A pulse which in the adult is persistently over 120, and in the child over 140, is of grave significance. Marked irregularity in force and rhythm is also unfavourable, especially in the young. Dicrotism may occur temporarily in quite favourable cases, but if it persists it indicates danger. The variations in respiration are of less import.

An extreme quickness of breathing, marked dyspnoea, and cyanosis are not uncommon in cases which terminate in recovery; at the same time, such symptoms must have more or less unfavourable significance. Sputa of a dark prune-juice colour are of somewhat evil augury; as is also an abundant liquid puriform expectoration. The indications from the pyrexia are of less prognostic value in pneumonia than in most other acute diseases. A temperature of 105° or 106° F. does not in itself indicate danger. Greater elevation is grave. In many fatal cases the temperature never attains 102°. The significance of a protracted defervescence has already been alluded to. On the side of the nervous system, it is to be remembered that slight delirium is not uncommon; but when it is marked, and especially when it occurs late in the disease, it is most grave. It is of greater significance in adults than in children. Tremors and a tendency to coma are also unfavourable. A dry, brown tongue is unfavourable, especially when associated with only a moderate degree of pyrexia. Gastric catarrh and diarrhoea add to the danger; and the liability to diarrhoea at the period of crisis, and the frequency with which it then endangers life, should be ever remembered. A slight amount of jaundice is not of bad import. The pre-existence of albuminuria, or the appearance of albumen in the urine early in the disease, is very unfavourable.

**TREATMENT.**—In considering the treatment of pneumonia, it is of the utmost importance to bear in mind the true nature of the disease. All rational and successful therapeutics must be based upon the recognition of the fact that it is not simply a local affection of the lung which we wish to influence. The 'heroic' methods of treatment by venesection, tartar emetic, &c., so much in vogue in the past, had for their object the controlling or cutting short of a simple pulmonary inflammation; hence the unfavourable results which attended them. As these methods have been abandoned, and there has existed a more correct appreciation of the pathology of the disease, the mortality attending pneumonia has diminished.

When discussing the pathology of pneumonia, reasons were adduced for the belief that it owes its origin to a specific cause. Whether this be so or no, the disease is so closely allied to the specific fevers that, in attempting to influence its course by treatment, we must be guided by the same general principles. As in these fevers, our object must be to endeavour to conduct the pneumonia to a favourable termination. We cannot arrest its progress, but we can often do very much both to maintain the strength of the patient, and to modify those elements in the disease which tend to destroy life.

The modes by which pneumonia tends to destroy life have been already considered—

that damage to the tissues generally which obtains in all acute specific diseases, and interference with the respiratory function due to the lung-consolidation. Of these, the former is much the more important, and impaired cardiac power is its most serious consequence. The natural course of the disease is also to be remembered. In the strong and robust, pneumonia usually terminates in health. It is in those who are debilitated by age, privation, mode of life, abuse of alcohol, or pre-existing disease, that such great mortality attends it. It is a question of the intensity of the disease on the one hand, and of the resisting power of the individual on the other. Such considerations as these indicate the importance not only of doing all that is possible to husband and support the strength of the patient, but also of not interfering too actively with the disease, unless circumstances arise which, if uninfluenced by treatment, would tend to prove rapidly fatal.

Such being the general principles which should guide us in the treatment of acute pneumonia, the manner in which they are to be best carried out may now be indicated.

Everything should be done to husband the strength of the patient, and the services of an efficient nurse are all-important towards the attainment of this object. Perfect rest must be enjoined, and all unnecessary speaking be forbidden. The patient should be kept in bed. The room should be large and airy; and the temperature about 60° to 62° F. It should be well-ventilated: a plentiful supply of fresh air is most important, and, although due care should be exercised in the ventilation, there is not the same necessity to keep the patient scrupulously protected from draughts as in the treatment of acute bronchitis.

The diet should be carefully regulated, nutritious, and easily digestible, consisting of milk, milk with egg, beef-tea, mutton or chicken broth, meat-essences, and such-like articles, given in varying quantities and at varying intervals, according to the condition of the patient. With the object of stimulating the secretion of saliva and promoting the appetite, it is well to keep the mouth cleansed with glycerine and lemon-juice. Some acid and bitter drink, as one made with hydrochloric acid and orange-peel, may also be given.

If in the earliest stage of pneumonia there is great constipation, loaded tongue, nausea, or other gastric symptoms, the administration of a small dose of calomel, or of blue pill and colocynth, is often followed by marked improvement, both in the power to take food, and in the general condition of the patient. The exhibition of purgatives, however, requires great care, as they occasionally set up a catarrhal condition of the intestine, and consequent diarrhoea, which may prove more or less persistent. It is important, therefore,

except in such cases as those above indicated to procure all necessary evacuation of the bowels, by a small dose either of castor oil, or of colocynth and hyoscyamus, or by simple enemata. With the object of promoting elimination by the skin and kidneys, some solution of acetate of ammonium with citrate of potassium may usually be prescribed with advantage in the earlier stages of the illness.

There are two circumstances which often tend greatly to interfere with the satisfactory progress of pneumonia—the pain in the side, and the cough. The former of these may usually be relieved by the application of large hot linseed poultices, or of hot fomentations, to the side. These must be frequently changed, and great care should be exercised in their renewal not to disturb or inconvenience the patient. If these means do not succeed, a small blister 2 by 2 inches, or three or four leeches, may be applied to the seat of the pain, and the hot applications then renewed. In exceptional cases small doses of morphine hypodermically may be necessary to relieve this symptom. The influence of the external application of cold will be alluded to subsequently. Cough is not usually a troublesome symptom, and, unless it greatly disturbs the patient, it is better not to interfere with it. If necessary, a linctus, containing from two to four minims of solution of hydrochlorate of morphine, with a similar quantity of spirit of chloroform, and ipecacuanha wine, given occasionally, is often beneficial. By means of the treatment above indicated, and by keeping the room quiet and darkened, the patient will often procure a sufficient amount of sleep. This can very frequently be promoted by carefully sponging the whole surface of the body, a portion at a time, with tepid or cold water, the last thing at night. Should it be necessary, some hypnotic must also be administered, but with great caution, so as not to interfere with freedom of expectoration; and it should only be had recourse to when other means have failed, and in the absence of contra-indications. Hyoscyamus and bromide of potassium may be safely used for this purpose; and, should these fail, opium may be given. This should be prescribed in a sufficiently large dose to ensure sleep, and is best administered hypodermically, as acetate of morphine. Chloral hydrate is usually contra-indicated, on account of its depressing effect upon the circulation.

A large number of cases of pneumonia terminate in health without the necessity of any further interference on the part of the physician than has been described. Frequently, on the other hand, circumstances arise indicative of danger, which require to be met by more active treatment. In those whose vitality is impaired by age, habits, or other causes, and in whom prognosis is less favourable, special care and watch-

fulness should be exercised in anticipation of any unfavourable symptoms. The chief source of danger, as already stated, is failure of cardiac power, and consequently all symptoms of such failure must be carefully watched for.

Any sign of cardiac failure will in the first place suggest the advisability of administering alcohol. The exhibition of small quantities of wine with food is sometimes useful in stimulating the appetite and assisting digestion, in cases where there are no symptoms of asthenia; but when such symptoms arise, alcohol must be employed in larger quantities. It may be stated generally that a pulse of over 120 calls for the employment of stimulants. Brandy appears in most cases to answer best. The amount administered must depend upon its effects; and although in most cases from four to eight ounces in the twenty-four hours will be sufficient, if the asthenia persist it must be given in much larger quantities. The disease is one of comparatively short duration, and there is perhaps no other acute illness in which the free exhibition of alcohol, when indicated, is more likely to be advantageous. Bark and ammonia are in some cases useful in addition to the alcohol; and digitalis in medium doses, as a means of maintaining the heart and circulation, is often of undoubted value.

In the treatment of failure of cardiac power, however, it is important to attempt to modify, as far as possible, those conditions upon which such failure principally depends. The most important of these probably is the pyrexia.

The remedies formerly employed with the object of diminishing pyrexia—aconite, tartar emetic, and veratrum—have such a depressing effect upon the heart and circulation that their use has long been abandoned. The recent antipyretics—antifebrin and antipyrin—are open to the same objection, and they occasionally cause so much prostration and collapse that they must certainly be regarded as dangerous remedies, and as requiring great caution in their employment. Although a much more extensive experience is necessary, at present the utility of these drugs in the treatment of pneumonia is extremely doubtful.

A remedy of much greater value than the preceding is quinine. In order, however, to produce any marked influence upon the pyrexia, this drug must be given in doses of from twenty to thirty grains; and inasmuch as even such large doses are rarely followed by a reduction in the temperature of more than 1° or 2° F., and are apt to cause nausea, vomiting, and other unfavourable symptoms, this method of using the drug is not generally to be recommended. It is in smaller doses of three to five grains that quinine is of undoubted use. Thus given three or four times in the twenty-four hours (perhaps best suspended in milk), it appears to steady the

nervous system and sustain the patient, although it may have little or no influence upon the temperature chart.

A much more efficient means of diminishing the pyrexia than by internal remedies, is by the external application of cold. This reduces temperature, not only by abstracting heat, but also by stimulating the nervous centres concerned in the regulation and control of heat-production and heat-loss; and its adoption in some form is in most cases useful.

The treatment of pneumonia by the external application of cold has been much more extensively employed in Germany than in this country. The application of frequently renewed cold wet compresses to the chest was advocated by Niemeyer twenty years ago, who stated that in the majority of cases thus treated, pain, dyspnoea, and pyrexial symptoms were markedly relieved, and the duration of the disease was often shortened.

The use of the cold bath is advocated by Juergensen in the earlier stages of the disease, before the heart has become enfeebled; but the difficulties attending such treatment, and the prejudice against it are such, that much more conclusive evidence of its utility is necessary before its adoption is to be recommended in any except, perhaps, those quite exceptional cases in which the temperature reaches 106° or 107° F. (hyper-pyrexia).

Sponging the whole surface of the body with cold or tepid water has already been alluded to as a useful means of promoting sleep. This is an easy method of cooling the patient, and may often be practised several times a day with distinct advantage. Packing in a wet sheet—a much more efficient means—is often useful, especially in cases attended with prominent nervous symptoms.

The most recent method of treating pneumonia by cold is by the application of an ice-bag to the chest over the inflamed lung. This treatment, which has been tested especially by Dr. Lees, is not only a very efficient means of reducing the temperature, but it appears also to control the lung-inflammation. Dr. Lees says the treatment is usually pleasant to the patient; in the great majority of cases a remarkable improvement follows the application of the ice; a reduction of temperature usually occurs at once, often amounting to 3° or 4° F.; if a subsequent rise occurs it usually rapidly yields to a second application; and the improvement is not confined to a mere reduction of temperature, but in many cases there is an arrest in the development of the physical signs. The results obtained by Dr. Lees justify the hope that the ice-bag, used with proper precautions, may prove a valuable aid in the treatment of pneumonia; and it is certainly deserving of a more extended trial.

Although failure of cardiac power is the

chief source of danger in pneumonia, dyspnoea is occasionally a most grave symptom, especially in those cases in which both lungs become extensively involved. This dyspnoea is due partly to the lung-consolidation, and partly to the pyrexia; hence it will be only partially relieved by anything which reduces the temperature. It has long been known that the dyspnoea of pneumonia is diminished temporarily by venesection, and at the present day this is probably the only symptom in this disease for the relief of which the practice of bleeding would be regarded as at all justifiable. In considering the advisability of removing blood in those cases in which dyspnoea constitutes an urgent symptom, it must, however, be borne in mind not only that the relief is merely temporary, but that the loss of blood must tend more or less to weaken the patient, and hence to favour that condition of asthenia which is of all things the most to be feared. Bleeding is certainly only to be thought of when the dyspnoea threatens life, and when at the same time the strength, as indicated by the pulse, is good; and it should not exceed the removal of eight or ten ounces of blood. Such cases are certainly not common.

Of the complications, delirium sometimes calls for treatment. Active delirium is not common in pneumonia, except in those who have been intemperate. When marked, it is always indicative of danger. In its management the practitioner must be guided by the general condition of the patient; but, speaking generally, it calls for the free exhibition of alcohol, and the external application of cold. Cold, applied in some of the ways already indicated, is of especial value in these cases, not only reducing the temperature, but quieting the patient and often producing refreshing sleep. The influence of an experienced nurse is all-important. In some cases it may be advisable to administer an opiate. This should only be done, however, as a last resource; and when there is marked asthenia it is inadvisable. The opiate should be given in one full dose sufficient to procure sleep. Morphine administered hypodermically is the best form of exhibiting it.

Of the management of other complications occurring in the course of pneumonia there is nothing special to be remarked; they must be treated on general principles. The existence of bronchial catarrh often requires small doses of ipecacuanha with carbonate of ammonium and salines. Diarrhoea and gastric symptoms are to be met by careful dieting, chalk, bismuth, and, if necessary, other astringent remedies. In pneumonia, as in other acute diseases, the administration of an opiate enema is an efficient and safe means of checking diarrhoea. Pericarditis rarely admits of any special interference.

*Convalescence.*—During the period which immediately succeeds the crisis, the utmost

care is required to support the patient, and to prevent any serious amount of prostration, which at this time so often supervenes. Stimulants are usually required for some days after the temperature has attained the normal standard. Convalescence in most cases is quickly established. Solid diet is soon desired, and may be safely given. Tonics—such as quinine, iron, and cod-liver oil—and change of air are useful in assisting the restoration to health.

**B. Secondary Pneumonia.**—*DEFINITION.*—Secondary pneumonia is an inflammation of the lung occurring in those who are the subjects of some other disease; the pneumonic process standing in more or less causal relation to the disease in the course of which it supervenes.

*Intercurrent pneumonia.*—Before proceeding to consider secondary pneumonia as thus defined, we have to remark that ordinary pneumonia occasionally occurs in the course of other diseases as an *accidental* complication. Such pneumonias may be termed *intercurrent*, and they are to be distinguished from the secondary affections. They usually closely resemble, in their clinical features, the acute disease, as it has been already described; although in some cases they are more or less modified by the disease in the course of which they supervene.

*Hypostatic pneumonia.*—There is also a class of consolidations of the lung very common in those who are the subjects of other diseases, which are often described as secondary pneumonias, though really for the most part non-inflammatory in their nature; but they may be conveniently alluded to in the present article. These are the consolidations so often met with at the bases and more dependent portions of the lungs, in the course of both chronic and acute diseases, and also in the aged. They have been termed *hypostatic pneumonias*, and consist, in the main, of collapse, hyperæmia, and œdema of the lung-tissue, resulting from weak inspiratory power, feeble circulation, and gravitation. The consolidation thus mechanically induced is increased by more or less exudation of liquor sanguinis and blood-corpuscles into the alveoli; which exudation is due to the damage to the walls of the capillaries caused by the blood-stasis. There is also some swelling and shedding of the alveolar epithelium.

*ÆTIOLOGY.*—Secondary pneumonias, as already stated, bear a causal relation to the disease in the course of which they supervene. They would seem to owe their origin almost exclusively to some abnormal condition which the pre-existing disease has induced. They occur in the course of many diseases, and sometimes appear to constitute the acute affection which determines dissolution. In Bright's disease they are the most

frequent. Ordinary pneumonia may, however, occur in the course of Bright's disease.

**ANATOMICAL CHARACTERS.**—The changes occurring in the lung in secondary pneumonia are, for the most part, precisely similar to those of the acute primary disease. In many cases, however, the consolidation is less dense; and epithelial changes sometimes constitute a more prominent feature in the process. The pleura is usually implicated, but not so invariably so as in ordinary pneumonia. With regard to the situation of the consolidation, it is more frequently situated in the upper and middle lobes, and is more often double than is that of the primary disease.

**SYMPTOMS.**—The clinical phenomena of secondary pneumonia differ from those of the acute primary disease, the symptoms so characteristic of the latter being almost entirely wanting. The symptoms which do exist are often but little pronounced, and the disease may even run an almost latent course. This latency of symptoms is often partly due to their being masked and modified by those of the disease to which the pneumonia is secondary.

The onset of the pneumonic process is usually unattended by rigors or other marked phenomena. Cough, expectoration, pain, and dyspnoea are often slight, and they may indeed escape observation. If cough be present, the sputa may be free from blood, and simply watery or muco-purulent. The pyrexia is moderate, the temperature often not being more than 100° F. Not infrequently slightly increased frequency of the respiration, with occasional cough, and symptoms of increased illness, are all that exist to indicate that inflammation of the lungs has supervened.

**DIAGNOSIS.**—Owing to the frequent latency of the symptoms of secondary pneumonia, the diagnosis often rests, for the most part, upon the existence of physical signs of pulmonary consolidation. When the consolidation occupies the posterior and inferior portions of the lung, it may be impossible to distinguish it from simple hypostasis.

**PROGNOSIS.**—The supervention of secondary pneumonia in the course of a chronic disease usually, but by no means invariably, indicates that the disease will shortly terminate in death. The pulmonary inflammation appears to determine dissolution. Pneumonia occurring in acute diseases materially increases the gravity of the prognosis.

**TREATMENT.**—The treatment of secondary pneumonia usually resolves itself into that of the disease in the course of which it occurs. Warm applications to the chest, small doses of carbonate of ammonium, and alcoholic stimulants may sometimes favourably influence the pneumonic process.

**C. Broncho-Pneumonia.** — **SYNON.:** Catarrhal Pneumonia; Lobular Pneumonia;

Fr. *Pneumonie Lobulaire*; Broncho-pneumonie; Ger. *Bronchopneumonie*.

**DEFINITION.**—Broncho-, catarrhal, or lobular pneumonia is inflammation of the lung-tissue, due to an irritant entering and spreading by the bronchi, which usually causes, in the first place, catarrh of the bronchial mucous membrane. This frequent association of the pulmonary inflammation with bronchial catarrh explains the terms *broncho-* and *catarrhal*, which are applied to this form of pneumonia. In the earlier stage the pulmonary inflammation is commonly limited to scattered groups of air-vesicles (*lobular*), but as the process advances, the inflammatory nodules may gradually coalesce so as to produce larger tracts of consolidation. The inflammatory products which fill the alveoli consist principally of cells, derived from the epithelium of the alveoli, and from the bronchial mucous membrane; exudation and emigration play a much less prominent part in the process than they do in ordinary pneumonia.

**ÆTIOLOGY.**—Broncho-pneumonia, as already stated, is generally associated with bronchial catarrh. In some cases it would appear that the irritant which produces the bronchial inflammation produces at the same time inflammation of the alveolar walls, but much more frequently the bronchitis precedes the pneumonia, and gives rise to it in a manner to be hereafter described. Whatever causes inflammation of the bronchial mucous membrane may thus be a cause of broncho-pneumonia.

Simple non-specific bronchitis is frequently followed by broncho-pneumonia, especially in childhood and in old age. All those conditions which favour the occurrence of such bronchitis must therefore be enumerated amongst the causes of this form of pneumonia. Of these conditions it will be sufficient to mention here the marked influence of cold and damp. It is, however, the specific forms of bronchitis, associated with measles, whooping-cough, diphtheria, variola, and influenza, which are the most liable to be followed by broncho-pneumonia. This is especially the case with that accompanying measles and whooping-cough. In both these diseases this form of lung-complication is exceedingly frequent.

Less frequent causes are dusts of various kinds—particles of earth, stone, steel, &c.; organisms, the most important of which is the bacillus of tubercle; and, lastly, putrid discharges from wounds or diseases of the pharynx, nose, or other parts of the passages. These various irritants may, by aspiration into the bronchi, give rise to bronchitis and broncho-pneumonia.

All conditions which tend to impair the general health and strength, favour the occurrence of broncho-pneumonia. The weakly and debilitated suffer most. Bad air and

insufficient food are important predisposing causes. Depressed health not only weakens the resistance of the tissues, and thus favours the development and spread of inflammation, but also diminishes the power of the respiratory muscles, and thus aids in the production of pulmonary collapse, a condition which, as will be seen presently, is especially favourable to the pneumonic process. Owing to the general debility and weakness of the thoracic parietes in rickets, bronchitis in the subjects of this disease is exceedingly liable to be followed by broncho-pneumonia.

Broncho-pneumonia is most common during the first four years of life—the period when bronchial catarrh, measles, and whooping-cough are so frequent. It is also common in old age. In young adults it is comparatively rare. Sex has no influence.

**ANATOMICAL CHARACTERS.**—The appearances presented by the lungs after death from broncho-pneumonia vary. The bronchi always exhibit signs of more or less bronchial catarrh. This may involve the whole of the bronchial mucous membrane, but it is usually most marked in the smaller tubes. These are found containing a thick, tenacious, and often puriform secretion, which is occasionally here and there drier or inspissated. The mucous membrane of these tubes is more or less softened, swollen, red, and thickened, and often presents irregular superficial erosions. Owing to this inflammatory swelling, the tubes stand out prominently on section of the lung. Cylindrical dilatations of the tubes are also frequently met with.

The lung-tissue itself exhibits, associated in various degrees, collapse, congestion, œdema, emphysema, and pneumonic consolidation. The bluish, non-crepitant, depressed portions of collapse, which become darker and more friable with age, are usually most abundant in the lower lobes and margins of the lungs. The collapse sometimes involves the whole of one lobe, but more commonly it is limited to much smaller areas of the lung. When scattered and limited in its distribution, there is usually more or less emphysema of the intervening portions of the lung; when collapse is very extensive in the lower lobes, emphysema is most marked in the upper.

Those portions of the lung in which the pneumonic process has supervened appear most commonly as scattered patches and nodules of consolidation, varying in size from a small pea to a hazel-nut. These are usually more or less conical in shape, with their bases towards the pleura, which membrane is generally unaffected over them, though it may be coated with lymph. On section, the patches are commonly ill-defined and pass insensibly into the surrounding tissue, which is variously altered by congestion, collapse, and emphysema. They are of a reddish-grey colour, slightly elevated, smooth, or faintly granular, and soft and friable in consistence.

As they increase in size they may become confluent, and thus are produced large tracts of consolidation. In a more advanced stage, the nodular and more diffuse consolidation becomes paler, firmer, and drier, and resembles in colour the greyish-yellow hepatisation of acute pneumonia. The cut ends of dilated bronchi, filled with pus, are occasionally seen in the centres of the pneumonic nodules. When the pneumonic process is due to the aspiration of putrid particles, abscesses are found instead of these solid patches.

**Microscopical characters.**—When examined microscopically, this consolidation is seen to consist of an accumulation within the alveoli of a gelatinous mucoid-looking substance, small cells resembling leucocytes, epithelial elements, and red blood-corpuscles. In many cases much of this accumulation is precisely similar to that contained in the smaller bronchi; and it is evidently the inflammatory and richly cellular bronchial secretion which has been inhaled. At the same time, it is doubtless partly the result of exudation and emigration from the pulmonary capillaries; such exudation and emigration, however, do not play nearly such a prominent part in the process as they do in acute pneumonia, and a fibrinous coagulum is rarely met with. The epithelial elements are swollen and granular, and are probably the alveolar epithelium which has been shed. The relative proportion of epithelium and leucocytes varies considerably in different cases, probably owing to differences in the intensity of the inflammation, and to the degree in which the inhalation of the bronchial secretion constitutes a part of the process.

The subsequent changes which take place in the lungs vary. When the disease does not end in death, resolution is the most common termination. The contents of the alveoli undergo fatty degeneration, and are removed by expectoration and absorption, the lung gradually regaining its normal characters. This process, however, is less readily effected than in the consolidation of acute pneumonia; and it often occupies such a lengthened period, that some thickening of the bronchial and alveolar walls and dilatation of the smaller bronchi remain. In still more chronic cases the fibroid thickening is much more marked, and a considerable amount of irregularly distributed, pigmented induration and bronchial dilatation may be produced. In these chronic forms the contents of the alveoli sometimes caseate, and the caseous products become encapsuled.

**PATHOLOGY.**—The inflammation of the bronchial mucous membrane which is usually associated with broncho-pneumonia, in the great majority of cases precedes and is an important cause of the pneumonic process. In some cases it would appear probable that the same injury which produces the bronchial inflammation produces at the same time the

inflammation of the air-vesicles. Inflammation of the bronchial mucous membrane may give rise to broncho-pneumonia in two ways: (1) by causing, in the first place, collapse of the lung-tissue; and (2) by the direct extension of the inflammation from the bronchi to the air-vesicles. The pneumonic process, being the result of the bronchitis, almost invariably involves both lungs.

(1) *Broncho-pneumonia consecutive to collapse*.—Collapse of the lung-tissue greatly favours the occurrence of broncho-pneumonia, and usually the pneumonic process is principally confined to those portions of the lung in which collapse has taken place. This is particularly the case in young children. Although it would be beyond the scope of the present article to discuss at length the relation which subsists between bronchial catarrh and pulmonary collapse, the mode of production of the latter may be briefly indicated. There are two circumstances principally concerned in the production of the collapse which is consecutive to bronchitis—the narrowing of the bronchial tubes, and the weakness of the inspiratory power. The mucous membrane of the bronchi becomes considerably swollen as the result of the inflammatory process, often being thrown into folds; and its surface is covered with thick tenacious mucus. These conditions may cause so much narrowing of the smaller tubes as to render the entrance of air exceedingly difficult, and they may even completely prevent it. In addition to the bronchial narrowing, the power of inflating the lungs is usually diminished. This is due partly to the general debility which so often exists prior to the bronchitis, and partly to the damage to the respiratory muscles caused by the febrile process. The superficial character of the respiration due to the fever also aids in the production of the collapse, as does also any weakness of the osseous structures of the thorax, such as exists in rickets. The collapse thus induced is especially frequent in the posterior and inferior portions of the lungs—those portions in which normally the inflation of the lung is the least complete. Commencing here, the process may gradually extend upwards till large areas of both lungs become involved. In other cases, owing to a more irregular distribution of the bronchial obstruction, the collapse is limited to small isolated portions of the lung. These portions vary in size from a hemp-seed to a walnut. They are commonly more or less wedge-shaped, with their apices towards the bronchus leading to the group of collapsed lobules; and the lung-tissue around them usually presents various degrees of congestion and emphysema.

The tendency of the pneumonic process to occur in the collapsed portions of the lung is due partly to the hyperæmia which is induced by the collapse, and partly to the irritation of inhaled bronchial secretion. Collapse of the

lung-tissue invariably induces more or less congestion. This is owing to the absence of the expansion and contraction of the air-vesicles which normally aid the pulmonary circulation, and also to the impeding to the blood-flow resulting from imperfect aëration. Thus results more or less damage to the vessel-walls, and consequent escape of some of the constituents of the blood. The congestion is quickly followed by œdema, and the bluish-purple collapsed portions of the lung become deeper in colour, less resistant, and more friable in consistence.

Another circumstance which often appears to play a prominent part in the causation of the pneumonic process, is the presence within the alveoli of the inflammatory products of the bronchial mucous membrane. Such products are frequently found in the lungs in cases of broncho-pneumonia. They occur in scattered groups of air-vesicles, and are evidently inhaled. They are found both in the air-containing and in the collapsed portions of the lung, but especially in the latter, the presence of collapse necessarily interfering with their removal by expectoration or absorption. These inhaled products are often found filling small groups of alveoli without any evidence of subsequent inflammation, and there can be little doubt that many of the patches of consolidation which are usually described as pneumonic are in reality non-inflammatory in their nature, and are thus produced. At the same time, owing to the irritation of the inhaled secretion, it tends to induce inflammatory changes within the alveoli, and these changes are frequently largely owing to its presence. Juergensen accounts for the pneumonic process occurring in isolated spots in the collapsed lung by regarding the inflammation as being determined by the inhaled bronchial secretion.

(2) *Broncho-pneumonia independent of collapse*.—The pneumonic process may occur independently of collapse, being due solely to the spreading of the inflammation from the smaller bronchi to the air-vesicles. The irritation of substances inhaled into the alveoli may also be the sole cause of the pulmonary inflammation.

**SYMPTOMS.**—The symptoms of broncho-pneumonia vary according to the rapidity with which the disease is developed, the extent of lung involved, and the nature of the disease in the course of which it supervenes.

The more severe and most common forms of the disease are met with in early childhood in association with simple bronchitis, and the specific bronchitis of measles and whooping-cough. In these cases the symptoms and dangers, are in the main, those of the capillary bronchitis which precedes the pneumonic process, and it is sometimes impossible to distinguish between them. Frequent cough, accelerated respiration, slight action of the nares, and other symptoms of severe bron-

chitis, with perhaps a little pyrexia, precede for a varying length of time the symptoms due to the implication of the lung-tissue. The earliest symptoms of the pneumonic process are by no means well-defined, and consequently the time at which the lung becomes involved cannot be fixed with certainty. Rigors, convulsion, and vomiting are but rarely observed. Usually an increase in the acceleration of the respiration or in the dyspnoea, and a marked rise of temperature, are the first signs of the pulmonary implication. The breathing becomes very rapid, and commonly causes much distress, the child tossing about and being exceedingly restless. This dyspnoea is more marked at some times than at others, and is occasionally more or less distinctly paroxysmal. The respiration is superficial, inspiration being short, and the expansion of the thorax imperfect. There is marked action of the accessory respiratory muscles, and the upper portions of the thorax are raised, whilst the lower and the soft parts are retracted during the inspiratory act. The action of the nares is very pronounced.

An increase in any pyrexia which attended the pre-existing bronchitis is, with few exceptions, observed as the lungs become involved, and such increase is to be regarded as one of the most valuable indications of the existence of a pneumonic process. The maximum temperature of acute simple bronchitis, even in the child, is seldom higher than 102° F., whereas that of the secondary pneumonic process is often 104° or 105°. This increase usually occurs more or less gradually; there is rarely the sudden rise of temperature met with in acute pneumonia. Unlike the temperature of this disease also, the fever of broncho-pneumonia runs no definite course. It varies with the extent of the lung-implication, and with the rapidity with which this implication is effected. There is no regular diurnal variation; the remissions and exacerbations are often considerable; and they occur at irregular times, the temperature being sometimes higher in the morning than at night. The cough, which before the implication of the lung was paroxysmal in character, gradually becomes less and less so, and it now often causes much pain to the patient. The sputa, when present, are bronchitic in character, usually tenacious, and occasionally streaked with blood; as, however, expectoration rarely occurs in the child, they are not often seen. The pulse is much increased in frequency, in children under five years often being 150. It may in the earliest stage of the disease be moderately full and strong, but it quickly becomes soft, small, and feeble. In addition to the above, there are often symptoms referable to the digestive organs. Of these diarrhoea is the most important. This is quite frequent, especially when the disease follows measles.

It is very readily induced by medicines and by improper feeding; and as it greatly weakens the patient, it is important that this liability to diarrhoea should be kept in mind. Vomiting, as already stated, is very rare as an initial symptom; it is, however, common in the course of the disease, especially as a result of cough, the bronchial secretion, together with the contents of the stomach, often being expelled.

As the bronchitis and the implication of the lungs increase, the breathing becomes still more rapid and superficial; the dyspnoea is more marked; the expression is anxious; the face is pale; and symptoms of carbonic-acid poisoning become evident. Strength now fails; the face and lips become cyanotic; and the extreme restlessness gives place to apathy and a semi-comatose condition, which is interrupted from time to time by ineffectual efforts to cough. With the rapid failure of strength and increasing cyanosis, cough almost ceases; the pulse becomes exceedingly feeble; and the child, often extremely emaciated, may die exhausted, and in a condition of more or less profound coma. Sometimes death occurs suddenly during a paroxysm of cough, or with convulsions.

In those cases in which capillary bronchitis constitutes a less prominent feature of the illness, the symptoms are usually less severe—the pyrexia is less marked and its onset more gradual; the pneumonic process often supervenes later; and the course of the disease is more protracted. A slight and markedly irregular pyrexia, increasing difficulty of breathing, with loss of strength and flesh, may continue for months, and the child ultimately die, or recover with more or less damaged lungs. In these more chronic forms of the disease, which are perhaps most common after whooping-cough, the pneumonic process is often less disseminated, and involves larger areas of the lung—not infrequently a whole lobe. This is owing to bronchitis constituting a less, and pulmonary collapse a more, prominent element in the causation of the disease.

When broncho-pneumonia occurs in adults and in the aged, the symptoms are for the most part much less pronounced than in the child. In strong adults the disease is perhaps most common after diphtheria, and here the pulmonary symptoms may be well-marked; but in the debilitated, and especially in the old, the course of the disease is much more latent, very slight pyrexia (100° F.), slight cough and dyspnoea, and marked debility being the principal symptoms observable.

In the aged and feeble, broncho-pneumonia is frequently associated with that form of lung-consolidation which results from weak inspiratory power, feeble circulation, and gravitation; and which consists mainly of collapse, hyperæmia, and œdema of the lung-

tissue (hypostatic pneumonia). This hypostatic consolidation may exist quite independently of bronchial catarrh; but when such catarrh occurs in the aged and feeble, gravitation often determines the supervention of the pneumonic process, which under such circumstances is consequently not infrequently unilateral.

When the more acute varieties of broncho-pneumonia terminate fatally, they usually do so from the tenth to the fourteenth day of the disease. Death, unlike that from acute pneumonia, is mainly due to the interference with the respiratory function, and, to a much less extent, to failure of cardiac power. The interference with the respiration is much greater than in acute pneumonia, for, in addition to the diminution of the respiratory area, due to the pulmonary consolidation and collapse, there is the much more important cause of interference—namely, the impediment to the entrance of air, caused by the swelling of the bronchial mucous membrane, and the accumulation of secretion in the bronchial tubes. These interferences with respiration necessitate increased action of the respiratory muscles; but with the progress of the disease these muscles become weakened, partly by the fever, and partly from the imperfect supply of oxygen. With this failure of respiratory power the incompleteness of oxygenation necessarily increases, until the supply of oxygen may become so small as to lead to complete muscular paralysis. The damage to the heart, as in acute pneumonia, is due partly to the diminished respiratory area, and partly to the fever; but this damage is a much less important element in the causation of dissolution in broncho-pneumonia, than it is in the acute disease. In the more chronic forms of broncho-pneumonia death may not occur for some months, and then it results as much from general failure of strength as from interference with the respiratory function.

When the disease does not terminate in death, improvement in the symptoms is always gradual. The temperature falls slowly, several days, and occasionally some weeks, being occupied in the completion of deferrescence; and this gradual decline is usually interrupted by more or less marked and frequent exacerbations and remissions of the fever. The cough and dyspnoea diminish, and the appetite gradually returns; but restoration to health is always protracted; and the child remains for some time especially liable to repetition of the bronchial symptoms.

Sometimes recovery from the broncho-pneumonia is not complete, and the disease leads to induration of the lung, with dilatation of the bronchi. Emphysema and pulmonary tuberculosis are occasional sequelæ.

*Physical signs.*—The physical signs of

broncho-pneumonia are in the main those of capillary bronchitis. Imperfect expansion of the thorax, elevation of the upper portions, and recession of the lower, during the inspiratory act; moist and dry *râles*, audible over both sides; and the absence of any marked alterations in percussion-resonance, are the principal signs observable, not only in the earlier stages, but throughout the whole course of the disease. The recession of the chest-walls is increased by collapse. The pulmonary implication is indicated rather by the symptoms—increase in the pyrexia and in the dyspnoea—than by any marked alteration in the physical signs. The difficulty of detecting the lung-consolidation is due to its usually being limited, in the earlier stages, at all events, to small areas, which are surrounded by healthy or emphysematous lung, so that resonance on percussion is but little impaired. It is only when these small areas have coalesced into larger areas of consolidation, that any marked alterations in percussion-resonance are discoverable. The impaired resonance due to collapse is not to be distinguished from that due to pneumonic consolidation; and, inasmuch as the collapse is so often symmetrical, involving both bases posteriorly, the difficulty of appreciating it is increased. Much more valuable aid in physical diagnosis is in most cases to be obtained from auscultation. Over those portions of the lung where consolidation has taken place, the moist bronchitic *râles* tend to assume a somewhat metallic quality; they also become finer, though not so fine as true pneumonic crepitation; and they are more superficial. The detection of these superficial, somewhat metallic fine moist *râles*, heard with inspiration, and not dispelled by cough, over small areas of the lungs, especially at the posterior bases, is a most valuable and often the only physical sign of the pulmonary implication. If larger areas become consolidated, there may be in addition some tubular breathing, and some impairment of resonance on percussion; and when a whole lobe is involved the physical signs become increasingly pronounced, and in the child a bronchophonic cry is often marked on auscultation.

*COMPLICATIONS.*—Pleurisy is less common than in acute pneumonia. Slight inflammation of the pleura is, however, usually found *post mortem* over those portions of the lung which are consolidated. Pleuritic effusion is rare. Intestinal catarrh is a very important and common complication. The liability to this in the child, and the mechanical congestion resulting from the obstructed pulmonary circulation, must be borne in mind in explaining its frequency.

Convulsions occasionally occur, and are of unfavourable augury. The nervous phenomena in some few cases have been described as simulating those of tubercular meningitis.

Catarrhal laryngitis, associated with much spasm and laryngeal stenosis, is sometimes observed, especially after measles.

**DIAGNOSIS.**—The diagnosis of broncho-pneumonia is occasionally difficult. This difficulty is mainly owing to the co-existence of capillary bronchitis. The recognition of the pulmonary implication in its earlier stages is often impossible. The increased pyrexia and frequency of respiration are the symptoms of the most diagnostic value. Owing to the small areas of lung involved, any alteration in the physical signs of the capillary bronchitis may be entirely wanting. The occurrence of extensive collapse in the earlier stages gives more marked physical signs of consolidation, and hence renders the diagnosis more easy. It is almost impossible to diagnose certainly, either by symptoms or by physical signs, between the collapse and the pneumonic consolidation. This, however, is of but little practical importance, inasmuch as the collapse is usually associated with, and often the immediate precursor of, the pneumonic process.

The diagnosis of broncho-pneumonia from the pulmonary consolidation of acute pneumonia may occasionally be difficult in those cases of the former in which an extensive area of the lung has become consolidated. The history of the case, and especially the course of the pyrexia, will usually suffice to distinguish them.

The distinction from pulmonary tuberculosis sometimes presents much difficulty, as does also the recognition of tuberculosis and phthisis as an occasional result of the disease. A careful consideration of the earlier symptoms, and the existence or not of marked predisposition, are here most important. Slight and irregular pyrexia, existing before the supervention of lung-symptoms, is greatly in favour of tuberculosis. In some cases, however, the phenomena of these diseases are so analogous that a certain diagnosis is impossible.

**PROGNOSIS.**—The two circumstances which have an especial influence upon prognosis in broncho-pneumonia are the age of the patient, and the general health. Before puberty, the younger the patient the graver the prognosis. In children under five years, the mortality is exceedingly great (probably about 20 per cent.) The disease is also especially fatal in weakly children, and in all those who are constitutionally feeble, or debilitated by previous illness. The existence of rickets materially increases the gravity of prognosis. The danger also increases greatly with the extent of lung involved, much more so than is the case in acute pneumonia. Of the value of the several symptoms as influencing prognosis, after the description which has been given of the disease and of the modes in which it tends to cause death, it is hardly necessary to speak further. Symptoms of

imperfect aëration of the blood are those most to be feared.

**TREATMENT.**—In the treatment of broncho-pneumonia, it is important to bear in mind—first, that the disease is generally associated with, and is in the main induced by, bronchial catarrh, and by its so frequently attendant collapse; second, that its occurrence is especially favoured by everything that weakens the patient; and, third, that it tends to destroy life principally by interfering with the function of respiration, which interference necessarily increases with the consequent weakening of the respiratory power. Such being the facts, it is obvious that the main object of treatment will be, first, to control bronchial catarrh, and endeavour so to modify it as to prevent the occurrence of collapse; and, secondly, to support as much as possible the strength of the patient, with the object of preventing not only collapse, but also that increased interference with the function of respiration which results from weakening of the respiratory muscles.

It would be out of place in the present article to enter into a detailed description of the management of acute bronchitis (*see BRONCHI, Diseases of*). It will be sufficient to indicate the more important means of controlling the disease, with especial reference to the prevention of the so frequently attendant collapse.

The patient should be kept in a warm room, the temperature of which should never be allowed to fall below 62° F. The room should be well, but carefully ventilated; and protection from draughts is important—much more so than in the treatment of acute pneumonia. It is also advisable to keep the air moist by means of a steam-kettle, as the exhalation of water from the lungs is thus diminished, and the bronchial secretion consequently rendered less tenacious, and more easily removed by cough. A little carbolic acid—1 in 60—may be added to the water. The diet, which must be regulated according to the age of the patient, should be nutritious and easily digestible, the importance of supporting the strength being kept in mind. When the disease follows measles, the liability to gastro-intestinal catarrh must not be forgotten. Small doses of ipecacuanha wine with salines, and a little carbonate of ammonium, should be administered frequently. The chest should be enveloped in lightly made linseed and mustard poultices; or, as it is all-important not to interfere with the respiratory movement, it is wiser in young children to use some stimulating liniment, and apply a jacket of cotton-wool covered with oil-silk. When the secretion in the tubes is abundant, its removal may be much aided by small doses of carbonate of ammonium. This may be either combined with the ipecacuanha and saline mixture, or given separately in a little milk. Senega,

as an infusion, and chloride of ammonium may also be given with the same object. The last-named drug appears to have the effect not only of rendering the secretion less tenacious and more easily removable, but also of diminishing its formation. An occasional emetic dose of ipecacuanha often materially relieves the patient, when numerous *râles*, audible over the chest, and increased dyspnoea, indicate an accumulation of the secretion. The exhibition of opiates is obviously contra-indicated.

In the acute forms of the disease, so common in early life, much may be done by the external application of cold. This not only reduces the temperature, but appears to be especially valuable in increasing the depth and force of respiration, and thus in preventing the occurrence of collapse. Its utility has been strongly advocated by both Bartels and Ziemssen, who recommend the frequently renewed application of cold wet compresses to the chest. The use of tepid baths, with cold affusions, is a more efficient way of obtaining the same result. The child is placed in a bath of from 85° to 90° F., and, whilst immersed, the head, back, and chest are quickly sponged with cold water. This repeated occasionally, with due precautions, is often followed by reduction of temperature, a diminution in the frequency and an increase in the depth of the respirations, and other signs of improvement. The application of ice-bags or ice-poultices to the chest often yields good results. The effects produced by the cold require in all cases to be carefully watched, and any depression caused should be met by the timely exhibition of stimulants.

Whilst these various means are being employed, it is all-important to support the strength of the patient. Brandy is here most valuable, and it is to be remembered that children bear stimulation well. The brandy is best given in milk, or with yolk of egg, the quantity being proportioned to the age of the patient. An infant may begin with from five to ten drops every two or three hours. Under its influence the pulse usually improves, the respirations become less frequent, and the distress and cyanosis diminish. The administration of brandy is usually advisable before and during the treatment by cold. When prostration is extreme, or deglutition difficult, both the brandy and other nutriment may be administered by the rectum.

In the more chronic forms of bronchopneumonia these active methods of treatment are but rarely called for. Here attention to strength and nutrition is most important, and small doses of cod-liver oil in the later stages, even before the complete disappearance of the pyrexia, are often useful. When the disease leads to induration of the lungs and dilatation of the bronchi, the treatment resolves itself into that of chronic pneumonia.

Convalescence, it must be remembered, is always slow, and there is a tendency to relapse. Great care is consequently requisite during this period. All causes of catarrh must be carefully guarded against; and the restoration to health assisted by nutritious diet, cod-liver oil, and preparations of iron. A change of air is especially valuable.

**D. Chronic Pneumonia.** — **SYNON.:** Chronic Inflammation of the Lungs; Cirrhosis of the Lung; Fr. *Pneumonie Interstitielle*; Ger. *Lungencirrhose*.

**DEFINITION.**—Chronic pneumonia is a comparatively rare disease, characterised by a gradual increase in the connective tissue of the lung, which leads to an induration of the pulmonary texture, and to progressive obliteration of the alveolar cavities. It is commonly associated with catarrh and dilatation of the bronchi, and often with ulceration of the bronchial walls, and excavation of the indurated lung. Cough, expectoration—often abundant, but varying with the bronchial catarrh—dyspnoea, gradual impairment of nutrition, and occasional accessions of slight pyrexia, are the most prominent clinical phenomena accompanying the disease, which runs an exceedingly chronic course, often subject to long periods of quiescence, but tending to terminate fatally in from five to fifteen years.

Chronic pneumonia is also known as *interstitial pneumonia*. In its most marked form it constitutes the disease which received from Corrigan the name of *cirrhosis*. The term 'fibroid phthisis,' which is sometimes applied to it, is altogether inapplicable.

**ÆTIOLOGY AND PATHOLOGY.**—It is doubtful if chronic pneumonia is ever a primary and independent disease. It probably in all cases owes its origin to some more acute inflammation of the pulmonary or bronchial textures, or of the pleura, although cases are sometimes met with in which no history of any such antecedent affection is discoverable. It may be stated generally that all inflammatory processes in the lungs, as in other organs, which become chronic, lead to an increase of the connective-tissue elements, and consequently to fibroid induration of the organ. In the lungs by far the most common cause of such induration is chronic pulmonary tuberculosis. In all cases of phthisis, excepting those which are the most acute, there is more or less fibroid growth; and the extent of this growth is, for the most part, in direct proportion to the chronicity of the disease. Those forms of phthisis which are the most chronic, and in which the fibrosis reaches its maximum, have been termed 'fibroid phthisis.' The most chronic cases of phthisis are, it must be admitted, somewhat closely allied to some forms of chronic pneumonia. The two diseases, however, differ pathologically in this respect—that, whereas much of the

pulmonary consolidation of phthisis tends to undergo molecular death and caseation, that of chronic pneumonia exhibits no such tendency; but any destruction and excavation of the indurated lung which may take place is due to secondary inflammation and ulceration commencing in the bronchial walls. In considering the pathology of chronic pneumonia, therefore, it is necessary to exclude in the first place the pulmonary fibrosis of phthisis. Chronic pneumonia must also be separated from that form of pulmonary induration which is produced by long-continued mechanical congestion—'brown induration;' and from those more localised indurations due to peri-bronchitis, old infarctions, syphilis, and atelectasis.

There appear to be four conditions which may give rise to chronic pneumonia, namely: (1) *Acute pneumonia*; (2) *Broncho-pneumonia*; (3) *Pleurisy*; and (4) *the inhalation of irritating particles of solid matter*. Each of these must be considered separately.

(1) *Acute pneumonia*.—Chronic pneumonia is an occasional, though very rare, result of the acute primary disease. The pulmonary consolidation of acute pneumonia almost invariably undergoes complete resolution. This resolution is usually effected rapidly, in from seven to fourteen days. Occasionally, however, the course of the disease is more protracted, and the consolidation persists beyond the third week. When thus protracted, the hepatised lung tends to become slightly indurated, owing mainly to thickening of the walls of the alveoli. This indurated hepatisation differs but little in its physical characters from ordinary red and grey hepatisation; it is simply somewhat firmer and more resistant. In very exceptional cases this small amount of induration commencing in the alveolar walls may gradually increase, so as ultimately to give rise to that extensive fibrosis of the lung which constitutes what is usually known as chronic pneumonia.

(2) *Broncho-pneumonia*.—Broncho-pneumonia appears to be a somewhat more frequent cause of the disease than the preceding (Wilson Fox). The greater liability of this form of pneumonia to lead to pulmonary induration is to be accounted for partly by its longer duration and greater tendency to become chronic, and partly by the existence of bronchial dilatation with which it is so frequently associated. That bronchial dilatation is favourable to an indurative pneumonic process has been especially insisted upon by the late Wilson Fox. Dilatation of the bronchi is exceedingly common in the simple and specific bronchitis of childhood, and especially in that associated with whooping-cough; it is also a direct result of pulmonary fibrosis. In whatever way originating, its existence favours the persistence of the catarrhal and pneumonic processes. The removal of secretion is rendered more difficult;

the retained secretion tends to increase and keep up the irritative process, both in the dilated bronchi and also in the pulmonary alveoli; and this persistence of the bronchial and pulmonary inflammation leads to fibroid thickening of the bronchial and alveolar walls. In this way more or less disseminated patches of indurative consolidation are produced, which as the process goes on gradually increase, so that ultimately they may involve large areas of the lung. The progressive tendency of the process is probably partly to be explained by the fact, already stated, that pulmonary fibrosis is a cause of bronchial dilatation, so that fibrosis once established, by inducing further dilatation of the bronchi, favours the extension of the bronchial and pulmonary inflammation.

(3) *Pleurisy*.—Pleurisy in exceptional cases leads to the development of a chronic pneumonia. It appears to be in those cases of pleurisy which are more or less chronic, and in which the lung remains long collapsed from the effusion, that such a result is most liable to occur. The induration of the lung thus induced is sometimes, however, exceedingly partial, consisting merely in some increase of the interlobular connective tissue originating and extending inwards as dense bands from the thickened visceral pleura. In other cases pleurisy gives rise to a much more general fibrosis.

(4) *Inhalation of solid irritating particles*. This, which occurs in miners, potters, stone-masons, grinders, &c., is the cause of the fibrosis of the lungs so common amongst persons so employed. The continuous irritation of the inhaled particles induces a bronchial and alveolar inflammation, and ultimately a progressive fibrosis, which, gradually extending, may involve large areas or even the whole of the lungs. These cases sometimes become tuberculous.

ANATOMICAL CHARACTERS.—The histological changes met with in the lungs in chronic pneumonia may be described generally as consisting in the development of a fibro-nucleated tissue from the walls of the alveoli, from those of the bronchi, and from the interlobular connective tissue; which new-growth, as it increases, and from its tendency to contract, gradually replaces and obliterates the alveolar structure. The character of these changes, however, varies somewhat according to the more acute inflammatory antecedents in which they originate. When the result of acute pneumonia, the primary, and usually the principal, change takes place in the walls of the alveoli, although ultimately the interlobular tissue is involved. The alveolar walls become thickened by the growth of a small-celled tissue, which presents all the appearances found in embryonic tissue which is undergoing fibroid development. This new-growth, in its earlier stages, usually contains new blood-vessels; but later the tissue con-

tracts, and the vessels become to a great extent obliterated. The alveolar cavities which are not obliterated are either empty, or contain exudation-products and a few epithelial cells.

When secondary to ordinary broncho-pneumonia, or to that induced by the inhalation of solid irritating particles, the new fibroid growth also originates principally from the alveolar walls. Here, however, in the earlier stages it is less uniform, and the peri-bronchial and interlobular connective tissues play a more prominent part in the process. The new peri-bronchial tissue invades the walls of the adjacent alveoli, and materially increases the fibrosis.

In the chronic pneumonia resulting from pleurisy, the change, as already stated, is sometimes more localised, consisting in the development of dense fibrous bands passing inwards from the thickened pleura. These are developed from the interlobular tissue. In other cases the fibrosis is more general.

In whichever of the pulmonary structures the new fibroid growth originates, all the connective tissue of the lung may become involved, as it increases, and the alveolar cavities may be completely obliterated. The new-growth, like that met with in the inflammatory indurations of other organs, although in the earlier stages of its development it may be richly cellular and contain new blood-vessels, tends gradually to become less cellular, denser, and more contractile. In its more advanced state it often consists either of closely packed wavy fibres, or more frequently of a dense homogeneous or obscurely fibrillated material, associated with which are a few small round or fusiform cells. Sometimes the new-growth is found richly cellular, even in the most advanced stages of the disease.

The macroscopical appearances of the lung vary with the extent of the fibroid change. In the earliest stages of the induration resulting from acute pneumonia, where there is merely a slight thickening of the walls of the alveoli, the consolidation very much resembles that of red or grey hepatisation. It differs in being firmer and less friable in consistence, and is somewhat less granular. In the later stages, and in all cases where the fibrosis is extensive and general, the appearances presented by the lung are very characteristic. The organ is diminished in size, dense, firm, fibrous, in parts almost cartilaginous in consistence. The cut surface is smooth; and the large amount of irregularly distributed black pigment usually present gives to it a peculiar grey, marbled appearance. Numerous dilated bronchi traverse it in all directions.

The bronchi are almost invariably found dilated in those portions of the lung where the induration is advanced. This dilatation is often very considerable, the dilated tubes forming cavities, which may occupy a large

portion of the indurated lung. The walls of the tubes are much thickened, and the mucous membrane is often ulcerated. This secondary inflammation and ulceration of the bronchi, which occurs especially in the dilated portions, is induced by the irritating and often putrid secretion which they contain. It may extend into and involve the indurated lung, and so lead to more or less excavation. The mucous membrane sometimes sloughs, and the gangrenous process may involve the lung. The large cavities so common in these lungs are in the main, however, dilated bronchi. *See BRONCHI, Diseases of.*

The pleura of the affected lung, except in the earliest stages of the disease, is much thickened and adherent.

*Site.*—Chronic pneumonia is in the majority of cases unilateral. The whole lung may be involved or only a portion. In the latter case the base is much more commonly affected than the apex. When it is due to the inhalation of irritating solid particles, both lungs are usually implicated.

*SYMPTOMS.*—In the earlier stages of chronic pneumonia the symptoms are often very obscure, and it is not uncommon to meet with advanced and extensive fibrosis in which the lung-affection must presumably have been of much longer duration than the symptoms accompanying it. In some few cases the symptoms are directly continuous with those of some more acute pulmonary inflammation—an acute or a broncho pneumonia. Under these circumstances, a prolongation of some of the phenomena of the original disease indicates the supervention of the pulmonary fibrosis. The pyrexia does not entirely disappear. There may be merely slight elevation of temperature towards evening, or the course of the fever may be very irregular. The cough usually persists, as does also some increase in the frequency of the respiration and pulse; and the patient, instead of improving, gradually loses strength and flesh. At the same time the physical signs of the pulmonary consolidation remain, and gradually give place to those of pulmonary induration. Where chronic pneumonia is secondary to pleurisy, a continuous sequence in the symptoms is even less commonly observed. When it is the result of the inhalation of irritating solid particles, the symptoms of bronchial catarrh are predominant.

When the fibrosis is fully established the symptoms are usually more pronounced. They vary considerably, however, with the extent of lung involved, the situation of the consolidation, and especially according to the presence or absence of bronchial catarrh. When bronchial catarrh is absent, and the disease is quiescent, a considerable area, or even the whole of the lung, may be involved without producing any marked pulmonary symptoms; and slight dyspnoea and cough,

with some general impairment of nutrition and failure of strength, may be almost the only phenomena present. Such quiescence and immunity from symptoms, however, although common in the course of the disease, is rarely observed over lengthened periods.

With the existence of catarrh of the bronchi, much more marked pulmonary symptoms are usually observable. Inflammation of the bronchi is especially favoured by their dilatation, and it is almost invariably present, to a greater or less extent, during the course of the disease. With it is usually associated activity of the indurating process. This dilatation of the bronchi, and secondary inflammation and ulceration of their walls, are most important factors in accounting for the symptoms. The course of the disease now often simulates that of chronic phthisis, but it is for the most part more chronic, less regularly progressive, and more frequently interrupted by periods of quiescence. The dyspnoea is now more marked, and cough becomes a troublesome symptom. The cough may be more or less constant, and it is usually attended by expectoration. Its characters vary, however, according to the situation and the extent of bronchial dilatation. When, as is most frequent, the lower lobe of the lung is involved, the secretion accumulates, and its removal by expectoration becomes exceedingly difficult. Under these circumstances the cough is often violent and paroxysmal. The patient may remain for several hours with but little or no cough, and then occurs a violent paroxysm, which results in the expectoration of large quantities of muco-purulent secretion. This violent paroxysmal cough and copious expectoration, occurring at long intervals, are exceedingly characteristic of bronchial dilatation in the lower portion of a lung. According to Niemeyer, the paroxysm occurs when the secretion which accumulates in the lower portions of the lung reaches and irritates the more healthy bronchi which retain their sensibility, the dilated tubes being so altered as to be completely insensible. The sputum may be simply puriform, but when there is much bronchial dilatation, owing to its accumulation and retention in the tubes, it often undergoes putrefaction, is of a greyish or greenish-black colour, and more or less fetid. This putrid secretion not only intensifies the inflammation in the bronchiectatic cavities where it originates, but is often conveyed by aspiration to other parts of the same, or the opposite lung, and so originates foci of broncho-pneumonia, which materially hasten the progress of the disease. Hæmoptysis is not infrequent, but it is usually small in quantity, and is in most cases due to ulceration of the bronchial walls.

Pyrexia is usually present to a greater or less extent in the course of chronic pneumonia.

The fever, however, is exceedingly irregular, and there are often long periods of perfect immunity. During the pyrexial periods the maximum evening temperature is rarely more than 101° or 102° F., and it may be only 100°. The morning temperature is often normal. The pyrexia appears in most cases to be due to inflammation and ulceration of the bronchi. It is not infrequently the result of a supervening tuberculosis.

With the progress of the disease the patient gradually emaciates; the fingers become clubbed; digestion is impaired, and diarrhoea is often present. Dropsy is a common symptom, although it is rarely extensive, and is, for the most part, confined to the lower extremities. It appears in most cases to be due to the anæmia and impeded pulmonary circulation. The pulmonary obstruction may also give rise to some enlargement of the right side of the heart, and cyanosis. Lardaceous disease of the viscera is occasionally met with. Death usually results from the general failure of strength, or from some intercurrent affection of the opposite lung.

*Physical signs.*—In the earliest stage of chronic pneumonia, when it is the result of a more acute pneumonic process, the physical signs are, in the main, those met with during the acute consolidation. It is the persistence of the signs of the pulmonary consolidation after the acute attack which indicates the possibility that the disease may become chronic. Dulness on percussion; increased vocal fremitus; bronchial breathing; and the existence of *râles*, which are larger, moister, and more metallic in quality than those of fine crepitation, are observable during this stage. When the induration is fully established, the physical signs are those of contraction and consolidation, with usually those of more or less dilatation of the bronchi, of a whole or a portion of the lung. The retraction is well-marked, and commonly affects the whole side, although when the lung is not universally involved it may be more limited. Expansion is exceedingly deficient, or completely absent. The heart is much displaced towards the affected side; the diaphragm and the abdominal viscera are drawn up; and the opposite lung encroaches considerably across the middle line in front. Percussion is hard, wooden, and high-pitched, sometimes more or less amphoric. The vocal fremitus is usually increased; and there is often bronchophony or pectoriloquy. The respiratory sounds will vary according to the extent of the bronchial dilatation and excavation, and the amount of secretion. They are for the most part bronchial; usually large and loud; and often distinctly cavernous. When there is much secretion in the dilated bronchi, high-pitched bubbling *râles* are heard, which are often amphoric and cavernous. These may be audible only after

cough. The opposite lung is usually enlarged, hyper-resonant, and the respiration exaggerated.

**DIAGNOSIS.**—The diagnosis of chronic pneumonia rests mainly on the physical signs. The diseases with which it is most liable to be confounded are chronic (tuberculous) phthisis, and retraction from pleurisy. In the most chronic forms of phthisis, where the fibrosis of the lung is considerable, the diagnosis from non-tuberculous consolidation may present some difficulty. This difficulty, however, rarely exists except in those quite exceptional cases in which the chronic pneumonia involves only the upper portions of the lung. Here the situation of the consolidation is very greatly in favour of its tuberculous nature. This probability is infinitely increased if the other lung be affected. In unilateral basic disease, and in induration of the whole of one lung, the other lung being healthy, the tuberculous nature of the disease is much less probable; although the occasional supervention of a tuberculosis in these cases should be borne in mind. An examination of the sputum for the bacillus of tubercle is usually the only means of settling the diagnosis. Disease of the larynx is in favour of the tuberculous, foetidity of the sputa of the non-tuberculous, nature of the consolidation.

The retraction resulting from chronic pleurisy may also be confounded with chronic pneumonia. Here, however, there are rarely physical signs of dilatation of the bronchi, respiration is weak and distant, and the vocal fremitus is diminished. The presence of abundant foetid sputa, of pyrexia, emaciation, &c., in chronic pneumonia will also in most cases render the diagnosis easy.

**PROGNOSIS.**—Chronic pneumonia, when it involves a considerable area of the lung, usually tends ultimately to terminate in death, although under favourable circumstances life may be prolonged for many years. When the disease is limited, and remains quiescent, the general health and duration of life may sometimes be but little affected. The most important element in the prognosis is the condition of the bronchi. The existence of bronchial dilatation, as evidenced by profuse, and often foetid, expectoration, is always unfavourable, as it not only weakens the patient, but is usually attended by extension of the induration, and ultimately leads, in the dilated tubes, to ulceration of the bronchial walls and surrounding tissue, and occasionally to gangrene. Pyrexia, as another evidence of inflammation of the bronchi and indurated lung, is likewise unfavourable, as is also hæmoptysis. The latter indicates deep ulceration, and it may in exceptional cases endanger life. The general condition of the patient must also be taken into account in making a prognosis. Failure of strength

and of digestive power, diarrhoea, and dropsy, are all of unfavourable augury.

**TREATMENT.**—In considering the treatment of chronic pneumonia, it is in the first place important to bear in mind that the usual origin of the disease is some more acute pulmonary inflammation. Hence the necessity for the most careful management and supervision of such inflammations in their later stages, with the object of procuring, if possible, a complete resolution of the pneumonic products.

When the fibrosis of the lung is established, it is hardly necessary to remark that the new-growth is incapable of removal, and by treatment we can only hope to influence the extension of the disease, and control the bronchial catarrh with which it is so frequently associated. The frequency and gravity of bronchial catarrh has been already insisted upon; and its management, in the majority of cases, constitutes by far the most important element in the treatment. Our object must be to prevent and control it; to promote expectoration, and to prevent that decomposition of the secretion which is so apt to result from its retention. Here the question of climate will necessarily present itself, and very much may usually be done by residence at some suitable station. One not subject to vicissitudes of temperature, and at the same time dry and moderately bracing, is most likely to be beneficial. The patient should be warmly clad, and everything should be done, by means of diet and medicine, to improve the general health, inasmuch as the better the state of nutrition, the less is the liability to bronchial inflammation. Cod-liver oil and iron are often useful for this purpose. If an attack of acute bronchial catarrh supervenes, it should be treated at once, and the importance of quickly controlling it should not be forgotten.

In the treatment of the more chronic catarrhal process which is so often associated with profuse secretion, much may usually be gained by the use of stimulating and antiseptic inhalations, of which creasote, eucalyptus, and carbolic acid are, perhaps, the most generally useful. These not only tend to diminish the amount of secretion, and to prevent its putrefaction, but induce coughing, and so assist in its evacuation. These substances may be administered internally with the same object. Counter-irritation, especially painting with iodine, appears sometimes to be serviceable. Gastric disturbance, diarrhoea, hæmoptysis, &c., must be treated, as they arise, on general principles.

In cases of localised basic disease, when other treatment fails, the question of paracentesis and artificial drainage of a large bronchiecatic cavity might be entertained.

**LUNGS, Inflation of.**—This term is used somewhat ambiguously. It is sometimes employed as synonymous with emphysema in its general sense. More correctly it has been limited to that condition in which the lungs are acutely and temporarily distended more or less with air, as from plugging of the bronchial tubes in some cases of bronchitis, a condition which is usually called 'acute emphysema.' It cannot be said to give rise in itself to any definite symptoms; but it can be made out by physical examination, the signs being those indicating excess of air in, and consequent distension of, the lungs.

When this condition exists, the aim in treatment should be to get rid of any obstruction leading to the imprisonment of the air, and to help the lungs in expelling it. It must be remembered that, even after a considerable degree of distension, the lungs may be able to return to their normal dimensions.

'Inflation' is also a term applied to intentional expansion of the lungs with air, as carried out in certain methods of artificial respiration.

FREDERICK T. ROBERTS.

**LUNGS, Malformations of.**—There are no malformations of the lungs which can be regarded as of much importance from a clinical point of view. As anatomical peculiarities, the shape of these organs, or the arrangement of their lobes, may be abnormal. In a case which came under the notice of the writer, one of the lungs was improperly developed and unexpanded, in connexion with the almost complete absence of one of the divisions of the pulmonary artery. The form of the lungs is frequently more or less altered as the result of various organic diseases of these organs.

FREDERICK T. ROBERTS.

**LUNGS, Malignant Disease of.**—**SYNON.**: Fr. *Carcinome du Poumon*; Ger. *Lungenkrebs*.

**DEFINITION.**—Malignant disease affecting the pulmonary tissues.

**ÆTIOLGY.**—Malignant disease of the lungs is of more frequent occurrence than was at one time supposed; but there are not sufficient trustworthy statistics to enable us to determine its relative frequency to other forms of thoracic organic disease. It has been met with in persons of all ages, from childhood to extreme old age; but the middle periods of life, from 20 to 60, are the most liable; and the two sexes are about equally obnoxious to the disease. As a primary disease, originating in the lungs, cancer is undoubtedly rare, though much less rare as first manifesting its presence in these organs, either by local or general symptoms. In by far the larger number of cases the disease in

the lungs is secondary to cancer in other parts; and in this way, with the exception of the liver, the lungs are more frequently implicated than any other internal organ. Thus, after the removal of an external cancer, pulmonary symptoms are among the most frequent and earliest indications that the disease has invaded internal organs.

**ANATOMICAL CHARACTERS.**—The right lung has been considered to be more frequently affected with malignant disease than the left. This, however, does not accord with the writer's experience. Of thirty-nine cases tabulated by him, the left lung was the principal seat in fourteen, and the right in nine only, whilst of the remainder either both lungs were affected, or the disease was confined to the mediastinum. Of the several varieties of cancer, encephaloid is by far the most common in the lungs; colloid and epithelioma are the rarest; and scirrhus holds a middle place. The intermediate varieties of these leading forms are also occasionally seen.

**SYMPTOMS AND DIAGNOSIS.**—In proceeding now to describe the various aspects under which these several varieties are presented to the clinical observer, their natural history, and diagnosis, it is not proposed to maintain any precise distinction between primary and secondary forms, nor to discuss the minute anatomy or general pathology of the several species, such questions having been considered in other parts of this work. The object of the writer is to treat the subject from a clinical point of view. It is important, however, to observe, *in limine*, that cancer may either commence in, or eventually implicate, any or all of the pulmonary textures; although undoubtedly both the primary localisation and the spread of the disease are influenced by the particular species. Both the early symptoms and the subsequent progress of the case will often be materially modified by the particular tissue that is mainly implicated. If the disease first manifests itself in the form of subpleural growths, both the early symptoms and the subsequent phenomena will differ from those which present themselves when the disease commences in the deeper tissues of the lungs. And it is observable that, when the disease commences as disseminated deposits in the lungs, these deposits are frequently most numerous in the vicinity of the pleura, so that this membrane is very early implicated, in many cases, when it is not the primary seat of disease.

For clinical purposes we cannot do better than divide intra-thoracic cancerous growths into three groups: 1. Where the disease is *disseminated* through the lungs, either in the form of isolated scattered nodules of varying magnitude, or as spreading along the mucous membrane and sides of the bronchial tubes and vessels, through a greater or less extent of the lung. 2. Where

the growth is more *localised*, occurring, for the most part, in large masses. 3. *Mediastinal tumours*, involving the various structures at the root of the lungs, and eventually giving rise to symptoms of pressure and distress of a more or less serious character.

**1. Disseminated Malignant Disease.**—In the disseminated form of pulmonary cancer the symptoms vary considerably, according to the seat of the growths. When the pleural surface is chiefly implicated, both the symptoms and the physical signs are essentially those of pleurisy, though the degree of febrile disturbance is usually very slight, and but little or perhaps no false membrane may be effused. But the more prominent symptoms are sometimes singularly localised even when the pleural surface is extensively implicated. The exudation is generally clear serum, or serum mixed with blood; and it may have a greenish or brown colour, but is rarely purulent or even semi-purulent. As the effusion increases in amount, the ordinary consequences of compression of the lung ensue, but dilatation of the side is generally much less marked than in simple pleurisy with effusion, in consequence of there being less giving way of the intercostals. The fluid generally returns speedily after paracentesis.

When the mucous and submucous membranes of the bronchi and the surrounding connective tissue are the chief seats of the disease, the physical signs are those of bronchial irritation and emphysema, which, however, may, for some time, be quite disproportionate to the dyspnoea and other symptoms of ordinary bronchitis. The expectoration is, for the most part, scanty, and either simply mucous or mixed with blood; or small bronchial casts may be expectorated. Examination with the microscope will occasionally reveal characteristic cancer-cells, and the absence of tubercle bacilli may decide, in an otherwise doubtful case, that it is not phthisis. The resonance of the chest may remain normal, when auscultation proves that there is a diminution of air entering the lung. But there will not be the hyper-resonance of emphysema. Wheezing and dry and moist sounds vary much with the amount of constriction of the tubes, and the amount and character of the secretion. But in advanced cases of this kind, by the spread of the disease along the interlobular septa and through the lung, its condition becomes similar to that of a cirrhotic lung, and the clinical aspects of the case may be greatly altered. Perhaps the most characteristic symptom of this class of cases is dyspnoea insidiously increasing, especially on exertion, without corresponding symptoms of either congestion of lung or compression. Of the general symptoms, the most characteristic is that of steadily advancing debility, which is common to other forms of cancer.

And it is from asthenia, or from general cachexia, that the patient usually dies, before much or any disintegration of tissue takes place. It is the scirrhus variety of cancer which most often thus follows and implicates the bronchi.

In the disseminated form, characterised by numerous masses, varying from the size of a millet-seed to that of a pea, scattered throughout the lungs, the clinical phenomena, both local and general, may very closely simulate those of tubercle, with recurrent attacks of bronchial irritation and congestion, and febrile disturbance. But as a rule, to which, however, some remarkable exceptions have been met with, there is little, if any, increase of temperature, nor is there the quickened breath and frequent dry cough of tubercle. The dyspnoea is chiefly on exertion, and seems more due to feeble circulation and general debility than to either pulmonary disease or febrile disturbance. Indeed, the absence of local signs of inflammation, or symptoms of functional disturbance, is frequently remarkable. Signs of bronchial irritation in some of these cases have been early noted and prominent symptoms; in other cases they have been slight and variable. The apices of the lungs, though often implicated, are not specially and early invaded as in tubercle, but rather the bases. If the cancerous growths are rapidly developed and extensively distributed through the lungs, both the signs and general symptoms become greatly modified, and the case proves speedily fatal. The similarity to acute tuberculosis is sometimes very close, especially in those instances in which there is marked febrile disturbance, and recurring slight hæmoptysis.

**2. Localised Malignant Disease.**—The second class of cases of malignant disease of the lungs, in which the disease manifests itself in the form of isolated masses of larger size, is the most common. There may be one such tumour or several, at first assuming a rounded form, but, as they gradually invade the lung, acquiring an indefinite shape, and involving a large portion or even the whole of the lung. Such tumours, being most frequently of the encephaloid varieties of malignant disease, often grow rapidly, and as rapidly disintegrate, giving rise to hæmorrhage and destruction, not only of the mass itself, but also of the surrounding tissues. In this way vomicæ may be formed, or portions of lung may become gangrenous.

The symptoms and progress of these cases necessarily vary much. If the growth or growths have attained any considerable size, there is dulness on percussion, and an absence of respiratory murmur over the affected portion of lung. The presence and character of other physical signs depend very much on the patency or occlusion of the bronchi. When, as is often the case, these are

completely occluded, nothing whatever may be detected on auscultation, and all vocal fremitus may be absent; the implicated portion being completely shut off from the rest of the lung, and from all communication with the trachea. If, however, the bronchi remain patent, or—as the result of breaking down of the cancerous mass—if communication with the larger bronchi has been re-established, we have evidence of abundant secretion, and the ordinary phenomena associated with a cavity. In such circumstances microscopic examination of the expectorated matters may give decisive evidence of the nature of the case. On the other hand, before any such considerations have arisen, we may have in the case of a large tumour involving the whole or the greater part of one lung auscultatory signs which are with difficulty distinguishable from those of extensive pleuritic effusion. In other instances, where the portion of lung implicated in the cancerous growth is limited and well-defined, the physical signs may so closely resemble those of phthisis as to lead astray the most expert. Thus we may have limited dulness on percussion, with absence of respiration; followed by signs of surrounding irritation, slight hæmoptysis, cough, expectoration, and indications of a cavity. In some rare instances there have been limited flattening and altered form of the chest-walls, such as characterise chronic phthisis. Copious hæmoptysis, except in connexion with extensive destruction of lung-tissue, is not common in cancer of the lung. But there may be extensive sanguineous effusion into the pleural cavity, increasing the difficulty of diagnosis. The diagnosis in those instances, where the local signs closely resemble phthisis, must be based mainly on the constitutional symptoms and the history of the case, together with careful microscopic examination of the sputa.

There is considerable difference as to the progress and mode of termination in the whole of the class of cases now under consideration. Long before the local changes have advanced far enough to admit of a decisive diagnosis, the patient may die from rapidly increasing debility and emaciation, with more or less of hectic fever, and even typhoid phenomena; or he may be carried off by rapidly occurring pleuritic effusion. In other instances, cancer developing in other organs is the cause of death. Indeed, in a large proportion of cases the manifestation of malignant growths in the neck, the axilla, or other parts, places beyond question the nature of the case. So long as the growth is confined to the substance of the lung, and does not implicate the nerve-trunks and larger vessels, there is usually little pain, paroxysmal dyspnoea, or disturbance of the heart's action, excepting such as may be due to feebleness of muscular power. Nor is there generally any external

œdema, or distension of the superficial veins. The reverse of all this characterises the cases in which the mediastinum and the roots of the lungs become involved.

**3. Mediastinal Tumours.**—This form of malignant disease of the lungs is described under a separate heading. See **MEDIASTINUM, Diseases of.**

**PROGNOSIS AND TREATMENT.**—The subject of the prognosis and treatment of malignant disease of the lungs in its various forms will be more conveniently discussed in the article **MEDIASTINUM, Diseases of.**

J. RISDON BENNETT.

**LUNGS, Malpositions of.**—Among malpositions may be regarded those conditions in which the lung is contracted more or less within its normal limits; or, on the other hand, distended so as to pass beyond its usual boundaries. One or both organs may be thus affected. These alterations may result either from more or less diminution of the amount of air in the lungs, as in cases of compression or collapse; from excess of the same, as in emphysema and hypertrophy; or from diseases which affect their structure. The lung may also be displaced by the pressure of tumours, in addition to being compressed. The most important malposition of the lung, however, is that known as *hernia*, in which a portion of the organ projects into the neck, or through some part of the chest-walls, or through the diaphragm into the abdominal cavity. If the hernia passes towards the surface of the body, it may be made out clinically, being indicated by a soft and compressible swelling, localised, resonant on percussion, and rendered more prominent by a cough. Pulmonary symptoms might possibly be present. It is practically impossible to detect a hernia of the lungs through the diaphragm.

FREDERICK T. ROBERTS.

**LUNGS, Morbid Growths in.**—The formations in the lungs which belong to the class of morbid growths may be thus enumerated, in the order of their importance: 1. *Tubercle.* 2. *Cancer.* 3. *Syphilitic gummata.* 4. *Hydatids.* 5. *Rare formations,* such as sarcoma, enchondroma, osteoid and myeloid growths, hæmatoma, lymphatic formations, actinomycosis, &c. Most of these are discussed under their appropriate headings, and it is unnecessary to allude to them any further here. Those belonging to the last group are usually rather of pathological interest than of clinical importance, with the exception of actinomycosis, as they rarely give rise to any local symptoms or physical signs during life, and are merely discovered, as a rule, at the *post-mortem* examination. It is a question how far some of these growths are to be regarded as being of a malignant nature. In some cases they are secondary

to similar growths elsewhere, or the lung may be involved by extension. Lymphatic formations in the lungs are sometimes observed in cases of Hodgkin's disease.

EFFECTS.—It will be useful to indicate the effects, if any, which morbid growths may produce in connexion with the lungs.

1. The lung-tissues may merely be more or less displaced and compressed; or, in course of time, they become absorbed or atrophied, in proportion as the growth progresses. 2. The distribution of air in the lungs may be modified by the mere presence of a growth, so that in one part it is in excess, and in another part deficient. 3. Similarly, the circulation of blood may be disturbed, leading to congestion in one part, and anæmia in another. 4. Morbid formations are very liable to cause local irritation. Hence they may induce bronchial congestion and catarrh, localised acute pneumonia and its consequences, or chronic interstitial pneumonia, which may lead to the formation of a fibrous capsule around a growth. 5. Certain formations are liable to undergo degenerative and destructive processes, which will probably involve the pulmonary tissues. In this way they originate ulcers or cavities, and may give rise to morbid products, which are not only injurious to the lungs, but also infect distant parts to which they may be conveyed. After destruction reparative processes not infrequently take place, with loss, however, of the involved portions of the lung-structures. It must be remarked here that some morbid growths seem to become infiltrated through the pulmonary tissues without destroying them; and under appropriate treatment the growth is absorbed, leaving the involved portion of the lung intact. This applies especially to some cases of syphilitic infiltration. 6. Growths in the lungs sometimes extend beyond these organs, so as to interfere with neighbouring structures, causing irritation, pressure, or destructive effects. Thus, local pleurisy, pressure on vessels or nerves, destruction of bones, and other consequences may ensue. In short, the growths become then practically intra-thoracic tumours, and produce similar effects.

SYMPTOMS.—What has been stated as to the effects of morbid growths in the lungs will readily explain the clinical signs which they tend to originate. They may be of such little consequence that they produce no sign whatever during life, not interfering in any way with the respiratory functions, or being capable of detection by physical examination. Indeed, some formations may invade the lungs to a considerable extent so insidiously that no evident symptoms are induced. The writer has known cases in which the lungs were extensively implicated in secondary cancer without any symptoms, except some feeling of shortness of breath on exertion. Usually, however, various degrees

and combinations of the ordinary pulmonary symptoms may be anticipated—namely, pain in some part of the chest, cough, expectoration, the sputum sometimes containing fragments of the growth, hæmoptysis, and disorders of breathing of various kinds. Pressure-symptoms in connexion with other structures are induced in some cases. Physical examination may detect the disease when there are no symptoms; or these may co-exist with physical signs, which reveal the presence either of the morbid formation itself—such as alteration in the shape and size of the chest, deficient expansion, dullness, bronchial or other abnormal breath sounds, modified vocal fremitus and resonance; of its effects on the lungs; of the formation of cavities; or of its interference with neighbouring parts. The particulars relating to these points are discussed in other articles. Definite general symptoms are associated with many forms of morbid growth in the lungs.

TREATMENT.—The principles of treatment of morbid growths in the lungs are, first, to get rid of them, if possible, by medicinal means, as in the case of syphilis; secondly, to treat their effects; thirdly, to treat local symptoms which may arise; and fourthly, to treat the general symptoms.

FREDERICK T. ROBERTS.

**LUNGS, ŒDEMA OF.**—SYNON.: Fr. *Œdème du Poumon*; Ger. *Lungenödem*.

DEFINITION.—Infiltration of the pulmonary tissue with serous fluid.

The serous fluid is effused from the pulmonary capillaries into the pulmonary textures, and into the alveolar and bronchial spaces.

ÆTIOLOGY.—The causes of this exudation are manifold, but of two sorts:—(a) *Disordered circulation*: (1) active congestion, attendant upon inflammatory conditions of the lungs and bronchi; (2) passive congestion; (3) mechanical congestion—in heart-disease, emphysema, or pressure upon the pulmonary veins; (4) want of tone of vessels after inflammatory conditions, as pneumonia or bronchitis, or pressure upon the vagus nerve or pulmonary plexus; (5) afflux of blood to the lungs in croup, and during the asthmatic paroxysms determined by the ineffectual efforts at inspiration. (b) *Morbid conditions of the blood*: in albuminuria, and to a less degree in other diseases in which the condition of the blood is altered or impaired—for example, scurvy, purpura, anæmia, hydræmia—the lungs partaking of the general disposition to dropsy.

ANATOMICAL CHARACTERS.—In cases of œdema pulmonum, the lungs are usually large, filling the thoracic cavity, and sometimes indented by the ribs. They are heavy; their pleural surfaces are wet; and the pleural cavities contain an excess of serum. Both

lungs are as a rule affected, their lower and most dependent portions chiefly; and one lung, on the side to which the patient has last inclined, is more highly œdematous than the other. The higher the degree of œdema, the less crepitant the lung, and the more distinctly the surface pits on pressure. A portion cut from a simply œdematous lung will, however, almost always float in water; but at the base of the lung there is usually some collapse in addition to the œdema, and a portion removed therefrom sinks. On section, the lung exudes abundant thin serum, and more or less frothy fluid, with which the bronchial tubes are also occupied. On first making a section, the succulent tissue will break down easily under the finger; but, after the excess of fluid has been squeezed out, the lung feels toughened. The fluid is almost entirely confined to the alveolar spaces and parenchyma proper, as distinguished from the interlobular septa of the lung. Œdema may be found at any portion of the lung—at the apex, for instance, determined there by the inflammatory process. The transition between œdema and inflammatory consolidation is very gradual. Œdema is also very apt to pass into, or to be complicated with, a certain degree of inflammation. The degree of friability, and of compressibility, and the application of the water test, are the readiest methods of distinguishing between the two. If a portion of œdematous lung be examined under the microscope, the alveoli are found to contain more or less numerous large granular cells, but these are never so numerous as to occupy entirely the alveoli.

**SYMPTOMS.**—The symptoms of œdema of the lungs are—in addition to those of the disease which has produced it—dyspnœa, which may amount to orthopnœa; troublesome ‘retching’ cough; and difficult, yet tolerably abundant, frothy, serous expectoration. The percussion-note is deadened at both bases, although the dulness is usually more extensive at one base than the other; the vocal fremitus is diminished; the respiratory murmur is enfeebled or lost; and a fine bubbling crepitation is heard.

**DIAGNOSIS.**—The diagnosis of pulmonary œdema is not usually difficult. The absence of pleuritic pains and of fever, and the double-sidedness of the disease, together with the absence of any true bronchial breathing or ægophony, will exclude pneumonia or pleurisy. The presence of dulness will also distinguish the condition from simple capillary bronchitis, with a certain degree of which, however, it is often combined. The general condition of the patient, and the presence or absence of those diseases or circumstances which are known to produce œdema of the lungs, must be carefully taken into consideration. If, for instance, after an asthmatic paroxysm we hear some fine

bubbling *râles* over the bases of the lungs, and find the patient expectorating an unusual quantity of frothy serous fluid, we may suspect pulmonary œdema rather than bronchitis.

**PROGNOSIS.**—The prognosis in œdema of the lungs depends mainly upon the general or local conditions with which it is associated. It is of very grave purport in chronic Bright’s disease, or in heart-disease. It is also a grave complication in chronic bronchitis, showing failure of heart-power. It is, however, often a transient and unimportant condition when it succeeds to acute chest-affections, as pneumonia or bronchitis, or to asthma. As a complication of acute chest-affections, it is rarely recognised clinically.

**TREATMENT.**—The treatment of pulmonary œdema is, in all important cases, derivative. Poulices are to be applied to the chest, with sufficient mustard to produce redness. Dry-cupping will often give great relief. Blisters should be avoided. Watery purgatives should be administered, according to the strength of the patient. Diuretics are useful in some cases, especially the vegetable diuretics, such as digitalis, juniper, and scopolium, as also spirit of nitrous ether; and the same is to be said of diaphoretics—for example, acetate of ammonium, warmth, hot-air baths. Moderate stimulation and support must be kept up. Kidney- or heart-disease, if present, will mainly determine the exact treatment. If there be failure of cardiac power, ether, ammonia, and alcoholic stimulants are required; and if the heart’s action continues hurried or irregular, digitalis is especially indicated. When we suspect a loss of tone of vessels, as after bronchitis or pneumonia and in anæmic states, perchloride of iron with some mineral acid is to be recommended.

In all cases rest in bed or on a couch is necessary. R. DOUGLAS POWELL.

**LUNG, Perforation of.**—**SYNON.**: The term *pneumothorax* is almost equivalent.

**DEFINITION.**—The formation of an opening through the pulmonary pleura, communicating with the interior of the lung.

**ÆTIOLGY.**—Perforation of the lung may arise in many ways. Its causes may be classified under the four following headings:

1. *Penetrating wounds*; for example, gunshot wound, punctured wound, or laceration by a broken rib.

2. *Rupture of the lung*; as sometimes occurs during violent expiratory efforts, e.g. in whooping-cough, parturition, &c.

3. *Diseases affecting the pleura or neighbouring structures*; such as empyema, hepatic abscess or hydatid, or suppuration of the bronchial glands.

4. *Disease affecting the lung itself*; for instance, phthisis, emphysema, gangrene, hydatids, or cancer.

Of all the causes of perforation of the lung,

phthisis is infinitely the most common. It is the rule in phthisis for pleuritic adhesions to form *pari passu* with the pulmonary lesion, and these adhesions are usually very firm and difficult to break down. In neither respect, however, does this rule always hold good. In some rare cases in the earliest stage of the disease a small tubercular nodule situated immediately under the pleura softens, and the pleura gives way. Again, at any stage of the disease an outlying tubercular mass, situated below the point to which the pleural adhesions have extended, may soften and rupture into the pleural cavity.

In the more acute pneumonic varieties of phthisis there is often a singular indisposition to the formation of pleural adhesions. The pulmonary pleura in such cases becomes covered with a thin, smooth, translucent layer of lymph, shining through which can be seen at several points opaque yellow spots. These spots are found to correspond with underlying masses of softened cheesy material, by which the pleura has been undermined and deprived of its vascular supply. Pneumothorax has its most frequent origin in rupture of the pleura at one of these yellow points.

Finally, sinuses are sometimes found leading from old cavities within the lung to the pleural surface. Occasionally these sinuses, the pleura being adherent, penetrate through the thoracic wall and point externally. In other cases, of which the writer has seen two examples, they may open into the opposite pleural cavity.

**ANATOMICAL CHARACTERS.**—The affected lung is in all cases collapsed, and in cases of old standing may be so completely so, and covered by such thick layers of lymph, as to be found only with difficulty. The opening may have closed. It is sometimes difficult to discern. It may consist of a small slit, communicating with a cavity by a slanting sinus, so as to form a complete valve; or it may be of considerable size, and communicate widely with a cavity or bronchus. All degrees of patency between these two extremes occur. The position of the opening is very variable; it is most commonly situated somewhere on the lateral or convex side of the lung. The rupture is almost always into the pleural cavity on the same side. It may, however, take place into the opposite pleural cavity, through the mediastinal fold of pleura. The pleura is inflamed, and covered with lymph; and its cavity contains air and a greater or less quantity of purulent fluid. The heart is displaced, unless in some rare case it be held by a strong adhesion. Some years ago the writer tested the degree of air-pressure present in ten cases of pneumothorax by means of a water-pressure gauge. In two cases it was *nil*; in one case it was equal to 1·25 inch; in two cases 2 inches; in one case 3·75 inches; in two cases 4 inches;

in one case 5·3 inches; and in a double case it equalled 3·5 inches in one pleura, and 2·7 in the other. The gas effused approximates in composition to that of expired air, containing from 8 to 16 per cent. of carbonic acid. Sometimes sulphuretted hydrogen also is found in foetid cases.

**SYMPTOMS AND SIGNS.**—The symptoms and signs of perforation of the pleura are those of pneumothorax, followed by hydro- or pyo-pneumothorax. At the moment of attack sudden acute pain is felt in the chest, at the seat of rupture, and is immediately followed by great dyspnoea and shock. In a well-marked case the expression of face is peculiarly agonised and terror-stricken; the extremities are cold; damp sweats break out; the pulse is quick and small; and the respirations are exceedingly rapid. The position of the patient is that of orthopnoea, with an inclination forwards, and to the sound side; it is, however, frequently changed in the endeavour to gain breath. The voice is feeble and whispering. The urgency of the shock and dyspnoea depends upon the amount of useful lung suddenly disabled. If the patient survive the attack, after two or three days fever of a hectic character, with sweats, supervenes. In some cases, however, the symptoms of pneumothorax come on very insidiously.

**Physical signs.**—The physical signs are very characteristic. There is enlargement of the side affected, and effacement or bulging of the intercostal spaces. The heart is displaced towards the sound side. The percussion-note is hyper-resonant or tympanitic over the side affected, except where (at the apex) the lung may perhaps be still adherent; and on auscultation either no respiration at all is audible, or amphoric breathing of a peculiar character may be heard at one or more points, sometimes accompanied with the characteristic metallic tinkle. A peculiar metallic echo is heard if the patient coughs. If, whilst the ear is applied, a coin placed on the diseased side is struck with another coin, a characteristic bell-note is heard. The vocal fremitus is diminished or lost. At a later stage, when more or less effusion has taken place, the signs of air and fluid in the pleural cavity present themselves, namely, dulness below and hyper-resonance above—in varying proportions, and shifting in relative position with the posture of the patient. If the amount of fluid be moderate, a splash or succussion sound may be elicited. This sound may be audible to the ear applied to the chest, or to bystanders. If the fluid effusion be considerable, intercostal fluctuation may be felt; and this fluctuation gives to the finger, on percussion at the level of junction of air and fluid, a peculiar sensation of thrill. The position usually assumed by the patient now is with the head raised, and leaning towards the diseased side.

**DIAGNOSIS.**—The diagnosis of perforation of the lung is to be made from other diseases; and also with respect to the probable nature of the opening, and the degree of pressure present. If the three essential signs of pneumothorax be remembered, namely, displacement of heart, tympanitic percussion-note, and either absence of respiration or amphoric breathing, there can scarcely be any difficulty in making the diagnosis. It cannot be confounded with a bilateral disease like emphysema. The shifting resonance and dulness, the succussion-splash, with perhaps metallic tinkle and amphoric breath-sound, are signs abundantly sufficient to distinguish hydro-pneumothorax from ordinary empyema. It is sometimes very difficult to distinguish between a *localised* pneumothorax and a large thin-walled cavity in the lung, the signs being almost identical. Respecting the nature of the opening, whether valvular or free, careful auscultation will usually gain the desired information. If amphoric breathing be well-marked, it may be assumed that the opening is a free and tolerably direct one; if, on the other hand, no respiratory sound be audible, the communication with the pleura is indirect and more or less completely valvular. In the latter case the pressure-symptoms become more urgent.

**PROGNOSIS.**—Of course the prognosis in every case of tubercular pneumothorax is necessarily very grave, but by no means equally grave in all cases. The following considerations will guide to a correct prognosis: (a) *Nature of opening.* If the communication with the pleura be valvular, signified by the entire absence of breath-sound, and the increasing urgency of dyspnoea, the patient will die in a few hours unless relieved by paracentesis. (b) *State of the opposite lung.* If the effusion of air have occurred on the side least affected by previous disease, the case is correspondingly hopeless. If, on the other hand, we know that the lung now collapsed was previously much diseased, and if the other lung be but little affected, the duration of life may not be greatly shortened by the accident. Life is then gradually extinguished by hectic fever, and progressive disease in the opposite lung. It is by no means impossible, and probably happens more frequently than is supposed, that the opening in the pleura may close, the air become absorbed, and the case converted into one of simple empyema. In pneumothorax arising from accidental wound or injury to the lung, the prognosis depends upon the visceral injury. The air in the pleura is absorbed with considerable readiness.

**TREATMENT.**—In all cases in which death is threatened by asphyxia, in consequence of air accumulating in the pleura, paracentesis with a fine trocar must be performed. This will in such cases give great relief, and may be repeated if necessary. There is a ten-

dency for an opening, at first completely valvular, to become at a later period more patent, or possibly to close, so that it is better to operate when necessary with a fine trocar than to make a permanent opening. Rest to the affected side should be secured, as far as possible, by the application of a broad piece of strapping extending round the side to beyond the middle line in front and behind. The shock and dyspnoea are best treated by opium in repeated small doses. Stimulants may also be necessary, but opium is far more useful.

R. DOUGLAS POWELL.

**LUNG, Rupture of.**—Rupture of the lung is an extremely rare occurrence. Cases of so-called rupture of the lung from external violence are, for the most part, really produced by perforation or laceration of the pleura by a fractured rib. It is said that rupture of the lung may occur in whooping-cough. See LUNG, Perforation of.

**LUNGS, Syphilitic Disease of.**—There can be no doubt that syphilis does sometimes originate specific lesions in the lungs, though much less frequently than in most of the other viscera. They are generally only met with in advanced cases of acquired syphilis, when the signs of the disease are markedly developed in other parts. Occasionally the lungs are involved in congenital syphilis. The presence of a tubercular or scrofulous diathesis has been supposed to predispose to the implication of the lungs in syphilitic disease.

**ANATOMICAL CHARACTERS.**—*Gummata* constitute the most certain and unquestionable lesions of a syphilitic nature in the lungs, but they are rare. When present, they vary in number from one to many. In the latter case they are disseminated, but are stated to have a predilection for the deeper parts of the organs. In size these growths usually vary from that of a pea to a walnut, but may reach the dimensions of a large egg. They are generally well-defined, rounded in shape, and often surrounded with a fibrous capsule. In their early condition gummata in the lungs appear on section greyish or brownish-red, homogeneous, firm, and dryish in consistence. Subsequently they tend to degenerate, becoming more or less caseous, yellow, and less consistent; and they may even break down in the centre, so as to form cavities. The structure of these gummata is found on microscopical examination to be made up of imperfect fibres, abortive nuclei, and a few fibre-cells, infiltrating the pulmonary tissues, and thickening the alveoli. Afterwards these are mixed with granular matter and other products of degeneration and disintegration.

There seems every reason to believe that a variety of *chronic interstitial pneumonia* is in exceptional instances due to syphilis. The result is a fibroid infiltration of the pul-

monary tissue, which in its general and microscopic characters cannot be distinguished from a similar condition due to interstitial pneumonia from other causes, but the new tissue is said to be more vascular in its early stages. The affected parts are much indurated; and any bronchi which are implicated tend to become more or less dilated. The morbid condition may be distributed in various parts of the lungs, but appears to have a preference for their bases and the vicinity of their roots. It frequently originates at the surface, and penetrates thence into the interior of the lungs in the form of fibrous bands, the pleura being generally thickened and adherent, and superficial puckering and depressions being visible. In other instances the new-growth commences around gummata; or from a chronic contracting peri-bronchitis, associated with ulcerative inflammation (Pye-Smith). Dr. Green states that it originates mainly around the small interlobular blood-vessels. Syphilitic fibroid infiltration has no tendency to caseation; but it may become the seat of ulceration or gangrene.

In connexion with congenital syphilis, a peculiar condition has been described as affecting the lungs in new-born or very young infants, under various names, such as *syphilitic pneumonia*, *white hepatisation*, and *epithelioma of the lungs*. It assumes a more or less diffuse or infiltrated arrangement, but is of variable extent, and may involve one or both organs. One lung may be affected throughout, while the other is quite free from disease. The more obvious characters are as follows: The pleura is usually unaffected. The lung is enlarged, and may be in a state of full expansion, so that its surface is marked by the ribs; it feels remarkably heavy; and at the seat of the disease is dense, firm, hard, and usually resistant, not breaking down under pressure. On section it presents a white or yellowish-white colour, being more or less bloodless; is uniform and smooth; and little or no fluid can be expressed or scraped from the cut surface. Careful examination reveals minute bands of fibrous tissue running in all directions. Microscopically the change seems to consist mainly in thickening of the alveolar walls and minute bronchi, due to an imperfectly fibrillated and nucleated tissue, which undergoes degenerative changes. Most observers further describe an increase in the epithelial cells, which fill the air-vesicles and minute air-tubes; but Wagner denies this. The vessels also become thickened and ultimately obliterated.

It may be remarked that the bronchial tubes or their divisions may be affected with syphilitic disease; their submucous tissue, or occasionally their deeper structures, becoming infiltrated with a fibro-nuclear growth. Ulceration is likely to take place, followed by cicatrisation, leading to thickening of

their walls, with narrowing or even complete closure of their channel.

**SYMPTOMS.**—In the majority of cases syphilitic lesions in the lungs have only been discovered after death, no symptoms having occurred during life pointing to these organs; or they having been obscured by symptoms affecting other parts. In a case of recognised constitutional syphilis, attention should be paid to the lungs as well as to other organs, and it would be advisable to examine them from time to time, as physical signs might occur without any obvious symptoms to attract the patient's attention. If pulmonary symptoms, such as local pain, shortness of breath, cough, expectoration, or hæmoptysis, should arise in a person undoubtedly syphilitic, or who had had syphilis, the possibility of the lungs being affected should specially be borne in mind. Hæmoptysis at an early period is said to be of diagnostic importance. Physical examination might possibly reveal the presence of gummata, as evidenced by localised dulness, bronchial breathing, increased vocal fremitus and resonance, and other signs of consolidation. The most significant signs, however, are those indicating marked induration of the lung from fibroid infiltration, especially if unilateral, and confined to the base or middle portion of the organ. In course of time signs of cavities might become evident, due to breaking-down of gummata, or to dilated bronchi. The general symptoms are those of constitutional syphilis, combined with those of phthisis. There is but little or no pyrexia accompanying the pulmonary lesions; and the progress of the case is essentially chronic. The effects of treatment may be of peculiar significance in the diagnosis of syphilitic disease of the lungs. If such symptoms and physical signs connected with these organs as have been indicated above should disappear under the use of anti-syphilitic remedies, a diagnosis of this disease might fairly be made. The absence of tubercle bacilli is considered an important element in distinguishing syphilitic disease from tubercular phthisis.

**TREATMENT.**—If syphilitic disease of the lungs be recognised or suspected, the appropriate treatment in most cases is to administer iodide of potassium freely and continuously. In some cases a mercurial course of treatment answers best; or perchloride of mercury might be combined with the iodide. It may be necessary to employ internal remedies or local applications for the relief of pulmonary symptoms. Cod-liver oil and tonics may be given with advantage for the amelioration of the general condition, in cases where such medicines are needed. The usual measures for the treatment of phthisical cases, as regards diet, hygiene, climate, &c., must of course be duly recognised, and adopted in suitable cases.

FREDERICK T. ROBERTS.

**LUNGS, Tuberculosis of.**—Tubercle is the most important morbid growth affecting the lung, but it is by no means a settled point what should be included under this term. Many pathologists only recognise as tubercle the so-called grey granulations; others regard the various masses and infiltrations noticed in cases of phthisis as of this nature, and they look upon tubercle in these organs as divisible into grey and yellow varieties, and arranged either in the form of granulations or as an infiltration. It is unnecessary in this place to discuss this subject further, as it is considered fully in other appropriate articles. See PHTHISIS; and TUBERCULOSIS.

FREDERICK T. ROBERTS.

**LUPUS ERYTHEMATOSUS** (*lupus*, a wolf, or a rodent disease; and *erythematosis*, related to erythema).—SYNON.: *Seborrhœa Congestiva*.

**DEFINITION.**—A chronic hyperæmia of the skin, attended by a new-cell-growth, followed by interstitial absorption, and ending in scar.

**ETIOLOGY.**—Women are more liable to lupus erythematosus than men. It is rare before puberty, and most often begins between the ages of 20 and 45 years; but no age of adult life up to 60 or 70 is exempt. Weakly persons are thought by some to be most liable, but the complaint may attack perfectly healthy people—at any rate there is no intimate connexion with any other special disease. An imperfect circulation, such as evidences itself by congestion of parts which are at a disadvantage, *e.g.* the nose or ears, is probably a causal factor; any cause which leads to temporary congestion, such as exposure to heat or cold, may act as a determinant. Lupus erythematosus is not hereditary. It attacks all classes of society.

**ANATOMICAL CHARACTERS.**—The capillaries of the cutis are dilated, and a small-celled new-growth develops in it, close under the epidermis, around the orifices of the sebaceous and hair follicles and the sweat-ducts. The cell-growth has lately been said to commence deeper down in the substance of the skin. There is an increased secretion of sebum; and crusts of epidermis and fatty matter form. The terminal scar is due to destruction of normal tissue by the new-growth; partial fatty degeneration and absorption of the latter; and conversion of the remainder into permanent connective tissue.

**SYMPTOMS.**—Lupus erythematosus most often attacks the face. Small patches of well-defined crimson or purplish redness form on the skin of the cheeks or nose, and remain for a long period unaltered; they then become covered in parts with thin, firmly adherent, dirty-looking crusts, which if removed are found to send processes into the dilated mouths of the sebaceous glands. In the final stage, the redness gives place to

a very superficial whitish scar. Confluence of several small patches gives rise to irregular patterns. In some cases it is impossible to recognise anything more than persistent redness due to dilated capillaries, and very superficial scarring where the disease is undergoing involution. When both cheeks are attacked at once, the patches tend to unite across the nose in the shape of a bat's wing or of a butterfly. The scalp often suffers, and there is permanent loss of hair. Other seats of lupus erythematosus are the ears, eyelids, lips, and backs of the hands; where it may closely simulate chilblains at first. No part of the body is exempt. There is no pain, or ulceration, but sometimes slight itching. Exposure to cold winds or to great heat aggravates the disease. In Germany an acute form has been observed, in which the eruption quickly covers a large part of the body; is attended with fever and prostration; and may end in death. The writer has seen such a case.

**COMPLICATIONS.**—Recurrent erysipelas has been noticed more often in lupus erythematosus than in lupus vulgaris.

**DIAGNOSIS.**—This disease is to be diagnosed from lupus vulgaris, by its commencement after puberty; by its very superficial character; and by the sebaceous crusts. From eczema, which it sometimes resembles at first sight, lupus erythematosus may be distinguished by the history, chronicity, trifling itching, and the presence of scars.

**PROGNOSIS.**—Lupus erythematosus is very chronic, and may last for years. Permanent cure is very doubtful, on account of the tendency to relapse. Except in the acute form, which is rare, there is no danger to life.

**TREATMENT.**—In the treatment of lupus erythematosus the first indication is to rectify any derangement of the general health. There is no specific internal remedy. Milk and cod-liver oil improve the nutrition. Externally, mild caustics suit some cases best. Mercury plaster should always be tried, spread thickly on linen, with enough oil of turpentine to make it soft, and applied every night for a long period. The liniment or tincture of iodine can be painted on until slight inflammation is set up, and repeated after an interval; or spirit of soap (℞ Saponis mollis ʒj, spiritus rectificati ʒss-ʒj) may be rubbed on with a piece of lint; after the serous crusts resulting from this separate, the same process should be repeated. Pyrogallic acid ointment (10 per cent.) has been recommended. It is applied for three or four days until a brown eschar forms; this is covered with iodoform bandage until the slough separates; the wound is then dressed with iodoform. Painting with a 3 per cent. solution of resorcin, and covering with an india-rubber mask, have been effectual. One of the best methods of local treatment is that by linear scarification (*see* LUPUS VULGARIS). Some

cases, especially those where there is much active inflammation, do best with soothing remedies—such as zinc ointment, or a lead lotion (℞ *Liquoris plumbi subacetatis* ℥j, *glycerini* ℥ij), applied warm after the crusts are removed; and such applications are always of service in the intervals between the caustics.

EDWARD I. SPARKS. ALFRED SANGSTER.

**LUPUS VULGARIS** (*lupus*, a wolf, or a rodent disease; and *vulgaris*, common). **SYNON.**: *Lupus Exedens*; Fr. *Scrofulide Maligne*; *Dartre Rongeante*; Ger. *Lupus*; *Fressende Flechte*.

**DEFINITION.**—A very chronic non-contagious disease of the skin and mucous membranes; chiefly due to an infiltration of small round cells into their substance; attended either with epidermic exfoliation, interstitial absorption, or destructive ulceration.

**ÆTIOLGY.**—The ætiology of lupus vulgaris is unknown. Some cases occur in scrofulous persons with enlarged or suppurating lymphatic glands, and diseases of the bones and joints, or in the children of consumptive parents. An association between lupus and struma is generally admitted in this country, although it is not traceable in every case. Most cases begin very early in life, from the second or third year up to puberty. The ulcerative form—*lupus exedens*—may begin still later, but it is very rare for it to do so after the thirtieth year. Women are more liable to lupus than men, and country people than townspeople. It is in no way due to syphilis, either hereditary or acquired. Local injury may determine the disease. Lupus is probably a local tuberculosis.

**ANATOMICAL CHARACTERS.**—The cutis in lupus is infiltrated with vast numbers of small round cells of about 0·004 mm. in diameter, which encroach on and gradually destroy its proper tissues. The new-growth has a certain resemblance to granulation-tissue (Virchow). It is highly vascular, and contains new-formed capillary vessels. Nodules have been described, disseminated through the cutis, each consisting of one or more central multinuclear giant-cells, surrounded by bands of smaller cells which shade off into the granulation-tissue. These nodules exactly resemble grey tubercle (Friedländer). Some authorities regard these giant-cells as retrograde products, due to central fusion of the endothelial cells of sweat-ducts, blood-vessels, and lymphatics, with persistence of their nuclei (Lang). The epidermis covering the lupus-tissue is thickened; the lower cells of the rete mucosum are fattily degenerated, and contain reddish pigment. Lupus-tissue is very persistent. It may either undergo fatty degeneration and subsequent absorption, without lesion of the epidermis; or else the latter breaks down too, and ulceration is the result. A certain

amount of organisation of the new-growth into connective-tissue takes place. Koch has demonstrated the existence of bacilli in lupus-tissue indistinguishable from the tubercle bacillus; other observers have had difficulty in finding bacilli. The structure of lupus and miliary tubercle are very closely alike.

**SYMPTOMS.**—Lupus usually begins as one or more small, smooth, reddish-brown or reddish-yellow blotches, from the size of a pin's head to a split pea, which may be level with the skin, or else raised in the form of small tubercles. Several of these may become aggregated into a patch, and a number of them may coalesce, so as to cover extensive surfaces of skin; but this is the exception rather than the rule. Any change that occurs is always slow, and a patch the size of a shilling or a florin may take years to form. In the non-ulcerative variety, thin epidermic scales are continually thrown off from the surface of the tubercles—*lupus exfoliatus*. After a while, interstitial absorption of the lupus-cells begins in the older tubercles, and a bluish-white papery scar is left. In other cases the tubercles soften, ulcerate, and become covered with greenish-yellow adherent scabs, beneath which the ulcer extends. The surrounding parts are swollen, and the edge of the ulcer is pinkish. Removal of the scab exposes an ulcerating surface bathed in pus; sooner or later granulations appear, and are liable to be exuberant. The ulceration may attack structures deeper than the skin, and destroy cartilage, fibrous tissues, and muscle. The mucous membranes are not often primarily affected by lupus, though it often extends to them from the skin. The gums, buccal membrane, pharynx, larynx, and epiglottis may be invaded, or the conjunctiva or vagina.

Lupus may occur on any part of the body or limbs, but it has a special preference for the skin of the face; afterwards, in order of frequency, the limbs, buttocks, trunk. The ulcerative form—*lupus exedens*—begins almost exclusively on the nose, attacking chiefly its anterior portion, either the tip or the edges of the alæ. Sometimes it begins within the nostril. Unless proper treatment is adopted the nose may be entirely destroyed, and severe inroads made into the tissues of the cheeks, lips, and other neighbouring parts. The varied aspects that may be assumed by lupus have suggested the terms *L. disseminatus*, *L. hypertrophicus*, where there is much infiltration; *L. serpiginosus* and *L. verrucosus* or *papillomatosus*, in cases where the disease assumes a papillary type.

**COMPLICATIONS AND SEQUELÆ.**—Lupus may co-exist with enlarged and suppurating glands in the neck and elsewhere; with various scrofulous affections; with phthisis; and with chronic Bright's disease. Great deformity may result from the contraction of

the scars which it leaves—for example, ectropion, stricture of the nares, and distortion of the mouth. About a dozen cases have been recorded in which epithelioma developed on a patch of lupus of many years' standing, or on a lupus scar. We may regard such cases as arising from the stimulus of the proliferative processes in the cutis on the neighbouring epithelium.

**DIAGNOSIS.**—The duration and position of lupus vulgaris, and the absence of thick scabiness and itching, will generally render it easy to distinguish this disease from circumscribed forms of psoriasis and eczema; from epithelioma there may be difficulty in middle-aged and old people. The age at which the disease commenced, pain, induration, position, and secondary involvement of glands, help the diagnosis in cases of epithelioma. It is with syphilis that lupus is most apt to be confounded; and the diagnosis between lupus exedens on the nose, and an ulcerating syphilide, is sometimes extremely difficult, or at first sight impossible. In these cases, after carefully considering the history, we must examine other parts of the body for traces of syphilis, and an inspection of the mouth and pharynx will often materially assist us; and lastly, the greater chronicity and slower extension of lupus, as well as its resistance to specific treatment, will generally lead to a correct opinion. Indolence is a character of special value in deciding between lupus and syphilis, particularly as affecting the mucous membranes. Syphilis has also more tendency to suppuration than lupus; and is liable to attack bone. Lupus has to be distinguished from scrofuloderma, which generally commences in softened gland-tissue, and has a tendency to attack the subcutaneous tissue primarily—hence pocketing and forming sinuses.

**PROGNOSIS.**—Lupus is never fatal *per se*, but it can never be looked on in a favourable light, owing to its tendency to relapse under treatment, and its invariable termination in a contracting cicatrix.

**TREATMENT.**—*Internal.*—Internal treatment is only of use in lupus vulgaris where the patient's general health is bad, or where well-marked symptoms of scrofula are present. In these cases great benefit may be derived from tonics, especially the iodide of iron, and from cod-liver oil in as large doses as can be tolerated. Nutritious food should be freely given; and the patient should take plenty of outdoor exercise, but be protected from damp, cold, and sudden changes of climate.

*External.*—The real cure for all forms of lupus must always consist in the destruction of the new tissue forming it, by caustic agents of various strengths. A number of such remedies have been proposed, but the successful application of each seems often to depend more on individual experience of its

use, than on the superiority of any one caustic over the rest. A few of those more generally used may be mentioned. Arsenical paste (Arsenious acid gr. 10; artificial cinnabar, ʒss.; rose ointment, ʒss. (Hebra)), to be spread on linen applied evenly, and bound on the affected part for twenty-four hours; one or two applications may be necessary. The diseased tissue is selected and destroyed by the paste, but the pain is severe. Only limited areas are to be treated. Chloride of zinc paste applied for twenty-four hours is well spoken of. Equal parts of caustic potash and distilled water may be applied with a tiny piece of sponge, so as to limit the action as much as possible. The pain which follows is not of long duration—a point of much importance where a caustic must be repeatedly used. Solid nitrate of silver may be bored freely into all ulcerated parts or soft tubercles. The lupus-tissue offers but slight resistance to it, whereas it will not penetrate or injure healthy parts. This is a safe and harmless method. Acid nitrate of mercury may be painted on with a glass brush. The crusts which form fall off in ten days or a fortnight, and it is not advisable to repeat the application at shorter intervals. As a rule, no dressing except zinc ointment is required. Some authorities prefer the actual or else the galvanic cautery, but both these measures have the disadvantage of disfiguring the parts, so that it is difficult to determine when healthy tissues are reached. In the treatment of the superficial patches of lupus non-exedens, the repeated application of mercurial plaster during several months has sometimes been followed by absorption of the growth. Pyrogallic acid ointment (1 to 10) has been used by Hebra and others with much success. Salicylic acid, enough to make a paste with glycerine, applied to affected parts, destroys the ulceration, and answers well. Where no ulceration exists, demanding active interference, milder remedies deserve a trial, such as lead or calamine lotions. It is often surprising how much improvement may be got by such measures, combined with suitable hygiene.

The local treatment of lupus which now finds most favour is that invented by Volkmann, known as scraping and multiple punctiform scarification.

1. *Scraping* has for its object, first, the removal of products of secretion, scabs and crusts; and, secondly, the removal of neoplasms formed in or on the skin. For this purpose 'spoons' or 'scrapers' of various sizes are made by the instrument-makers. It is difficult to make any impression on the sound skin with such blunt instruments, and on this fact the simplicity and safety of scraping mainly depend. It is almost impossible to do harm, for all that can be made to break down (using moderate force), under the scraper or spoon, is best removed.

2. *Multiple punctiform scarification* is the second mechanical method employed by Volckmann. By this the operator seeks, first, to destroy newly formed vessels; and, secondly, to favour absorption of the neoplasm, by traumatic irritation of the part. It is practised by making hundreds of punctures close together, about two lines in depth. For this purpose a narrow-bladed bistoury may be used; or the same end is gained in less time by employing one of the various instruments made for the purpose, composed of two or more knives set close together. This method of treatment is more applicable to non-ulcerated parts, where the cell-infiltration is diffuse. Such tissue might in some cases be made to break down under the spoon, but less scarring or deformity results if the neoplasm can be made to disappear by the method of puncture.

There is considerable bleeding at first, after either scraping or puncture. This, however, is soon controlled by the application of sponges squeezed out of iced water. The part may be dressed after operation with some simple antiseptic dressing.

Another method is that by linear scarification. This is more suitable to extremely superficial varieties, and especially to lupus erythematosus. It is practised by making in the part numerous parallel incisions about  $\frac{1}{8}$  of an inch apart. Different instruments have been invented for the purpose. After a number of parallel incisions are made in one direction, others are made crossing these, so as to mince up the skin, so to speak, and by this means destroy the new-growth and occlude the vessels.

*The treatment of lupus by Koch's tuberculin.*—This depends on the subcutaneous injection of a toxine derived from a bacillary extract, giving rise to general and local phenomena: the former as constitutional disturbance, fever, congestion of viscera, &c., the latter as inflammatory and absorbent effects at the seat of lesion. The treatment was carried out systematically at the St. Louis Hospital, Paris, soon after its introduction. An analysis of the thirty-eight cases treated will be found in the *British Journal of Dermatology* for March 1891. It was observed that the reaction varied in individuals, as to intensity and duration, even when the dose was very small: as complications were noted—congestion of the lungs, endocarditis, hæmaturia, albuminuria, scarlatiniform eruptions, and erysipelatous inflammation of the face and scalp. As to local effects, not one case was reported cured, in twelve cases the results were *nil*, in eighteen there was 'very slight improvement.' The treatment has been generally abandoned on account of its danger to life and its doubtful efficacy. It is still believed in by some dermatologists, as of use in supplementing operation: it helps to get rid of deep-seated lupus

tissue, out of reach of the curette or antiseptics (Crocker). There seems some reason for believing that the long-continued systematic employment of minute doses of tuberculin (1 mg.) may cause absorption of lupus or of scar-tissue, without producing local inflammation or general disturbance (Pringle).

EDWARD I. SPARKS. ALFRED SANGSTER.

**LYMPH** (*λύμη*, a nymph, water).—Physiologically, lymph signifies the fluid which circulates in the lymphatic system. Pathologically, the term is applied to the coagulable exudation which escapes from the vessels in inflammation. The name 'vaccine lymph,' or 'lymph,' is also given to the fluid contained in the vaccine-vesicle. See INFLAMMATION; and VACCINIA.

**LYMPHADENITIS** (*lymphā*, lymph; and *adenitis*, inflammation of a gland).—Inflammation of lymphatic glands. See LYMPHATIC SYSTEM, Diseases of.

**LYMPHADENOMA**.—SYNON.: Hodgkin's Disease; *Anæmia Lymphatica* (Wilks); Fr. *Adénie* (Trousseau); *Lymphadénie* (Ranvier); Ger. *Pseudoleukämie* (Wunderlich).

DEFINITION.—A disease characterised by more or less widely spread enlargement of the lymphatic glands, accompanied frequently by enlargement of the spleen, and by progressive anæmia.

HISTORY.—Cases of coincident enlargement of the lymphatic glands and spleen were noted by Malpighi (1669) and Morgagni (1752). The nature of the glandular change was first carefully described by Craige (1828); and the general clinical history of the affection was pointed out by Hodgkin (1832), and by Wilks (1856). The most important subsequent observations are those of Virchow (1864), Wilks (1865), Trousseau (1865), Wunderlich (1858 and 1866), Murchison (1870), and Meisner (1884).

NATURE.—The enlargement of the lymphatic glands, which consists at first of mere hyperplasia, and subsequently of fibroid induration, varies much in its extent. A few glands only may suffer, or every gland in the body may be enlarged. The former cases have the characters of a local growth; the latter is distinctly a general disease, for which the term *lymphadenosis* seems the most exact. The glands vary in consistence: when soft there may be a considerable excess of leucocytes in the blood; when hard there may be simple anæmia. This difference does not afford sufficient ground for separation. The enlargement of the spleen is usually due to disseminated growths, arising in the Malpighian bodies; sometimes there is also hyperplasia of the splenic pulp, as in splenic leucocythæmia.

ÆTIOLGY.—In two-thirds of the cases of lymphadenoma, no cause can be traced, and the ascertainable antecedents of the disease,

in most of the remaining cases, evidently constitute only a small part of the influences to which it is due. Hereditary transmission has not been distinctly proved. The disease is three times as frequent in males as in females. It is met with at all ages, but is most frequent in early and late adult life. It occurs, but is not specially frequent, in children under ten years; and, having regard to the numbers living, it is least frequent between the ages of forty and fifty years. Intemperance, mental depression, insufficient food, and over-exertion have been noted, in rare cases, as antecedents. Exposure to cold, in several instances, has appeared to be the exciting cause of the affection. It is doubtful whether the disease has any relation to constitutional syphilis. In several cases the symptoms have first appeared after childbirth. Various febrile affections have, in a few instances, preceded the affection. The exciting cause which has been noted most frequently is some local irritation, as of a decayed tooth, discharge from the ear, sore-throat, inflammation of the lacrymal sac, or eczema. In these cases the glands nearest the source of irritation first enlarged, and then more distant ones became affected.

**ANATOMICAL CHARACTERS.**—The several groups of glands are affected in the following order of frequency, beginning with those most commonly diseased: cervical, axillary, inguinal, retro-peritoneal, bronchial, mediastinal, mesenteric. Subsidiary adjacent glands are often enlarged together with the chief groups; and nodular growths, similar to enlarged glands, arise in the course of the lymphatics in places in which the existence of glands is not usually recognised, so that continuous chains of nodules connect the various groups. The size attained by the glands in lymphadenoma varies from that of a bean to that of a hen's egg. At first the individual glands are separate and movable one on another. Ultimately they often unite to form a conglomerate mass, in consequence, in most cases, of the perforation of the capsules of the glands by growth, which may also invade adjacent parts. The cervical glands are usually enlarged in both the anterior and posterior triangle; and the sub-maxillary glands may encircle the neck beneath the lower jaw. They may press on the trachea or larynx, displace the latter, compress the internal jugular vein, and cause paralysis of the recurrent laryngeal nerve. The occipital glands are usually also enlarged. The axillary glands often form a mass of very large size, and prolongations may extend beneath the pectoral muscle. The glands in the anterior mediastinum frequently suffer, and the growth may extend to adjacent structures, such as the pericardium, which may be perforated. The thymus may be involved, secondarily or primarily, or may escape. The bronchial glands are diseased

more frequently than the cardiac glands, and the trachea and bronchi may be pressed upon, or the lung invaded. The retro-peritoneal glands often form a mass of large size, which may surround and compress the solar plexus, causing symptoms similar to those of Addison's disease. Enlargement of the mesenteric glands is neither common nor considerable. The inguinal group is frequently diseased, and the femoral vessels and crural nerves may be thereby compressed.

The consistence of the enlarged glands may be either soft or very hard. Usually the longer the enlargement has existed, the firmer are the glands. Their section is more uniform than in health. The colour is yellowish or whitish-grey. In the firmer glands dense tracts of fibrous tissue are seen to pass in different directions. Rarely the follicles have a different appearance, being opaque and yellowish from fatty degeneration, whilst the septa are white and conspicuous, from fibroid thickening. Caseation is, however, rare, and when it occurs is commonly confined to one or two glands. The softer glands yield a juice on scraping; the firmer glands yield no juice. In the former, the only histological change is an enormous increase in the cellular elements—the lymph-corpuscles of the reticulum; but the relations of the septa and follicles often remain normal. Sometimes the cell-growth invades the septa, which become split up and disappear; and it may even, in a similar manner, perforate the capsule. The firmer glands present much fibrous tissue, which may be confined to the septa, or invade also the delicate network in the substance of the gland; and then the cells gradually disappear, and the whole substance of the gland may be transformed into a fibrous mass. The tracts of fibrous tissue may have under the microscope a peculiar vitreous aspect, especially around the arteries.

The spleen is diseased in at least four-fifths of the cases, usually in consequence of disseminated growths, often irregular in shape, arising from the Malpighian corpuscles, yellowish or greyish-white, rarely caseating, and usually corresponding in consistence with the glands in the same case and resembling them in structure. The splenic pulp may be normal in quantity, or may be compressed and atrophied. In some cases it is also increased in quantity, and this increase may even be the sole change. In such cases the morbid changes of lymphadenoma and splenic leucocythæmia coexist, and there is often a much greater increase in the white corpuscles of the blood than when the spleen is the seat of simple growths. The size attained, in the cases of nodular growths, is not great, the weight being from ten to thirty ounces. When the splenic pulp is increased, the size attained is rather greater. In the latter case the enlargement is uniform, while it may be irregular when there are

growths. The medulla of bones has been found, in rare cases, to present a change similar to that met with in splenic leucocythæmia and pernicious anæmia. Collections of adenoid tissue elsewhere often undergo changes similar to that of the lymphatic glands. The tonsils, the mucous membrane of the pharynx, the œsophagus, the stomach, and the large and small intestines, may all be the seat of growths, originating in the follicular glands, and sometimes ulcerating. The liver is often the seat of scattered lymphoid growths, usually minute, varying in size from a pin's head to a small pea. They occupy the interlobular spaces. Rarely larger nodular growths are found. In other cases the liver is simply congested. Similar minute growths are often found in the kidneys, chiefly in the cortex; and these organs may also be the seat of parenchymatous degeneration. The peritoneum may be inflamed over enlarged glands, or may be the seat of growths. Growths have also been found in the testicles; and frequently in the lungs, where they may break down and form cavities.

**SYMPTOMS.**—The most important symptoms of lymphadenoma are due to the altered blood-state, and to the enlarged glands. The latter cause the earliest symptoms, and the cervical glands are commonly the first to enlarge. When the internal glands are primarily affected, pain and pressure-signs may precede other symptoms. Occasionally the signs of anæmia precede those of the local change; and, in rare instances, irregular febrile disturbance may occur before the glandular enlargement. The affected glands are smooth, and present, at first, a peculiar mobility, which may disappear when they become adherent, and constitute an irregular lobular tumour of some size. They are usually painless, except during periods of rapid growth. A diminution in size has been observed before death. The enlargement of the cervical glands may cause the neck to equal, or even exceed, the head in circumference. The pressure on the veins may cause symptoms of passive cerebral congestion. The larynx may be displaced; and the movements of the lower jaw may be interfered with. Pressure on the trachea, by the glands in the neck and in the posterior mediastinum, may cause dyspnoea and even death by suffocation. That on the pharynx and œsophagus may obstruct deglutition, and cause death by starvation. The enlargement of the axillary and inguinal glands may interfere with the movement of the limbs, and impede the circulation. Various and serious pressure-effects result from the enlargement of the thoracic and abdominal glands, obstruction in veins, pressure on nerves, &c. The enlargement of the spleen can usually readily be felt, but does not commonly give rise to symptoms. Anæmia is one of the conspicuous symptoms, and

may precede, or succeed, obtrusive affection of the glands. The red corpuscles may be reduced to fifty, thirty, and even twenty-five per cent. of the normal. In most cases there is no marked excess of white corpuscles, but occasionally they are much more numerous than normal. In almost all cases in which their excess is comparable to that met with in splenic leucocythæmia, the splenic pulp is increased in quantity, and the lesions of splenic leucocythæmia and of lymphadenoma are conjoined. The liver may be enlarged from the disseminated growths, and from congestion. Jaundice only occurs from the pressure of enlarged portal glands upon the bile-ducts. Ascites may be due to similar pressure, or to the blood-state, being then part of general dropsy. The function of the kidneys is rarely affected. Stomatitis, sometimes ulcerating, results from the lymphoid growth in the mucous membrane; and a similar change in the stomach causes interference with digestion and vomiting—symptoms which are increased by the anæmia. Slight dyspnoea results from the blood-state, while intense difficulty of breathing, and even actual suffocation, may occur from the pressure of enlarged glands on the trachea or bronchi. The functions of the nervous system are variously deranged by the ill-nourished blood. Towards the end there may be convulsions, delirium, and coma. Pyrexia is a frequent, but not invariable, symptom. It is almost always present in early life, much less common at advanced ages. The temperature may be considerably raised, even when the glandular enlargement is slight; the elevation varies from two to six degrees, and may be continuous, or with daily remissions, or periods of considerable elevation may alternate with periods in which it is only slightly raised.

**COMPLICATIONS.**—The pressure-effects of the enlarged glands, already mentioned, are sometimes so considerable as to give rise to complications, such as thrombosis in vessels, pleural and pericardial effusions, and bronzing of the skin from disease of the solar plexus. Intercurrent affections occasionally met with are Bright's disease, pneumonia, fatty degeneration of the heart and liver, erysipelas, pemphigus, boils, and other effects.

**COURSE AND DURATION.**—Lymphadenoma may remain local for a long time, even years, affecting one group of glands only, and subsequently slowly becoming general. When the general enlargement of glands is established, the disease rarely lasts more than two years. It usually terminates fatally by asthenia; but not rarely by some secondary effect of the morbid process, as asphyxia, starvation, or diarrhœa; or by a complication, especially by pneumonia.

**PATHOLOGY.**—The changes in the glands in lymphadenoma resemble, in the early stage, those which result from simple irritation;

and, as has been seen, the first enlargement often appears to be excited by local irritation. Clinically, however, the disease has a semi-malignant aspect. Dr. Wilks therefore assigned to it a position between cancer and tubercle. Its history suggests that it is due to both constitutional and local causes, and that the extent of these two elements varies in different cases. The constitutional predisposition apparently affects chiefly the lymphatic structures. The assumption of such a predisposition is necessary to explain the general affection of the glands which characterises some cases in the beginning, and also the persistence of the affection when it begins locally, as well as its subsequent extension. In the latter case, however, a process of secondary infection may be at work, the lymphatic tissues, already predisposed, becoming affected by the circulation in the blood of a *materies morbi* derived from the structures first diseased. The existence of an excess of white corpuscles in the blood does not present valid ground for separating certain cases from the rest, and calling them 'lymphatic leucocythæmia.' Most of such cases are, as has been said, forms of mixed disease. In simple lymphadenosis the Malpighian follicles of the spleen are diseased; and when there is a considerable excess of leucocytes in the blood, the splenic pulp is usually also increased in quantity. The anatomical lesions of splenic leucocythæmia and lymphadenosis are conjoined, and to the increase in the pulp the leucocytal excess is due. Occasionally, however, when the diseased glands are soft, lymphoid corpuscles, changed in character, pass from them into the blood, and persist there, leading to an excess of the pale cells. When the glands are hard, the production of lymphoid cells, and their passage into the blood, seem interfered with, and thus simple anæmia results.

**DIAGNOSIS.**—Local glandular growths cannot be sharply separated from cases of generalised lymphadenoma, although they may be clinically distinguished. Generalisation may ultimately occur, even though one group of glands has alone been diseased for many years. In splenic leucocythæmia the glands are only affected late in the disease, after considerable enlargement of the spleen has existed alone for a long time. When the spleen presents great enlargement, and the glands are affected early, the case is usually of the mixed form above described, both splenic pulp and follicles being diseased. In strumous enlargement of the glands, the disease is commonly confined to a single group of glands which have been subjected to local irritation; some of the glands often soften and suppurate; the affection occurs chiefly in early life; and other constitutional signs of tubercular cachexia may be present. Cancer of the glands differs widely in its

microscopical characters from lymphadenoma, but clinically the distinction from a local lymphoma may be difficult, and turns chiefly on the slow extension of cancer to neighbouring glands, and its subsequent localisation in organs rather than in lymphatic structures.

**PROGNOSIS.**—When lymphadenoma is widely spread, or the local growths are considerable in size, a fatal termination is almost certain. The duration, however, in each case, varies much. The younger the patient and the better the preceding health, the longer is the duration of the disease. The consistence of the glands has little prognostic value. The softer they are, the more rapid is the course of the disease; but, on the other hand, if it is influenced by remedial agents, the soft glands can be restored to a better functional condition than the hard. The prognosis is worse the more profound the anæmia. Elevation of temperature as a rule indicates a rapid course, but to this there are some striking exceptions, as in one case under the writer's (W. R. G.) care, in which the glandular enlargement continued slight, although the temperature for twelve months was always above the normal.

**TREATMENT.**—The possibly infecting influence of the primary glandular enlargements has led to their extirpation. Where other glands have been involved, or the spleen has been enlarged, the operation has done no good; and, in such cases, surgical interference is only justified by impending death from the local pressure. But where the affection has been confined to one group of glands, the progress of the disease has been retarded by their removal, and in some cases the malady has even been cured. The degree of anæmia is of great importance as influencing the prospect of benefit, and even of survival from the operation, and the actual proportion of corpuscles should, in all cases, be estimated by the hæmacytometer. An operation should never be performed if the proportion of red corpuscles is less than 60 per cent. of the normal. A slight excess of white corpuscles does not militate against the success of an operation. Other methods of local treatment have been employed, with some benefit, especially rubbing and shampooing, the alternate application of heat and cold, compression, and blistering. Galvanopuncture is useless. Various substances have been injected into the glands—iodine, nitrate of silver, carbolic acid, arsenic. The last has alone appeared useful (Winiwarter), but it was, in all cases, given internally at the same time. Of internal remedies arsenic is incomparably the most potent. It should be pushed to the largest doses the patient can bear, such as ℥xv. of liquor arsenicalis three times daily. It often causes some pain in the glands, followed by their diminution in size, and even, in a few recorded cases, by

their complete disappearance. Although such a favourable result has not come under the writer's personal observation, he (W. R. G.) has seen a marked diminution obtained in the size of glands which were before steadily enlarging, a diminution which has been maintained for years. Phosphorus has been given in the disease (first by Verneuil), but it is far less useful than arsenic. Iodine and iodide of potassium are of little service. Cod-liver oil is useful when there is any evidence of a scrofulous diathesis. Mercury and carbolic acid have been given internally without success. Iron, unless alone, has sometimes appeared to do good when given in conjunction with other remedies. Change of air, general tonics, and careful diet are often of considerable service, especially when employed along with other measures.

W. R. GOWERS. FREDERICK TAYLOR.

**LYMPHANGIEITIS** (*λύμφη*, water, or lymph; and *ἀγγείον*, a vessel).—Inflammation of lymphatic vessels. See LYMPHATIC SYSTEM, Diseases of.

**LYMPHANGIECTASIS** (*lymp̄ha*, lymph; and *angiectasis*, vascular dilatation). Lymphatic varix, or varicose dilatation of lymphatic vessels. See LYMPHATIC SYSTEM, Diseases of.

**LYMPHATIC SYSTEM, Diseases of.**—SYNON.: Diseases of the Absorbent System; Fr. *Maladies du Système Lymphatique*; Ger. *Krankheiten des Lymphsystems*.

There is no essential difference between the lymphatic and lacteal systems, which together constitute the absorbent system. The present article deals with those diseases of the lymphatic vessels and glands which are more or less of a local nature; and what is stated with regard to the former will apply generally to the lacteals, but attention will be directed to any points connected with these vessels calling for special notice. Some of the affections involving these structures are considered in separate articles, and need, therefore, merely be mentioned here. It may be remarked that important relations exist between the absorbent system and certain diseases, namely, some of those belonging to the zymotic class, and those depending upon septic conditions, such as plague, typhus and typhoid fever, diphtheria, scarlatina, rubeola, dengue, erysipelas, glanders, malignant pustule, snake-bite, dissection or *post-mortem* wounds, and certain forms of serous inflammation, such as puerperal peritonitis. The lymphatics of the pleura and lung are in rare instances the seat of suppuration. Moreover, the lymphatic vessels seem to be materially involved in some skin-diseases, such as erythema and elephantiasis; while there are certain structures in the body which consist mainly of lymph-

atic follicles, and their diseases principally affect these follicles. The absorbent system is also concerned in disseminating such diseases as cancer, syphilis, or tubercle through the system. These points are more fully dwelt upon in their appropriate articles, and now the individual diseases of the lymphatic system will be severally discussed.

1. **Acute Inflammation.**—According to the structures involved, acute inflammation, connected with the lymphatic system, presents three varieties, namely: (a) where the vessels are alone affected—*lymphangitis* or *angieoleucitis*; (b) where the condition is limited to the glands—*adenitis*; or (c) where both vessels and glands are involved. It will be convenient to consider these varieties together. As a rule the disease is localised, but under certain circumstances the lymphatic system is more or less widely implicated, especially if the inflammation is of a septic character. It may be set up and extend with great rapidity.

**ETIOLOGY AND PATHOLOGY.**—The causes of acute inflammation of the lymphatic vessels or glands may be thus indicated: 1. *Traumatic*, including such injuries as wounds, contusions, or a severe strain. 2. *Irritation from without*. Strong heat, as that of the sun, may set up inflammation of the superficial lymphatics. Pressure or friction may also produce this effect upon the vessels or glands. It is not an uncommon practice to excite inflammation artificially in the glands, for the cure of certain of their diseases, by injecting irritants into their substance. 3. *Irritation from within*. This may be due to inflammation in the vicinity, suppuration, ulceration, diseases of joints or bones, and other causes. In medical practice the implication of the glands under the jaw, in cases of diphtheria and scarlatina, is a familiar illustration of this class of cases; or the inflammation of the glands behind the ear in cases of impetigo of the head. The lymphatic vessels are also more or less involved in phlegmasia dolens. 4. *Specific irritations*. These deserve separate recognition, and include syphilis, gonorrhœa, and various septic poisons, which frequently affect the lymphatic structures. Inflammation of the absorbent glands is also an important feature in plague, glanders, and certain other specific diseases.

With regard to the modes in which the inflammation is set up, this may happen in several ways. In the first place, the cause may act directly upon the lymphatic vessels or glands, as in the case of injury. Secondly, these structures may be involved by extension from neighbouring parts. Glands are frequently affected in this way; and lymphatic vessels may be involved by continuous extension of irritation from inflamed organs, serous membranes, or other

structures with which they are connected. Thirdly, the cause of the inflammation is often more or less remote from the situation in which it appears, especially in the case of the glands. This may arise from the inflammatory change passing continuously along the vessels from some seat of irritation to the glands in their course; or morbid products may be carried by the current of lymph to the glands, the vessels themselves being unaffected, when the inflammation thus set up is said to be *sympathetic*. In other instances pus has been found within the lymphatic vessels, having made its way from some seat of suppuration. It should also be mentioned that lymphatic inflammation can originate a similar condition in other structures, such as the joints, and this may be of a purulent character.

Inflammation is much more readily excited in the lymphatic structures in some persons than in others, and especially in those who are strumous or tubercular. The glands are more liable to be affected in the early periods of life. A low state of the general health may predispose to inflammation of these structures from slight causes. Glands which are chronically inflamed are very liable to become the seat of acute inflammation from slight causes.

**ANATOMICAL CHARACTERS.**—Inflammation affecting the lymphatic vessels presents two forms, but they may be met with together. When the minute capillary network is involved, the condition is termed *reticular lymphangitis*; the skin and its capillaries are generally affected at the same time, so that there is more or less diffused redness, but it may present a reticulated arrangement. *Tubular lymphangitis* signifies that the main vessels are implicated. They are visible on the surface as red lines, straight or wavy, passing to the glands. They become dilated, and their walls thickened. Their internal coat is opaque and uneven, and the endothelium often disappears. Coagulation of the lymph within the vessels takes place, closing up their channel. The coagulum may become organised, so that they are permanently obliterated; or it may soften and even suppurate at the centre, and the products may enter into the general circulation, thus causing septicæmia or pyæmia. The inflammation is liable to extend to the surrounding cellular tissue, leading to exudation, hyperplasia of cells, and consequent swelling and thickening.

Inflammation of lymphatic glands is characterised in the early stage by swelling, congestion, and increased firmness. The lymph accumulates, exudation takes place, and abundant cells are present. The inflammatory process may soon subside, terminating in resolution. In many cases, however, suppuration ensues, especially in certain forms of inflammation, this change

commencing in the centre of the glands, the cavities of which become more or less speedily filled with pus. The inflammation spreads to the surrounding cellular tissue, and, an abscess being formed, the pus makes its way to the surface. If the glands are situated within the body, they may burst internally, or irritate adjacent structures, and thus lead to serious consequences. Sometimes the glands remain permanently enlarged and indurated, especially after repeated attacks of inflammation; and they may become adherent to the parts around. A single lymphatic gland may be inflamed, but it is common for a cluster or chain of these structures to be involved. In some cases the inflammation assumes a more or less sub-acute character, and the progress of events is slower.

**SYMPTOMS.**—These are *local* and *general*. The *local* phenomena consist of subjective sensations, and objective signs. Pain is felt at the seat of inflammation, which may be very severe, often accompanied with a sense of heat or burning, and stiffness or tension. There is usually marked tenderness, and this may be present when little or no spontaneous pain is complained of, while it is often remarkably limited to the line of an affected lymphatic vessel. Movement also increases the pain. The subjective sensations are more severe as a rule when the glands are involved. When suppuration takes place, the pain tends to assume a shooting and throbbing character. As regards objective signs, inflamed lymphatic vessels, if superficial, are usually visible as red lines, either straight or wavy, running in the direction of the glands; or there may be separate red patches. Should they be deeply situated, however, the vessels cannot be seen. The larger trunks may be felt by the fingers, being cord-like, firm, and knotted. The surrounding tissues are seen and felt to be more or less swollen and indurated. If the circulation of the lymph is much interfered with, a limb may be considerably enlarged, and presents a feeling of firmness and solidity, owing to the occurrence of lymphatic œdema.

When superficial lymphatic glands become inflamed, their enlargement can be made out on examination, and the surrounding tissues may also be swollen. At first they feel firm, but if suppuration takes place they become more and more soft, and at last present a sensation of fluctuation. The overlying skin is markedly red, and there is often subcutaneous œdema. Suppurative inflammation of lymphatic glands constitutes the condition known as 'bubo.' If not opened artificially, the abscess ultimately bursts externally, but it may burrow considerably before doing so, and the opening is often imperfect. *See* BUBO.

The *general* symptoms accompanying inflammation of the lymphatic structures vary

in their intensity in different cases, according to its severity, extent, and results. In the slighter cases there is no obvious constitutional disturbance. As a rule, however, more or less fever, with its accompanying symptoms, sets in, preceded often by shivering or even distinct rigors. If suppuration occurs, the rigors may be repeated; the pyrexia increases; and more or less wasting and weakness follow, should there be prolonged discharge of pus. Where the inflammation is of a septic character from the first, or when septic matters are conveyed into the circulation, the general symptoms are indicative of septicæmia, such as repeated rigors, high and erratic fever, great weakness and prostration, low nervous symptoms, weak and rapid cardiac action and pulse, and other typhoid phenomena. The termination is then usually fatal.

**TREATMENT.**—In the management of any acute inflammation affecting the lymphatic vessels or glands, the first indication is to get rid of its cause, if this be practicable. In the next place rest is of essential importance, and the affected part should be so placed as to avoid all pressure or tension. As regards *local* treatment, the application of heat and moisture, by means of fomentations and poultices, usually answers best. To these anodynes may be added, if necessary, especially belladonna; and the latter may be often applied with advantage in the form of extract, mixed with glycerine. It is not uncommonly advisable to take away blood locally from the neighbourhood of inflamed glands, by means of leeches. Some authorities maintain that suppuration may sometimes be prevented by counter-irritation around the glands, effected by applying blistering-fluid or strong iodine. If suppuration take place, the progress of the pus towards the surface must be encouraged by the usual means, and the abscess opened as soon as practicable. Should *general* treatment be required, at first it is usually necessary to keep the patient on low diet, to open the bowels well, and perhaps to administer some simple saline mixture. When suppuration occurs, a more or less supporting tonic and stimulant treatment is called for. Septicæmia demands the free use of alcoholic stimulants, the administration of full doses of quinine, and other appropriate remedies.

**2. Chronic Inflammation.**—Only the lymphatic glands can be said to be liable to this affection—*chronic adenitis*. They may remain in a condition of chronic inflammation after one or more acute or sub-acute attacks; or this is set up as a chronic affection from some continued or repeated irritation. Formerly a low form of chronic inflammation was regarded as the primary lesion in scrofulous or tubercular glands, and some pathologists still hold this view. The

affected glands are enlarged and firm, and often somewhat painful and tender. These changes may be due partly to a hyperplasia of the gland-structures, partly to an exudation into their midst. Frequently they continue in this condition for a long time, without undergoing any obvious change, but they are liable to acute exacerbations from slight causes. They may ultimately become the seat of caseous degeneration, or of suppuration. The circulation of the lymph through the involved glands is prevented to a greater or less degree. Usually there is no constitutional disturbance, unless a considerable number of glands are implicated, or they undergo degeneration or suppuration.

**TREATMENT.**—It is not desirable to allow chronic adenitis to continue, as unpleasant or even serious consequences may ensue, and therefore the affected glands should be subjected to proper treatment without delay. Any source of irritation must be removed at the outset. Gentle friction over the enlarged glands, with some simple oleaginous or greasy material, may be effectual in reducing them, or it may be necessary to rub in weak iodine or iodide of potassium ointment, or to paint the surface with tincture of iodine. Counter-irritation by blisters may be sometimes useful. Internally cod-liver oil and quinine are frequently of much value; preparations of iron are also often very serviceable, especially the syrup of the iodide and phosphate. If the affected glands do not yield to treatment, it may become a question whether they should not be removed by operation.

**3. Scrofulous Disease.**—By this term it has been customary to designate a chronic disease of the absorbent glands, characterised by enlargement, followed by degeneration and caseation of the tissues and morbid products, and ending in unhealthy suppuration. It will be convenient to discuss briefly the condition under this heading, as the cases in which it occurs are definite enough, although there is a difference of opinion as to the nature of the primary lesion. By many it is regarded as essentially *tubercular*, and as a manifestation of the tubercular or scrofulous diathesis, but others regard it as a low form of inflammation.

**ÆTIOLOGY.**—Scrofulous disease of the glands often occurs in those who are obviously of a strumous or tubercular habit, or who are hereditarily predisposed; but this is by no means always the case, for the subjects of the glandular affection may be apparently strong and healthy. Children and young persons are by far most frequently affected. The change is often originally set up by some local irritation, but in many instances there is no such obvious cause, and it appears to commence spontaneously. Once an absorbent gland becomes the seat of scrofulous lesions, others in connexion with it, or even at a

distance, are very liable to become secondarily implicated.

**ANATOMICAL CHARACTERS.**—Scrofulous disease may involve the lacteal as well as the lymphatic glands, and of the latter those within the body may be affected, as well as those which are external. Of the external glands, those in the neck and under the jaw are most commonly first involved. The changes always go through a more or less chronic course. At first the glands become enlarged and firm, and the enlargement has been attributed to inflammatory products, hyperplasia of the lymphatic elements, or tubercle; and tubercle-bacilli have been found. Subsequently as a rule caseation takes place, the substance of the glands becoming yellow and softened; then a slow process of unhealthy suppuration generally ensues, leading to the formation of chronic abscesses. The skin over them presents a congested appearance, and is often undermined for some distance, the subcutaneous tissues being involved in the suppurative process. If the abscesses are not properly treated, they are liable to cause much destruction of the skin, and to leave unhealthy sinuses and ulcers when they burst of their own accord. If they subsequently heal, this is often attended by extensive scarring, and the scars are permanent, but become less marked in course of time. In some cases the glands do not suppurate, but after caseation they become calcified and inert; it appears probable that this result may take place even after the formation of pus, which then becomes inspissated, and mixed up with calcareous matter.

**SYMPTOMS.**—In the case of the external glands, with which we are now concerned, the changes above described can be observed clinically. They are attended with little or no pain, but there is usually more or less tenderness. Constitutional symptoms are as a rule prominent, when the glands become involved to any considerable extent, as evidenced by wasting, anæmia, general weakness, and more or less pyrexia, which, if there should be abundant suppuration, is apt to assume a hectic type. If the glandular mischief is limited, however, the system may suffer but little or not at all; and even after it has been extensive and severe, so as to lower the patient very much, recovery may take place under appropriate treatment, the patient ultimately becoming strong and robust. When the internal glands are affected, they may give rise to symptoms from their mere mechanical presence, such as those indicative of pressure or irritation; and if destructive changes occur in them, very serious results are liable to be produced. The general symptoms are also usually more marked in these cases, and may become extreme in degree. It is highly probable that phthisis may be set up by an

infective process, in connexion with suppurating or caseous scrofulous glands. *See* BRONCHIAL GLANDS, Diseases of; and MESENTERIC GLANDS, Diseases of.

**TREATMENT.**—*General* treatment is of essential importance in the treatment of scrofulous glands. The patient should be placed under the most satisfactory sanitary conditions that can be obtained; but in many cases this is a very difficult matter, and it is of great importance, if possible, to remove from their unhealthy and often wretched homes those suffering from this affection, and to treat them in suitable sanatoriums or hospitals. They should be as much as possible in the open air, and a change of climate and surroundings will often prove of decided benefit. Residence at the seaside, with sea-bathing, is also of essential service, or a sea-voyage may be desirable. Margate is deservedly in great repute as a seaside resort for cases of scrofulous glands. The digestive functions require careful attention and regulation; and the food must be nutritious, including abundance of good milk, fresh eggs, and such articles of diet. As regards medicines, those which are usually indicated are cod-liver oil, quinine, and preparations of iron, especially the syrup of the iodide, steel-wine, or Parrish's syrup. In certain cases marked benefit has been found to result from the administration of minute doses of sulphide of calcium, and from chloride of calcium.

*Local* treatment is usually called for. In the early stages attempts may be made to cause absorption of the enlarged glands, but these must be cautiously conducted. Gentle friction, the application of preparations containing iodine or certain iodides, and the use of poultices or fomentations of sea-weeds, are the measures usually adopted. In some cases it certainly seems the best plan of treatment to try to encourage suppuration in glands which are in a torpid state, and which cannot be absorbed. For this purpose they have been injected with irritants. Suppuration and its consequences must be treated on ordinary principles; but it should be remarked that abscesses should not be allowed to burst of their own accord, but need surgical interference, as otherwise they may lead to much destruction of the skin and subcutaneous tissues. In suitable cases the best treatment consists in clearing out caseous materials, or removing masses of glands by surgical methods. Injection of tuberculin has been tried, but, to say the least, with doubtful results.

**4. Hypertrophy and Atrophy of Glands.**—The lymphatic glands become *hypertrophied* under different circumstances. In some cases there is a mere local hypertrophy, which shows no tendency to progress towards other parts, and which may be due to some obvious irrita-

tion, or is independent of any known cause. This morbid change is most important, however, in the disease termed *lymphadenoma*, *lymphoma*, or *Hodgkin's disease*, in which there is a progressive enlargement of the lymphatic glands; and in one form of *leucocythæmia*. These affections are discussed in separate articles. Without entering upon any description, therefore, it will suffice to remark that in these affections the enlargement varies much in degree and extent in different cases; and that the glands usually show no tendency towards any degenerative or destructive change. Clinically they are recognised by their obvious physical characters when superficial; or by physical signs when situated in internal cavities. As a rule they are painless; but may give rise to various symptoms by causing mechanical pressure, irritation, or destructive effects. In many cases more or less severe general symptoms are present. See *LEUCOCYTHEMIA*; and *LYMPHADENOMA*.

*Atrophy* of lymphatic glands may occur from various causes, as after inflammation; as a senile change; or after the removal of a limb, or its long-continued want of use. No definite effects can be referred to this condition, but if there should be extensive glandular atrophy, it might obviously interfere with the due nutrition of the blood and general system.

**5. Morbid Formations and Deposits in Glands.**—Under this heading the following may be considered:—

(a) *Cancer and Sarcoma.*—The various forms of malignant growth frequently involve lymphatic glands. In most cases the disease is secondary to cancer in some neighbouring part, and the glands are very prone to become involved, owing to the direct communication through the lymphatics. This is well exemplified by the implication of the axillary glands when the breast is the seat of cancer. Not uncommonly, however, the formation in the glands is primary, and then involves other structures secondarily by direct extension or convection. All forms of cancer are met with, but the encephaloid variety is most common. When secondary, however, it generally approximates in characters to the primary formation, and hence may be of a scirrhus or melanotic type. Secondary sarcoma occurs in the lymphatic glands; and there is also a primary disease named *multiple* or *lympho-sarcoma*, which particularly affects the mediastinal glands. A malignant glandular tumour may attain a considerable size, and it is more or less nodulated. The consistence will depend on the variety of the growth; often it is soft, and a milky juice escapes on pressure. If cancerous glands are external, they can be recognised on examination, and are usually painful and tender. When situated internally they give rise to physical

signs of their presence, either in the chest or the abdomen, and to more or less pressure-symptoms, which may be combined with the constitutional symptoms of malignant disease. It may be very difficult to distinguish clinically between cancerous glands and lymphadenomatous growths situated internally.

(b) *Albuminoid disease.*—The absorbent glands are liable to be involved in conditions which give rise to albuminoid degeneration. On section they present the peculiar waxy, pale, translucent, homogeneous appearance characteristic of tissues which are the seat of this change. The glands may be enlarged; but when they attain a considerable size, this is partly due to hypertrophy. In other cases they are small and firm. This condition may give rise to symptoms by pressure, as sometimes happens in the case of albuminoid glands in the portal fissure, which may cause ascites or jaundice.

(c) *Pigmentation.*—The bronchial glands are usually the seat of a deposit of black particles, in cases where the lungs are similarly affected, as in miners, colliers, &c. They are enlarged to some extent, and black; and a black liquid escapes on pressure. This condition does not give rise to any obvious symptoms. More or less pigmentation of these glands is often observed with advancing age.

(d) *Syphilitic disease.*—The glands in the groin are affected from the irritation of the primary syphilitic sore, and others are often involved in connexion with its secondary and tertiary manifestations. Syphilitic glands are of moderate size, indurated, indolent, painless, and freely movable.

(e) *Tubercle.*—In addition to what has been stated under *SCROFULA*, it must be mentioned that distinct grey granulations are sometimes found in connexion with the glands in acute tuberculosis. A class of cases of progressive diffused tuberculous disease of the lymphatic glands has also been described, occurring in adults, in which nearly all the glands in the body are simultaneously, or in rapid succession, affected with tubercle, leading to suppuration. They are attended with fever and rapid wasting, and end fatally in from six months to a year. (*Principles and Practice of Medicine*: Fagge and Pye-Smith.)

**TREATMENT.**—Practically the treatment of the morbid formations in glands just considered, if any be called for, consists in measures directed against the constitutional condition of which they are a manifestation. Operative interference may be demanded in suitable cases. Symptoms, especially those resulting from pressure, may also require special treatment.

**6. Chronic Changes affecting Lymphatics.**—The lymphatic vessels are subject to two principal classes of chronic changes,

namely: (a) *Dilatation* and *hypertrophy*; and (b) *Obstruction*.

(a) *Dilatation and Hypertrophy*.—*Lymphangiectasis*.—Leaving out of consideration the thoracic duct and receptaculum chyli, the lymphatics, either superficial or deep, and also the lacteals, may become more or less dilated and hypertrophied. Even the vessels of internal mucous or serous membranes may be thus affected. In most cases the larger trunks are implicated, but the capillary plexuses are sometimes chiefly or alone involved. With regard to the causes of this condition, it is often congenital, and has then been attributed to a want of specialisation in the lymphatic system of certain parts. In other cases it is evidently due to some obstruction to the circulation of the lymph, and consequent enlargement of the vessels behind the impediment. Such obstruction may be seated in the glands or vessels, and in the latter case may be due to internal plugging or to external pressure. In some instances the enlargement of the lymphatics partakes of the character of a primary hypertrophy, either alone or along with other tissues, as is the case in connexion with elephantiasis and certain other growths, of which enlarged lymphatics constitute an important element. Dilatation has been also attributed to a supposed paralysis of the muscular coat of the lymphatic vessels. *Lymphangiectasis* is most common in warm and moist climates.

There are various forms which enlarged lymphatics assume. Thus, there may be simply a localised dilatation of the capillaries, constituting a visible freely anastomosing reticulum or network. More commonly the trunks are enlarged, assuming a tubular, fusiform, varicose, saccular, or cirroid form. Or a distinct growth may be produced, which has been specially termed *lymphangiectasis*, and has been divided by Wagner into three varieties—(i) *simple*; (ii) *cavernous*; (iii) *cystoid*—names which sufficiently indicate their several peculiarities. The walls of the vessels are often more or less thickened from hypertrophy. Dilated lymphatics are liable ultimately to give way, with consequent escape of the lymph.

Clinically, the conditions now under consideration are visible when superficial, or when occurring on a surface which can be inspected. The appearances will differ according to the particular morbid change present. There may be a distinct tumour; or the enlarged lymphatics may only form one element in certain growths. Cystic formations originating in the lymphatic system are said to be most common in connexion with the upper lip, tongue, and neck. Enlargement of the superficial lymphatics is chiefly observed on the inner side of the thigh, the sides of the abdomen, and the scrotum and penis; they appear in the form

of vesicles like grains of sago, grouped regularly or irregularly. Sometimes only ampullæ are formed, which are generally soft and painless. These conditions have been mistaken for hernia, abscess, scrofulous glands, and other diseases. Should the dilated lymphatics rupture subcutaneously, vesicles containing a clear or milky fluid appear. They may rupture on the surface of the skin, the lymph being discharged externally, which is an important point in the diagnosis of doubtful cases. When dilated absorbents are situated internally, they cannot be recognised unless they should happen to rupture, with the escape of their contents by some outlet. This applies mainly to the lacteals, the contents of which may pass out with the stools, and to the urinary mucous membrane, it being supposed by some pathologists that the condition termed *chyluria* is merely due to the rupture of dilated lymphatic vessels in this membrane. See CHYLURIA.

(b) *Obstruction*.—As in the case of dilatation, the capillary plexuses or larger lymphatics may be obstructed. This may arise chiefly from plugging of their channels by coagulated lymph; inflammation of the vessels; pressure by enlarged glands, aneurysms, or other tumours, or merely as a result of inflammation of the cellular tissue around the vessels. It may be remarked here that lymphatic tissues, similar to those observed in the glands, sometimes form here and there in the course of the lymphatics in cases of lymphadenoma. The lymphatics of the urinary mucous membrane are also supposed by some pathologists to be blocked by *filaria*, and they consider that this is the cause of chyluria. See FILARIA SANGUINIS HOMINIS.

The effects liable to be produced by obstruction of lymphatic vessels are swelling, from so-called lymphatic œdema; and dilatation of the vessels behind the obstruction, which may lead to their rupture. It is by these effects alone that this condition can be recognised clinically.

TREATMENT.—But little can be done for the chronic changes affecting lymphatic vessels now under consideration. Proper bandaging, or the use of some elastic support, may be of use in treating dilated vessels, if they happen to be conveniently situated. Friction and kneading may assist in removing lymphatic œdema due to obstruction. Growths come under the treatment of the surgeon, and do not call for any special remark here.

FREDERICK T. ROBERTS.

**LYMPHATIC TEMPERAMENT.**  
See TEMPERAMENT.

**LYMPHOMA.**—A synonym for lymphadenoma. See LYMPHADENOMA.

**LYMPHORRHAGIA** or **LYMPHORRHŒA** (*lymphā*, lymph, from *λύμψη*, water; and *ρήγνυμι*, I burst forth, or *ρέω*, I flow).

**DEFINITION.**—These terms literally signify a flow of lymph, but they are used to indicate an abnormal discharge from any part of the absorbent system, whether it be of lymph or of chyle.

**ÆTIOLOGY AND PATHOLOGY.**—Lymphorrhagia may take place from the lymphatic capillaries or trunks; from the lacteals; from the absorbent glands; or from the receptaculum chyli or thoracic duct. Cases in which this condition occurs are usually divided into *traumatic* and *idiopathic*, according to their apparent causation. In the former the cause is a wound, which generally affects either the thoracic duct, the larger lymphatic trunks, or the glands. A discharge of lymph has in rare instances followed even a slight wound, particularly in the neighbourhood of joints, and this was attributed by the late Mr. Messenger Bradley to a constitutional defect—a *lymphorrhagic diathesis*, corresponding to the hæmorrhagic diathesis. *Idiopathic* lymphorrhœa is almost always the result of dilatation of one or more vessels, which ultimately rupture. They are often greatly distended before they give way. Allusion may be made here to the supposed relation of chyluria to the presence of *filariæ* in the lymphatic vessels of the urinary organs, these parasites causing them to rupture, and the lymph consequently being discharged with the urine. See *FILARIA SANGUINIS HOMINIS*.

**SYMPTOMS AND EFFECTS.**—Should an escape of lymph take place upon any part of the surface of the body, it differs much in its quantity and characters in different cases. It may be less than an ounce, or amount to five and even ten pounds within the twenty-four hours; while in the same case its quantity is liable to variation from time to time, and the flow has even been known to assume a periodic character, increasing during the period of digestion. In traumatic cases the discharge either presents the ordinary appearance of lymph, being clear and limpid,

or it is mixed more or less with blood or with inflammatory products. When rupture takes place spontaneously after dilatation of the vessels, the fluid is more like chyle, being more or less milky and white, from the presence of particles of fat, but its characters are liable to alter from time to time. It contains a variable quantity of fibrinogenous elements, and is proportionately disposed to coagulate spontaneously. Internal lymphorrhagia causes different results. In the case of the intestines and urinary organs, the fluid is discharged with the fæces and urine respectively, in the former case being supposed to give rise to fatty stools, and in the latter to chyluria. The late Mr. Bradley attributed some cases of effusion into serous cavities, such as certain forms of hydrocele, hydrocephalus, pleuritic effusion, and ascites, to a lymphorrhagia into the respective cavities; and the writer has met with a case of ascites which seemed to support this view. Fatal peritonitis has resulted from the entrance of chyle into the peritoneum, owing to the rupture of a dilated receptaculum chyli. The escape of lymph or chyle out of the system tends to affect the general health, and, if it is in large amount, this is likely to lead to marked emaciation, debility, and anæmia.

**TREATMENT.**—In external lymphorrhagia all that can be done is to check the flow of lymph by pressure of bandages, and the application of astringents. In cases where it takes place into internal passages, tincture of perchloride of iron in full doses may be of service. The general condition must be attended to, and improved by nutrients and tonics, if required.

FREDERICK T. ROBERTS.

**LYPEMANIA** (*λύπη*, grief; and *μανία*, madness).—The name applied by Esquirol to the form of insanity characterised by mental depression, usually called melancholia. See *MELANCHOLIA*.

**LYSIS** (*λύω*, I dissolve).—This word had formerly various significations, but is now generally applied to the *gradual* decline of any disease or pathological process, especially fever. See *FEVER*.









